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POLYCYCLIC AROMATIC HYDROCARBON HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

by

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SUMMARY

This account synthesizes available technical literature on ecological and toxicological aspects of polycyclic aromatic hydrocarbons (PAHs) in the environment, with special reference to natural resources. Subtopics include: chemical properties, sources, and fate; background concentrations in biological and nonbiological samples; toxic and sublethal effects to flora and fauna; and proposed criteria and research needs for the protection of sensitive species.

PAHs consist of hydrogen and carbon arranged in the form of two or more fused benzene rings. There are thousands of PAH compounds, each differing in the number and position of aromatic rings, and in the position of substituents on the basic ring system. Environmental concern has focused on PAHs that range in molecular weight from 128.16 (naphthalene, 2-ring structure) to 300.36 (coronene, 7-ring structure). Unsubstituted lower molecular weight PAH compounds, containing 2 or 3 rings, exhibit significant acute toxicity and other adverse effects to some organisms, but are noncarcinogenic; the higher molecular weight PAHs, containing 4 to 7 rings, are significantly less toxic, but many of the 4- to 7-ring compounds are demonstrably carcinogenic, mutagenic, or teratogenic to a wide variety of organisms, including fish and other aquatic life, amphibians, birds, and mammals. In general, PAHs show little tendency to biomagnify in food chains, despite their high lipid solubility, probably because most PAHs are rapidly metabolized. Inter- and intraspecies responses to individual PAHs are quite variable, and are significantly modified by many inorganic and organic compounds, including other PAHs. Until these interaction effects are clarified, the results of single substance laboratory tests may be extremely difficult to apply to field situations of suspected PAH contamination.

PAHs are ubiquitous in nature--as evidenced by their detection in sediments, soils, air, surface waters, and plant and animal tissues--primarily as a result of natural processes such as forest fires, microbial synthesis, and volcanic activities. Anthropogenic activities associated with significant production of PAHs--leading, in some cases, to localized areas of high contamination--include high-temperature (>700 ^{o}C) pyrolysis of organic materials typical of some processes used in the iron and steel industry, heating and power generation, and petroleum refining. Aquatic environments may receive PAHs from accidental releases of petroleum and its products, from sewage effluents, and from other sources. Sediments heavily contaminated

with industrial PAH wastes have directly caused elevated PAH body burdens and increased frequency of liver neoplasia in fishes.

At present, no criteria or standards have been promulgated for PAHs by any regulatory agency for the protection of sensitive species of aquatic organisms or wildlife. This observation was not unexpected in view of the paucity of data on PAH background concentrations in wildlife and other natural resources, the absence of information on results of chronic oral feeding studies of PAH mixtures, the lack of a representative PAH mixture for test purposes, and the demonstrable--and, as yet, poorly understood--effects of biological and nonbiological modifiers on PAH toxicity and metabolism. By contrast, criteria for human health protection and total PAHs, carcinogenic PAHs, and benzo(a)pyrene have been proposed for drinking water and air, and for total PAHs and benzo(a)pyrene in food: drinking water, 0.01 to <0.2 ug total PAHs/l, <0.002 ug carcinogenic PAHs/l, and <0.0006 ug benzo(a)pyrene/l; air, <0.01 ug total PAHs/m^3, <0.002 ug carcinogenic PAHs/m^3, and <0.0005 ug benzo(a)pyrene/m^3; food, 1.6 to <16.0 ug total PAHs daily, and 0.16 to <1.6 ug benzo(a)pyrene daily. In view of the carcinogenic characteristics of many PAH compounds and their increasing concentrations in the environment, it now seems prudent to reduce or eliminate them wherever possible, pending acquisition of more definitive ecotoxicological data.

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INTRODUCTION

Several polycyclic aromatic hydrocarbons (PAHs) are among the most potent carcinogens known to exist, producing tumors in some organisms through single exposures to microgram quantities. PAHs act at both the site of application and at organs distant to the site of absorption; their effects have been demonstrated in nearly every tissue and species tested, regardless of the route of administration (Lee and Grant 1981). The evidence implicating PAHs as an inducer of cancerous and precancerous lesions is becoming overwhelming, and this class of substances is probably a major contributor to the recent increase in cancer rates reported for industrialized nations (Cooke and Dennis 1984). PAHs were the first compounds known to be associated with carcinogenesis (Lee and Grant 1981). Occupational skin cancer was first documented in London chimney sweeps in 1775 and in German coal tar workers in the late 1800's. By the early 1900's, soot, coal tar, and pitch were all found to be carcinogenic to humans. By 1918, it was shown that topical applications of coal tar produced skin tumors in mice and rabbits; benzo(a)pyrene, a PAH, was identified as one of the most carcinogenic compounds in coal tar (Dipple 1985). The carcinogenic activity to man of soots, tars, and oils is beyond dispute. In addition to the skin cancers noted initially, higher incidences of respiratory tract and upper gastrointestinal tract tumors were associated with occupational exposures to these carcinogens (Dipple 1985). PAH-induced cancers in laboratory animals is well documented. Benzo(a)pyrene, for example, has produced tumors in mice, rats, hamsters, guinea pigs, rabbits, ducks, and monkeys following administration by oral, dermal, and intraperitoneal routes (Pucknat 1981). Teratogenic or carcinogenic responses have been induced in sponges, planarians, echinoderm larvae, teleosts, amphibians, and plants by exposure to carcinogenic PAHs (Neff 1979, 1982b). An unusually high prevalence of oral, dermal, and hepatic neoplasms have been observed in bottom-dwelling fish from polluted sediments containing grossly-elevated PAH levels (Couch and Harshbarger 1985). PAH compounds have damaged chromosomes in cytogenetic tests, have produced mutations in mammalian cell culture systems, and have induced DNA repair synthesis in human fibroblast cultures (EPA 1980). While some PAHs are potent mutagens and carcinogens, others are less active or suspected carcinogens. Some, especially those of biological origin, are probably not carcinogens (Jackim and Lake 1978). Certain lower molecular weight, noncarcinogenic PAHs, at environmentally realistic levels, were acutely toxic to aquatic organisms, or produced deleterious sublethal

responses (Neff 1985). However, few generalizations can be made about the class of PAH compounds because of the extreme variability in toxicity and physicochemical properties of PAHs and their various effects on individual species (Lee and Grant 1981).

PAHs are widely-distributed in the environment, almost ubiquitous, and have been detected in animal and plant tissues, sediments, soils, air, surface water, drinking water, industrial effluents, ambient river water, well water, and groundwater (EPA 1980). Man probably has always been exposed to PAHs from the natural background level in soils and plants (Harrison et al. 1975); avoiding exposure to nanogram quantities of these substances on a daily basis is now considered essentially impossible for all living resources (Dipple 1985). Ever since benzo(a)pyrene was recognized as a carcinogen at the beginning of this century, the presence of it and of other PAHs in the environment has received continuous attention. As one consequence, many reviews have been published on ecological and toxicological aspects of PAH in the environment, with special reference to their carcinogenic properties.[1]

In this report, I summarize selected data on environmental aspects of PAHs, emphasizing PAH effects to aquatic and wildlife resources. This brief review is part of a continuing series prepared in response to informational requests from environmental specialists of the U. S. Fish and Wildlife Service.

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[1] Harrison et al. (1975); Barnett (1976); Suess (1976); Gelboin and Ts'o (1978a, 1978b, 1981); Jackim and Lake (1978); Jones and Freudenthal (1978); Lo and Sandi (1978); Jones and Leber (1979); Neff (1979, 1982a, 1982b, 1985); Tsang and Griffin (1979); Bjorseth and Dennis (1980); EPA (1980); Cooke and Dennis (1981, 1983, 1984); Futoma et al. (1981); Lee and Grant (1981); Pucknat (1981); Sims and Grover (1981); Stegemen (1981); Cooke et al. (1982); Richards and Jackson (1982); Couch et al. (1983); Edwards (1983); Grimmer (1983); Quaghebeur et al. (1983); Sims and Overcash (1983); Couch and Harshbarger (1985); Harvey (1985); Johnson et al. (1985); Sugimura (1986).

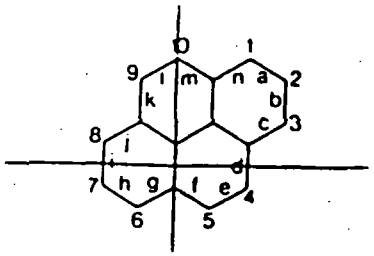
ENVIRONMENTAL CHEMISTRY, SOURCES, AND FATE

PROPERTIES

Polycyclic aromatic hydrocarbons (PAHs), also known as polynuclear aromatic hydrocarbons (PNAs) and polycyclic organic matter (POM), are composed of hydrogen and carbon arranged in the form of two or more fused benzene rings in linear, angular, or cluster arrangements, which may or may not have substituted groups attached to one or more rings (Sims and Overcash 1983). In some cases, the newly defined substituted PAH has strikingly greater toxicological effects than does the parent compound (Cooke and Dennis 1984). The nomenclature of PAH compounds has been ambiguous in the past due to different peripheral numbering systems. The currently accepted nomenclature is shown in Figure 1.

Of major environmental concern are mobile PAHs that vary in molecular weight from 128.16 (naphthalene, $C_{10}H_8$) to 300.36 (coronene, $C_{24}H_{12}$). Higher molecular weight PAHs are relatively immobile because of their large molecular volumes and their extremely low volatility and solubility. Among the mobile forms are thousands of compounds that differ in the number and position of aromatic rings, and in the position of substituents on the basic ring system. The lower molecular weight unsubstituted PAH compounds, containing 2 to 3 rings, such as naphthalenes, fluorenes, phenanthrenes, and anthracenes (Figure 2), have significant acute toxicity to some organisms, whereas the higher molecular weight 4- to 7-ring aromatics do not. However, all known PAH carcinogens, cocarcinogens, and tumor producers are in the high molecular weight PAH group (Figure 3).

Physical and chemical characteristics of PAHs generally vary with molecular weight. With increasing molecular weight, aqueous solubility decreases, and melting point, boiling point, and the log Kow (octanol/water partition coefficient) increases (Table 1), suggesting increased solubility in fats, a decrease in resistance to oxidation and reduction, and a decrease in vapor pressure. Accordingly, PAHs of different molecular weight vary substantially in their behavior and distribution in the environment and in



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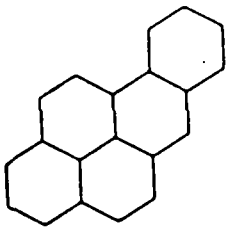

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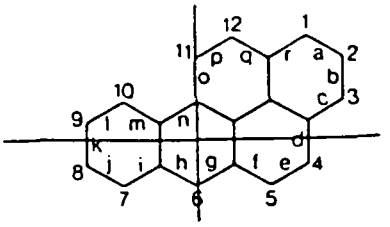

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Figure 1. Nomenclature of PAHs (modified from Lee and Grant 1981, and Grimmer 1983). The PAH formula is oriented so that the greatest number of rings are in a horizontal row and a maximum number of rings are above and to the right of the horizontal row. The first carbon atom that belongs to the uppermost ring and is not engaged in ring fusion with another ring is given the number C-1; numbering continues in a clockwise direction omitting those carbon atoms which do not carry a hydrogen atom. The bond between C-1 and C-2 is designated as side "a"; other peripheral sides continue in clockwise direction in alphabetical order. Examples are: (1) pyrene (correctly oriented, numbered, and lettered), (2) benzo(a)pyrene (not oriented correctly), (3) benzo(a)pyrene (correctly oriented, numbered, and lettered).

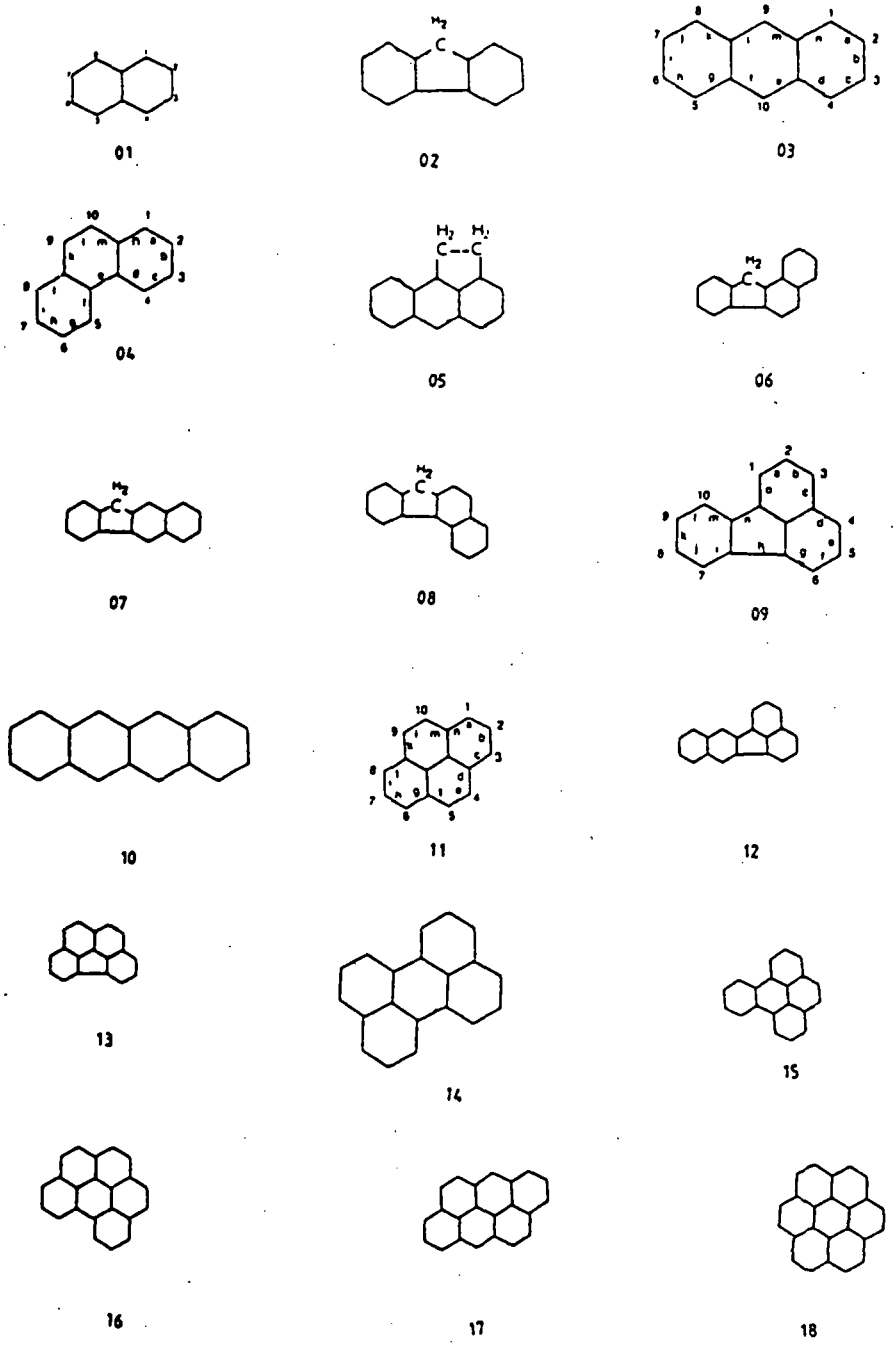

Figure 2. Ring structures of representative noncarcinogenic PAHs (modified from Lee and Grant 1981, and Neff 1985). The numbering and lettering system for several PAHs is also given. Compounds are: (1) naphthalene, (2) fluorene, (3) anthracene, (4) phenanthrene, (5) aceanthrylene, (6) benzo(a)fluorene, (7) benzo(b)fluorene, (8) benzo(c)fluorene, (9) fluoranthene, (10) naphthacene, (11) pyrene, (12) benzo(k)fluoranthene, (13) benzo(g,h,i)fluoranthene, (14) perylene, (15) benzo(e)pyrene, (16) benzo(g,h,i)perylene, (17) anthanthrene, (18) coronene.

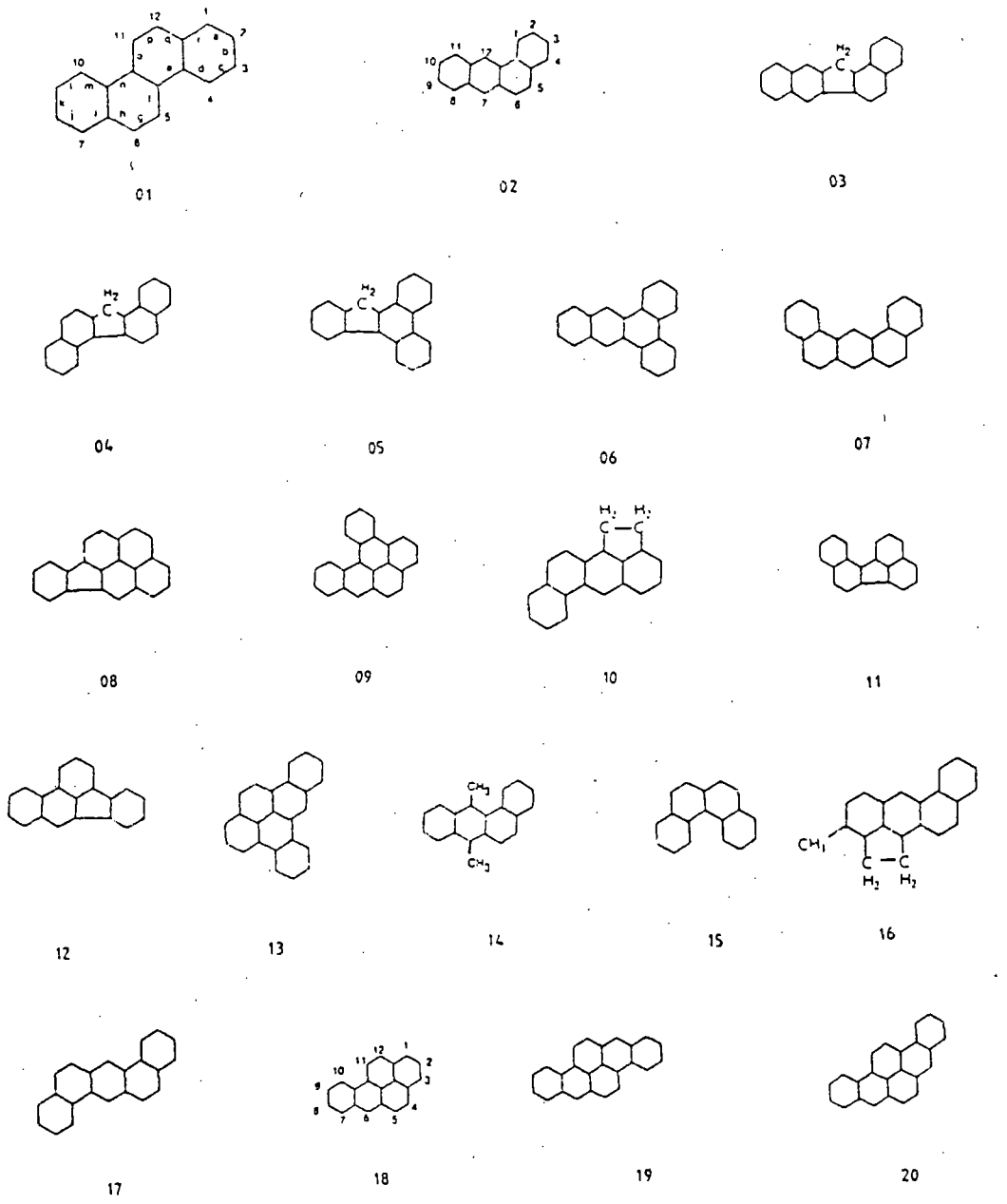

Figure 3. Ring structures of representative tumorogenic, cocarcinogenic, and carcinogenic PAHs (modified from Lee and Grant 1981). The numbering and lettering system for several PAHs is also given. Compounds are: (1) chrysene, (2) benz(a)anthracene, (3) dibenzo(a,h)fluorene, (4) dibenzo(a,g)fluorene, (5) dibenzo(a,c)fluorene, (6) dibenz(a,c)anthracene, (7) dibenz(a,j)anthracene, (8) indeno (1,2,3-cd) pyrene, (9) dibenzo(a,l)pyrene, (10) cholanthrene, (11) benzo(j)fluoranthene, (12) benzo(b)fluoranthene, (13) dibenzo(a,e)pyrene, (14) dimethylbenz(a)anthracene, (15) benzo(c)phenanthrene, (16) 3-methylcholanthrene, (17) dibenz(a,h)anthracene, (18) benzo(a)pyrene, (19) dibenzo(a,h)pyrene, (20) dibenzo(a,i)pyrene. Compounds 1 to 9 are weakly carcinogenic, cocarcinogenic, or tumorogenic; compounds 10 to 13 are carcinogenic; compounds 14 to 20 are strongly carcinogenic.

Table 1. Some physical and chemical properties of selected PAHs.

Compound	Number of rings	Approximate molecular weight	Melting point (°C)	Solubility in water (mg/l)	log Kow
Naphthalene	2	128	80	30.0	3.37
Anthracene	3	178	216	0.07	4.45
Benz(a)anthracene	4	228	158	0.014	5.61
Benzo(a)pyrene	5	252	179	0.0038	6.04
Benzo(g,h,i)perylene	6	276	222	0.00026	7.23

their biological effects. Additional and more comprehensive data on the physical and chemical properties of PAHs are given in Barnett (1976), Lo and Sandi (1978), Neff (1979, 1985), EPA (1980), Futoma et al. (1981), Lee and Grant (1981), Pucknat (1981), Edwards (1983), Grimmer (1983), Sims and Overcash (1983), and Whitehouse (1985).

SOURCES

About 43,000 metric tons of PAHs are discharged into the atmosphere each year, and another 230,000 tons enter aquatic environments (Table 2). PAHs are ubiquitous in nature as a consequence of synthesis in terrestrial vegetation, microbial synthesis, and volcanic activity, but quantities formed by these natural processes are small in comparison with those produced from forest and prairie fires and anthropogenic sources (Barnett 1976; Suess 1976; Lo and Sandi 1978; Neff 1979, 1985; EPA 1980; Lee and Grant 1981; Pucknat 1981; Edwards 1983; Grimmer 1983; Sims and Overcash 1983). Anthropogenic activities associated with significant production of PAHs include: coke production in the iron and steel industry; catalytic cracking in the petroleum industry; the manufacture of carbon black, coal tar pitch, and asphalt; heating and power generation; controlled refuse incineration; open burning; and emissions from internal combustion engines used in transportation. Thus, the formation of PAHs in the environment is due to an endogenous synthesis by microorganisms,

Table 2. Major sources of PAHs in atmospheric and aquatic environments (modified from Lo and Sandi 1978; Neff 1979; Edwards 1983; Sims and Overcash 1983).

Ecosystem and sources	Annual input, in metric tons
ATMOSPHERE	
Total PAHs	
Forest and prairie fires	19,513
Agricultural burning	13,009
Refuse burning	4,769
Enclosed incineration	3,902
Heating and power	2,168
Benzo(a)pyrene	
Heating and power	
Worldwide	2,604
USA only	475
Industrial processes (mostly coke production)	
Worldwide	1,045
USA only	198
Refuse and open burning	
Worldwide	1,350
USA only	588
Motor vehicles	
Worldwide	45
USA only	22
AQUATIC ENVIRONMENTS	
Total PAHs	
Petroleum spillage	170,000
Atmospheric deposition	50,000
Wastewaters	4,400
Surface land runoff	2,940
Biosynthesis	2,700
Total benzo(a)pyrene	700

algae, and macrophytes which provide natural background, and to a second process which is connected to man-controlled high-temperature (>700 °C) pyrolysis of organic materials, to open burning, and to natural volcanic activities. The discovery in fossil fuels of complex mixtures of PAHs spanning a wide range of molecular weights has led to the conclusion that, given sufficient time (i.e., millions of years), pyrolysis of organic materials at temperatures as low as 100 to 150 °C can also lead to production of PAHs (Neff 1985).

Forest and prairie fires release much greater amounts of PAHs to the atmosphere than does fossil fuel burning. Nearly all of the airborne PAHs produced by flame pyrolysis are associated with the particulate fraction produced during combustion, and these are significantly modified by the chemical composition of the fuel, the pyrolysis temperature, the duration of exposure to elevated temperatures, and to other factors (Neff 1979; Edwards 1983). In one study, a PAH profile was established for a series of laboratory fires simulating the prescribed burning of pine needle litter (McMahon and Tsoukalas 1978). Heading fires (moving with wind) produced more total particulate matter than backing fires (moving against wind), but backing fires produced significantly higher amounts of PAHs, with the actual amounts formed dependent on fuel loading and the residence time of combustible gases in the burning zone. Emission factors for benzo(a)pyrene varied from 238 to 3,454 ug/kg in backing fires and 38 to 97 ug/kg in heading fires.

PAHs present in the atmosphere enter rain as a result of in-cloud and below-cloud scavenging (van Noort and Wondergem 1985). Total PAHs deposited on land and water are almost equivalent to PAH content in rainfall; significant quantities of PAHs are found in presumed pollution-free areas, indicating the importance of rain in transport and distribution of PAHs (Quaghebeur et al. 1983).

PAHs may reach aquatic environments in domestic and industrial sewage effluents, in surface runoff from land, from deposition of airborne particulates, and especially from spillage of petroleum and petroleum products into water bodies (Jackim and Lake 1978; Lake et al. 1979; Neff 1979; EPA 1980; Martens 1982; Boehm and Farrington 1984; Hoffman et al. 1984; Prahl et al. 1984). The majority of PAHs entering aquatic environments remains close to sites of deposition, suggesting that lakes, rivers, estuaries, and coastal marine environments near centers of human populations are the primary repositories of aquatic PAHs (Neff 1979). Large variations in aquatic PAH contents were evident due to localized source inputs and physicochemical conditions. For example, urban runoff from stormwater and highways to Narragansett Bay, Rhode Island, accounted for 71% of the total inputs for higher molecular weight PAHs, and 36% of the total PAHs (Hoffman et al. 1984). More than 30% of all combustion-derived PAHs in coastal sediments of Washington State is supplied by riverine transport of suspended particulate materials, while direct atmospheric input accounts for a maximum of 10% (Prahl et al. 1984). In contrast, concentrations of PAHs in sediments from the

vicinity of Georges Bank, off the US northeastern coast, varied from 1 to 100 ug/kg dry weight, and were directly related to total organic carbon, silt, and clay contents in sediments; combustion-derived PAHs dominated at the higher concentrations, while lower levels were often associated with a fossil fuel origin (Boehm and Farrington 1984).

Discharge water from hydrostatic testing of natural gas pipelines is a significant source of PAH loading into aquatic environments, contributing as much as 32,000 ug PAHs/l of discharge water, mostly as naphthalenes (Eiceman et al. 1984). More than 25 PAHs, primarily anthracenes and pyrenes, were detected in pipeline residues on inner walls of natural gas pipelines at concentrations up to 2,400 ug/m^2 of inner surface; the same compounds may be reasonably expected in aqueous wastes from pipeline maintenance (Eiceman et al. 1985). Release of these, or similar, discharge waters directly into aquatic environments will result in contamination similar to that caused by oil spills; however, these sites for pollution may occur in locations far distant from oil production and refinery activities (Eiceman et al. 1984). PAHs are also present in tap water at concentrations of 0.1 to 1.0 ng/l, primarily as mono- and dichlorinated derivitives of naphthalene, phenanthrene, fluorene, and fluoranthene (Shiraishi et al. 1985). The presence of PAHs and chlorinated PAHs in tap water indicates the reaction of PAHs with chlorine; however, their significance to human health and to aquatic biota is unknown.

FATE

Concern about PAHs in the environment is due to their persistence and to the fact that some are known to be potent mammalian carcinogens, although environmental effects of most noncarcinogenic PAHs are poorly understood (Neff 1985). Prior to 1900, a natural balance existed between the production and the degradation of PAHs. Synthesis of PAHs by microorganisms and volcanic activity and production by man-made high temperature pyrolytic reactions and open burning seemed to be balanced by PAH destruction via photodegradation and microbial transformation. With increased industrial development and increased emphasis of fossil fuels as energy sources, the balance has been disturbed to the extent that PAH production and introduction into the environment greatly exceeds known PAH removal processes (Suess 1976; Sims and Overcash 1983).

When released into the atmosphere, PAH compounds will become associated with particulate materials. Their residence time in the atmosphere and transport to different geographic locations are governed by particle size, meteorological conditions, and atmospheric physics. The highly reactive PAHs photodecompose readily in the atmosphere by reaction with ozone and various oxidants; degradation times range from several days to six weeks for PAHs adsorbed onto particulates <1 um in diameter (in the absence of rainfall) to <1 day to several days for those adsorbed to larger particles (Suess 1976).

Smaller atmospheric particulates containing PAHs are easily inhaled (Lee and Grant 1981), and may pose special problems, as yet unevaluated, for airborne organisms such as birds, insects, and bats. Photooxidation, one of the most important processes in the removal of PAHs from the atmosphere, can also produce reaction products that are carcinogenic or mutagenic, although little is known of their persistence (Edwards 1983). One of the more common photooxidation reactions of PAHs is the formation of endoperoxides that ultimately undergo a series of reactions to form quinones (Edwards 1983). Various parameters may modify chemical and photochemical transformation of PAHs in the atmosphere, including light intensity, concentration of gaseous pollutants (O_3, NO_x, SO_x), and chemicophysical characteristics of particulates or substrates into which the PAHs are adsorbed; depending on these variables, the half-life of benzo(a)pyrene in the atmosphere varies from 10 minutes to 72 days (Valerio et al. 1984). Atmospheric PAHs are transported over relatively long distances from industrial areas and from natural forest and prairie fires (Edwards 1983); however, sites nearer urban centers have much higher PAH deposition rates than more rural areas (Hites and Gschwend 1982).

Much of the PAHs released into the atmosphere eventually reaches the soil by direct deposition or by deposition on vegetation. The PAHs may be adsorbed or assimilated by plant leaves before entering the animal food chain, although some adsorbed PAHs may be washed off by rain, chemically oxidized to other products, or returned to the soil as the plants decay. PAHs assimilated by vegetation may be translocated, metabolized, and possibly photodegraded within the plant. In some plants growing in highly contaminated areas, assimilation may exceed metabolism and degradation, resulting in an accumulation in plant tissues (Edwards 1983).

In water, PAHs may either evaporate, disperse into the water column, become incorporated into bottom sediments, concentrate in aquatic biota, or experience chemical oxidation and biodegradation (Suess 1976). The most important degradative processes for PAHs in aquatic systems are photooxidation, chemical oxidation, and biological transformation by bacteria and animals (Neff 1979). Most PAHs in aquatic environments are associated with particulate materials; only about 33% are present in dissolved form (Lee and Grant 1981). PAHs dissolved in the water column will probably degrade rapidly through photooxidation (EPA 1980), and degrade most rapidly at higher concentrations, at elevated temperatures, at elevated oxygen levels, and at higher incidences of solar radiation (McGinnis and Snoeyink 1974; Suess 1976; Bauer and Capone 1985). The ultimate fate of those PAHs that accumulate in sediments is believed to be biotransformation and biodegradation by benthic organisms (EPA 1980). PAHs in aquatic sediments, however, degrade very slowly in the absence of penetrating radiation and oxygen (Suess 1976), and may persist indefinitely in oxygen-poor basins or in anoxic sediments (Neff 1979). PAH degradation in aquatic environments occurs at a slower rate than that in the atmosphere (Suess 1976), and the cycling of PAHs in aquatic environments, as is true for other ecological systems, is poorly understood (Neff 1979).

Animals and microorganisms can metabolize PAHs to products that may ultimately experience complete degradation. The degradation of most PAHs is not completely understood. Those in the soil may be assimilated by plants, degraded by soil microorganisms, or accumulated to relatively high levels in the soil. High PAH concentrations in soil can lead to increased populations of microorganisms capable of degrading the compounds. Of equal importance to PAH cycling dynamics is the physical state of the PAH, i.e., whether in vapor phase or associated with particles such as flyash. Particles may increase or decrease the susceptibility of PAHs to degradation, depending on the PAH and particles involved (Edwards 1983).

PAHs can be taken into the mammalian body by inhalation, skin contact, or ingestion, although they are poorly absorbed from the gastrointestinal tract. The main routes of elimination of PAHs and their metabolites include the hepatobiliary system and the gastrointestinal tract (Sims and Overcash 1983). In mammals, an enzyme system variously known as the cytochrome P-450-dependent mixed-function oxidase, mixed-function oxidase, mixed-function oxygenase, aryl hydrocarbon hydroxylase, or drug metabolizing system, is responsible for initiating the metabolism of various lipophilic organic compounds, including PAHs. The primary function of this system is to render poorly water soluble lipophilic materials more water soluble, and therefore more available for excretion. Some PAHs are transformed to intermediates, which are highly toxic, mutagenic, or carcinogenic to the host. Oxidative metabolism of PAHs in this system proceeds via high electrophilic intermediate arene oxides, some of which bind covalently to cellular macromolecules such as DNA, RNA, and protein. Most authorities agree that metabolic activation by the mixed-function oxidase system is a necessary prerequisite for PAH-induced carcinogenesis and mutagenesis (Neff 1979). This enzyme system is known to be present in rodent tissues, and human liver, skin, placenta, fetal liver, macrophages, lymphocytes, and monocytes (Lo and Sandi 1978). Studies with rodents have shown that the mixed-function oxidase system can convert PAHs to various hydroxylated derivatives including phenols, quinones, and epoxides, and can also activate PAHs to produce carcinogenic metabolites (Lo and Sandi 1978). Fish and most crustaceans tested to date possess the enzymes necessary for activation (Statham et al. 1976; Varanasi et al. 1980; Fabacher and Baumann 1985), but some molluscs and other invertebrates are unable to efficiently metabolize PAHs (Jackim and Lake 1978; Varanasi et al. 1985). Although many aquatic organisms possess the requisite enzyme systems for metabolic activation of PAHs, it is not certain in most cases whether these enzymes produce the same metabolites as those produced by mammalian enzymes (Neff 1979).

PAHs are metabolized by liver mixed-function oxidases to epoxides, dihydrodiols, phenols, and quinones. The intermediate metabolites have been identified as the mutagenic, carcinogenic, and teratogenic agents (Sims and Overcash 1983). The activation mechanisms occur by hydroxylation or

production of unstable epoxides of PAHs which damage DNA, initiating the carcinogenic process (Jackim and Lake 1978). Metabolic formation of bay region diol epoxides represents an important pathway by which PAHs are activated to carcinogens (Figure 4). Such metabolic activation proceeds via initial formation of the dihydrodiol with the bay region double bond, followed by subsequent oxidation of the dihydrodiol to the bay region diol epoxide (Sims and Overcash 1983). Active epoxides may be converted to less toxic products by various enzymatic and other reactions (Neff 1979). In the case of benzo(a)pyrene, the "ultimate carcinogen" (7 beta, 8 alpha-dihydroxy-,7,8,9,10 tetrahydrobenzo(a)pyrene- 9 alpha, 10 alpha-epoxide) reacts with the guanine of RNA and DNA, the linkage taking place between the C-10 atom of benzo(a)pyrene and the C-2 amino group of guanine (Grimmer 1983; Dipple 1985; Figure 4). Additional information on actual and theoretical mechanisms involved in the metabolic activation of PAHs are given in Cavalieri et al. (1978, 1980), Bjorseth and Dennis (1980), Herd and Greene (1980), Cooke and Dennis (1981), Sims and Grover (1981), Grimmer (1983), Szentpaly (1984), Harvey (1985), and Yan (1985).

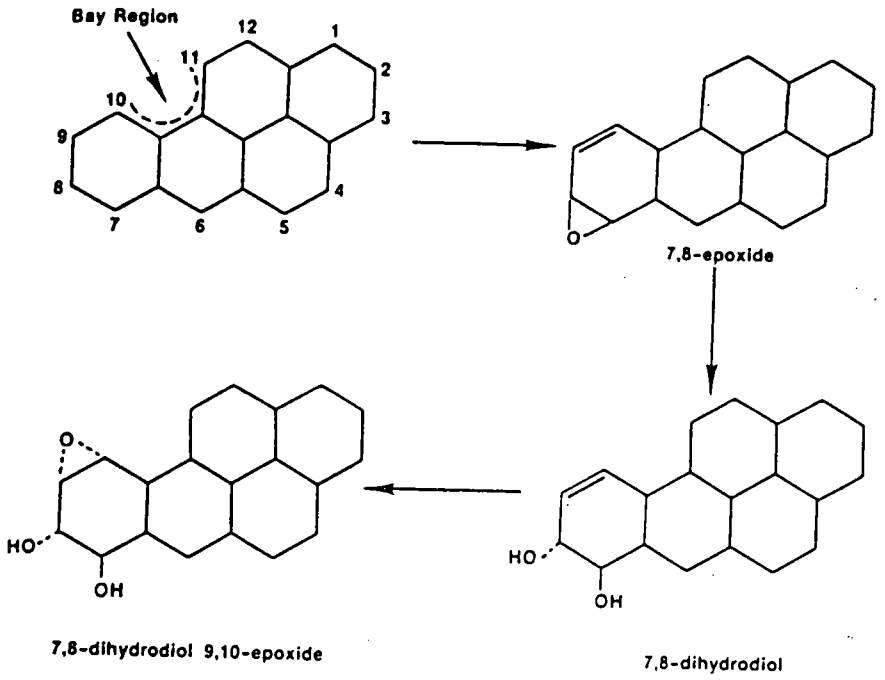

Figure 4. The bay region dihydrodiol epoxide route of benzo(a)pyrene (modified from Dipple 1985).

BACKGROUND CONCENTRATIONS

GENERAL

PAHs are ubiquitous in the environment. In nonbiological materials, concentrations are elevated in the vicinity of urban industrialized locales, and from areas of significant wood burning activities such as forest fires and residential home heating. Terrestrial vegetation and aquatic invertebrates can accumulate significant concentrations of PAHs, possibly due to inefficient or missing mixed-function oxidase systems. Fish do not appear to contain grossly elevated PAH residues; this may be related to their efficient degradation system. At present, data are lacking or unavailable on PAH background concentrations in natural populations of birds and other wildlife --although it seems unlikely that significant accumulations will occur. Some investigators have shown that aquatic invertebrates, fish, and amphibians collected from areas of high sediment PAH content show elevated frequencies of hyperplasia and neoplasia (Rose 1977; Mix 1982; Black 1983; Malins et al. 1984, 1985a, 1985b; Black et al. 1985; Baumann et al., in press), and, recently, that hepatic carcinoma has been induced in rainbow trout (Salmo gairdneri) by benzo(a)pyrene through dietary and intraperitoneal injection routes (Hendricks et al. 1985).

More comprehensive information on PAH background levels in various biological and nonbiological compartments is given in Lo and Sandi (1978), Neff (1979, 1985), Pucknat (1981), Edwards (1983), Grimmer (1983), and Sims and Overcash (1983).

NONBIOLOGICAL SAMPLES

Total PAH levels in air are usually much higher in winter than in summer, higher in urban communities than in rural areas (Table 3; Grimmer 1983), and appear to be related primarily to the weight of total suspended particulates in the atmosphere (Hites and Gschwend 1982; Greenberg et al. 1985; Srivastava et al. 1985; Ang et al. 1986). PAH levels in precipitation are significantly higher in winter than in summer, primarily due to emissions from household heating (Quaghebeur et al. 1983; van Noort and Wondergem 1985). Among

Table 3. PAH concentrations in selected nonbiological materials.

Material (units), and other variables	Concentration	Reference[a]
AIR (ng/m^3)		
USA cities, 1959, total PAHs		
Detroit	95.1	EPA 1980
Birmingham	63.4	
Nashville	60.6	
New Orleans	33.6	
Los Angeles	31.8	
Atlanta	26.3	
San Francisco	13.7	
Sydney, Australia		
Winter	8.2	Barnett 1976
Summer	0.6	
USA cities, 1971-1977		
Benzo(a)perylene = BaPER	0.2-9.2	EPA 1980
Benzo(e)pyrene = BeP	0.9-4.6	
Benzo(k)fluoranthene = BkFL	0.03-1.3	
Pyrene = PYR	0.18-5.2	
Coronene = COR	0.2-6.4	
Perylene = PER	0.01-1.2	
Anthracene = A	0.07-0.3	
Naphthalene = NA	Max. 0.4	
Benz(a)anthracene = BaA	Max. 4.6	
Indeno(1,2,3-cd)pyrene = IP	Max. 1.3	
Steel mill, Ontario, Canada, 1971-1979		
Station 0.8 km distant		
Benzo(a)pyrene = BaP	9.4 (Max. 110.0)	Potvin
BkFL	8.9 (Max. 142.0)	et al. 1981
Fluoranthene = FL	7.0 (Max. 43.3)	
PER	9.1 (Max. 106.0)	
Benzo(g,h,i)perylene = BghiPER	13.7 (Max. 90.0)	
Station 2.8 km distant		
BaP	0.4 (Max. 7.9)	
BkFL	0.7 (Max. 5.1)	
FL	1.1 (Max. 4.8)	
PER	0.7 (Max. 9.1)	
BghiPER	1.4 (Max. 8.5)	
Benzo(a)pyrene = BaP		
Urban areas	0.1-61.0	Edwards 1983
Downwind from coal gasification plant, Yugoslavia	Max. 80.0	

Table 3. (Continued)

Material (units), and other variables	Concentration	Reference[a]
Urban areas		
1966	3.2	EPA 1980
1970	2.1	
1976	0.5	
Rural areas	0.01-1.9	Edwards 1983
Rural areas		
1966	0.4	EPA 1980
1976	0.1	
SOILS		
Near M6 Motorway, Lancaster, UK (maximum deposition rate, ng/m^2/week)		
Distance from roadway		
3.8 meters		
A	2,300	Johnston and
FL	15,200	Harrison 1984
BaA	5,800	
Benzo(b)fluoranthene = BbFL	7,300	
BkFL	2,800	
BaP	4,900	
9.0 - 47 meters		
A	420	
FL	1,700	
BaA	260	
BbFL	690	
BkFL	470	
BaP	290	
Vicinity slash burn site, Oregon (g/ha)		
0-2 cm depth		
Preburn		
Phenanthrene = PHEN	0.5	Sullivan
FL	0.6	and Mix 1985
103 days postburn		
PHEN	9.8	
FL	3.6	
365 days postburn		
PHEN	ND	
FL	0.8	
2-5 cm depth		

Table 3. (Continued)

Material (units), and other variables	Concentration	Reference[a]
105 days postburn		
PHEN	1.3	
FL	0.3	
365 days postburn		
PHEN	ND	
FL	ND	
BaP (ug/kg)		
Rural areas	0.4	Barnett 1976
Industrial areas	400.0	
Nonpolluted areas	up to 1,000	Edwards 1983
Near known sources	>100,000	
Near coal-tar pitch disposal site, Germany	650,000	Lee and Grant 1981
Near recreation area, USSR	0.4	Harrison
Forest soil	1.5-4.0	et al. 1975
LITTER		
Forest, Oregon (g/ha)		
3 days postburn		
PHEN	603	Sullivan and
FL	245	Mix 1985
32 days postburn		
PHEN	ND	
FL	ND	
Coniferous trees (ug/kg)		
BghiPER	42	Thomas
BaP	51	et al. 1984
IP	47	
FL	164	
SEDIMENTS (ug/kg)		
Buffalo River, near Buffalo, NY		
Sediments		
BaA	7,300	Black 1983
Chrysene = CHRY	4,300	
BbFL	3,500	

Table 3. (Continued)

Material (units), and other variables	Concentration	Reference[a]
BaP	4,500	
Dibenz(a,h)anthracene = DBA	1,000	
IP	4,400	
Sediment extracts		
BaA	16,000	
CHRY	14,000	
BbFL	13,900	
BaP	15,400	
DBA	3,300	
IP	12,300	
Cayuga Lake, Ithaca, NY, 1978		
Total PAHs		
Within marinas	4,600-13,900	Heit 1985
Deepwater	1,260-2,500	
Near power plant	104-6,800	
FL		
Within marinas	1,700	
Deepwater	285	
Near power plant	8-1,000	
Penobscot Bay, Maine		
Total PAHs	286-8,794	Johnson
PHEN	17-252	et al. 1985
A	ND-49	
FL	156-3,700	
Pyrene = PYR	16-539	
BaA	14-540	
CHRY	9-578	
BbFL	17-1,000	
BkFL	14-696	
BaP	10-540	
DBA	2-120	
BghiPER	23-641	
IP	9-228	
Casco Bay, Maine, total PAHs	215-14,425	
Charles River, Mass., total PAHs	87,000-120,000	
Boston Harbor, Mass., total PAHs	8,500	
New Bedford Harbor, Mass., total PAHs	63,000	
Lake Erie, total PAHs	530-3,750	
Adirondack Lakes, total PAHs	4,070-12,807	

Table 3. (Continued)

Material (units), and other variables	Concentration	Reference[a]
Alaska, total PAHs	5-113	
Tamar estuary, UK, total PAHs	4,900	
Southampton estuary, UK, total PAHs	91,000-1,791,000	
Severn estuary, UK, total PAHs	1,600-25,700	
Monaco, total PAHs	5,200-12,100	
Gulf of Finland, total PAHs	437	
Norway, total PAHs	284-99,452	
Walvis Bay, Africa, total PAHs	68	
Amazon River system, total PAHs	ND-544	
SEWAGE		
Waters, worldwide, total PAHs(ug/l)	100-500	Lee and Grant 1981
Sludge, total PAHs		
United Kingdom, 12 sites, (ug/kg)		
Fresh weight	80-1,760	McIntyre et al. 1981
Dry weight	200-50,300	
Texas, Reese Air Force Base		
Effluent lagoon (ug/kg fresh weight)		
PER	300.0	Rose 1977
PYR	5.8	
FL	5.7	
BaA	1.4	
CHRY	1.3	
BaP	0.5	
BeP	0.2	
A	0.2	
MOTOR OILS (ug/l)		
Unused		
BaP	115	Pasquini and Monarca 1983
CHRY	56	
PER	11	
Used		
BaP	1,382	
CHRY	10,170	
PER	1,024	

Table 3. (Continued)

Material (units), and other variables	Concentration	Reference[a]
GROUNDWATER (ug/l)		
Worldwide		
Total PAHs	0.01-0.05	Lee and Grant 1981
Total PAHs	0.045-0.51	Harrison et al. 1975
Carcinogenic PAHs	0.00-0.081	
Germany		
Total PAHs	0.04	EPA 1980
Carcinogenic PAHs	0.003	
Champaign, Illinois		
Total PAHs	0.007	
Carcinogenic PAHs	0.003	
Elkhart, Indiana		
Total PAHs	0.02	
Carcinogenic PAHs	0.004	
DRINKING WATER (ug/l)		
USA, total PAHs	0.015	Lee and Grant 1981
Europe, total PAHs	0.04-0.06	
Monongehala River, Pittsburgh, PA		
Untreated		
Total PAHs	0.6	EPA 1980
Carcinogenic PAHs	0.14	
Treated		
Total PAHs	0.003	
Carcinogenic PAHs	0.002	
Ohio River, Wheeling, WV		
Untreated		
Total PAHs	1.59	
Carcinogenic PAHs	0.57	
Treated		
Total PAHs	0.14	
Carcinogenic PAHs	0.011	
Lake Winnebago, Appleton, WI		
Untreated		
Total PAHs	0.007	
Carcinogenic PAHs	0.002	

Table 3. (Concluded)

Material (units), and other variables	Concentration	Reference[a]
Treated		
Total PAHs	0.006	
Carcinogenic PAHs	0.002	
SURFACE WATER (ug/l)		
Worldwide		
Low level contamination	0.05-0.25	Lee and
Medium polluted	0.2-1.0	Grant 1981
Germany, Rhine River		
Total PAHs	1.12	EPA 1980
Carcinogenic PAHs	0.49	
Thames River, UK		
Total PAHs	0.5-1.33	
Carcinogenic PAHs	0.18-0.56	

[a]Each reference applies to data in the same row and in the rows that immediately follow for which no reference is indicated.

industrial sources, the production of metallurgical coke is the single most significant source of atmospheric PAHs in Ontario, Canada. Coke production in 1977 represented about 52% of all PAH emissions from Ontario sources versus about 46% formed as a result of forest fires (Potvin et al. 1981). Beyond 2 km distant from the coke point source, PAH concentrations in air were typical of those measured in major urban nonindustrialized areas (Table 3; Potvin et al. 1981). A variety of PAHs have been detected in ambient air in the USA and elsewhere. Benzo(a)pyrene, because of its carcinogenic properties, has been monitored extensively, and has frequently been used as an indicator of PAHs (EPA 1980). In general, total PAHs in air is about 10X higher than benzo(a)pyrene levels, although this relation is extremely variable (Lee and Grant 1981). Benzo(a)pyrene levels, like total PAHs, were higher in winter than summer, probably due to residential and industrial heating; air levels in urban areas with coke ovens were 40% to 70% higher than in cities without coke ovens, but this may be related to higher industrial emissions in those cities (Lee and Grant 1981). In one case, benzo(a)pyrene levels in air from the center of a remote mountain community in Colorado were several times higher than what is usually found in U.S. metropolitan areas, and was attributed to extensive residential wood burning (Murphy et al. 1982). Average concentrations of benzo(a)pyrene in urban air Nationwide declined from 3.2 ng/m^3 in 1966 to 0.5 ng/m^3 in 1978, an 80% decrease (Lee and Grant 1981). These decreases are believed to be due primarily to decreases in coal consumption for commercial and residential heating, improved disposal of solid wastes, and restrictions on open burning (EPA 1980).

A major source of PAHs in soils and soil litter is from emissions and deposition from forest fires. In a controlled burn study, Sullivan and Mix (1985) showed that lower molecular weight PAHs, such as phenanthrene and fluorene, which had been deposited in soil litter, degraded to nondetectable levels within 2 years after burning. Higher molecular weight PAHs such as benzo(k)fluorene, benzo(a)pyrene, benzo(g,h,i)perylene, perylene, and indeno(1,2,3-cd)pyrene, were more persistent in litter, decreasing after 5 years to about 20% of initial deposition. Although movement into the top 2 cm of the soil profile was initially more pronounced for lower molecular weight PAHs, all compounds appeared to reach equilibrium between litter and soil on the basis of organic content within one year postburn. Differential persistence and fate of PAHs on slash burn sites is explained by solubility, Kow, and other physicochemical properties (Sullivan and Mix 1985). PAHs from vehicle emissions constitute a minor, but measurable, source of soil PAHs (Table 3). The majority of highway-derived PAHs appears to be deposited within 3.8 m of the road, but the influence of the highway may extend to nearly 70 m (Johnston and Harrison 1984). The use of composted municipal wastes for conditioning of agricultural soils is not recommended, as these contain at least nine identified carcinogenic PAHs (Martens 1982).

Some sediments were found to be highly contaminated with PAHs. Sediments and sediment extracts from the Buffalo River, New York, contained elevated levels of carcinogenic PAHs (1,000-16,000 ug/kg). Brown bullheads (Ictalurus

nebulosus), in response to repeated applications of Buffalo River sediment extracts, showed epidermal hyperplasia and neoplasia when compared to controls (Black 1983). PAH concentrations in sediments from the Great Barrier Reef, Australia, were always <0.8 ug/kg dry weight, except in small areas close to sites frequently visited by power boats; in those instances, total PAH levels exceeded 13,400 ug/kg (Smith et al. 1985). Highest PAH levels measured in sediments of Cayuga Lake, New York, were found in marinas or areas of the lake receiving urban runoff, and were apparently not related to stack emissions from a nearby coal-fired power plant; Heit (1985) believed that stack emissions were either masked by other sources or were atmospherically transported and deposited elsewhere. Coastal and offshore sediments are subject to highly elevated PAH levels from a variety of sources, mostly unknown, relative to preindustrial times (Johnson et al. 1985). For example, PAH levels in sediments of Penobscot Bay, Maine, fell within the range found in sediments near industrialized regions, and were significantly higher than expected for an area previously considered to be uncontaminated (Table 3; Johnson et al. 1985).

Sewage effluents usually contained measurable levels of PAHs, although extreme variability between and among sites is common. For example, during a heavy storm, individual PAH levels in a sewage works may increase more than 100X over a dry weather period (Harrison et al. 1975). Conventional sewage treatment plant processes remove up to 90% of carcinogenic PAHs, and this may be increased to 99% using percolating filters and activated sludge processes (Harrison et al. 1975). Tiger salamanders (Ambystoma tigrinum), collected in 1975 from a 13-ha sewage effluent lagoon at Reese Air Force Base, Texas, showed a remarkably high incidence (53%) of neoplastic and other lesions (Rose 1977). Analysis of sludge composites showed elevated PAH levels, especially perylene; levels of organochlorine and organophosphorus pesticides, nitrosamines, and heavy metals were judged to be nonelevated (Rose 1977).

Careful disposal of used motor oils is warranted, as these contain high quantities of mutagenic and carcinogenic PAHs (Table 3; Pasquini and Monarca 1983).

All but the most heavily contaminated fresh and marine waters contain total PAH concentrations in the part-per-trillion or low part-per-billion range (Table 3; Neff 1982b). A large proportion of the PAH content in water is probably adsorbed onto suspended solids (Harrison et al. 1975). In Lake Michigan, concentrations of total PAHs in the surface microlayer varied from 0.15 to 0.45 ug/l, representing on a relative scale 10^6 times the concentration in air, suggesting that aerosols are a major source of these compounds and that the microlayer is a repository until the PAHs are removed by adsorption and sedimentation (Strand and Andren 1980).

BIOLOGICAL SAMPLES

Carcinogenic PAHs have been extracted from a large variety of fresh plants, including root and leaf vegetables, fruits, grains, and edible mushrooms, as well as from various marine bacteria and phytoplankton under circumstances suggesting that PAHs were present due to local biosynthesis (Suess 1976). Vegetation and soil near known PAH sources are more highly contaminated with PAHs than those collected at greater distances (Edwards 1983). PAH levels in lettuce (Lactuca sativa) grown in Sweden seemed to be directly related to its proximity to local recognized point sources of PAH emitters (Table 4; Larsson and Sahlberg 1982). Washing lettuce with water had little effect on phenanthrene levels, but significantly reduced other PAHs, such as benzo(a)pyrene, benz(a)anthracene, and benzo(g,h,i)perylene by 68% to 87% (Larsson and Sahlberg 1982). Fruits and vegetables grown in polluted atmospheres may contain up to 100X higher levels of total PAHs than those grown in unpolluted environments (EPA 1980; Lee and Grant 1981). PAH concentrations for plants are generally greater on plant surfaces than internal tissues, greater in above ground plant parts than those below ground, and greater in plants with broad leaves (greater surface area) than those with narrow leaves (Edwards 1983). Plants can become contaminated with PAHs through environmental pollution, particularly through deposition from the atmosphere, and also through food processing. For example, the bran portion of milled wheat, as well as finished bran cereal, had a considerably higher PAH content than other fractions or finished products (Lawrence and Weber 1984b). Enrichment of PAHs in plants is associated with deposition of atmospheric particulate matter with relatively small particle sizes; thus, PAH content is usually in the order of humus > mosses > lichens (Thomas et al. 1984). Mosses appear to be good indicators of regional PAH air pollution and have been recommended for this purpose (Herrmann and Hubner 1984). Concentrations of total PAHs in soils, usually the sum of 5 to 20 PAHs, typically exceeded benzo(a)pyrene levels by at least one order of magnitude; however, concentrations of benzo(a)pyrene in vegetation were generally less than those in soil where plants were growing (Edwards 1983).

PAH accumulations in marine molluscs have been reported (Table 4); however, some of these data may be misleadingly low. For example, lengthy cold storage of 10 months can result in loss of volatile PAHs, such as anthracene, in tissues of mussels (Smith et al. 1984); accordingly, background concentrations in these organisms may be underreported. Bivalve molluscs tend to accumulate high PAH levels due to their inability to metabolize and excrete them (Lawrence and Weber 1984a), presumably due to inefficient or missing mixed-function oxidase systems (Sirota and Uthe 1981). Cellular proliferative disorders, resembling neoplastic conditions in vertebrates, were found in mussels with the greatest PAH concentrations: 9.5% vs. 0.7% in control site (Mix 1982). Baseline levels of PAHs in indigenous bivalve molluscs reflected the degree of human onshore activity at the various sample sites, and presumably the level of water contamination; however, little relation was

Table 4. PAH concentrations in field collections of selected biota. Values are shown in ug/kg (ppb) fresh weight (FW), or dry weight (DW).

Taxonomic group, compound, and other variables	Concentration	Reference[a]
ALGAE AND OTHER PLANTS		
Marine algae, Greenland		
Total PAHs	60 FW	Harrison et al. 1975
Marine algae, Benzo(a)pyrene=BaP	Up to 60 DW	Lee and Grant 1981
Freshwater alga,		
Chlorella vulgaris, BaP	10-50 DW	Suess 1976
Bacteria, BaP	2-6 DW	
Moss, Hypnum cupressiforme		
Southern Finland, 1982		
Near center of industrial town		
BaP	110 DW	Herrmann and Hubner 1984
Fluoranthene=FL	250 DW	
Benzo(g,h,i)perylene=BghiPER	90 DW	
Indeno(1,2,3 cd)pyrene=IP	41 DW	
Vegetation		
Total PAHs		
Nonpolluted areas	20-1,000 DW	Edwards 1983
Near known source	25,000 DW	
BaP	0.1-150.0 DW	
Lettuce,		
Lactuca sativa, total PAHs		
Sweden, summer 1980		
Grown near highway		
8-15 m distant	50 FW	Larsson and Sahlberg 1982
15-50 m distant	26 FW	
Near airport, 150-800 m	24 FW	
Aluminum smelter		
0.5-1.5 km distant	654 FW	
2.0-6.5 km	128 FW	
Industrial areas	13 FW	
Residential areas		
Urban	13 FW	
Rural	12 FW	
Seedlings, wheat and rye, BaP	10-20 DW	Suess 1976

Table 4. (Continued)

Taxonomic group, compound, and other variables	Concentration	Reference[a]
INVERTEBRATES		
Rock crab, Cancer irroratus		
Edible portions, 1980		
New York Bight		
Total PAHs	1,600 FW	Humason and
BaP	1 FW	Gadbois 1982
Long Island Sound		
Total PAHs	1,290 FW	
BaP	ND	
American oyster, Crassostrea virginica, soft parts		
South Carolina, 1983, residential resorts		
Total PAHs		
Spring months		
Palmetto Bay	520 FW	Marcus and Stokes 1985
Outdoor Resorts	247 FW	
Fripp Island	55 FW	
Summer months		
Palmetto Bay	269 FW	
Outdoor Resorts	134 FW	
Fripp Island	21 FW	
American lobster, Homarus americanus		
Edible portions, 1980		
New York Bight		
Total PAHs	367 FW	Humason and
BaP	15 FW	Gadbois 1982
Long Island Sound		
Total PAHs	328 FW	
BaP	15 FW	
Softshell clam, Mya arenaria		
Coos Bay, Oregon, 1978-1979		
Soft parts		
Contaminated site		
Total PAHs	555 FW	Mix 1982
Phenanthrene = PHEN	155 FW	
FL	111 FW	
Pyrene = PYR	62 FW	
BaP	55 FW	
Benz(a)anthracene = BaA	42 FW	

Table 4. (Continued)

Taxonomic group, compound, and other variables	Concentration	Reference[a]
Chrysene = CHRY	27 FW	
Benzo(b)fluoranthene = BbFL	12 FW	
Others	<10 FW	
Uncontaminated site		
Total PAHs	76 FW	
PHEN	12 FW	
FL	10 FW	
Others	<10 FW	
Bay mussel, Mytilus edulis		
Oregon, 1979-1980		
Soft parts, total PAHs		
Near industrialized area	106-986 FW	Mix and Schaffer 1983b
Remote site	27-274 FW	
Sea scallop, Placopectin magellanicus		
Baltimore Canyon, east coast USA		
Muscle		
BaA	1 FW	Brown and
BaP	<1 FW	Pancirov 1979
PYR	4 FW	
New York Bight, 1980		
Edible portions		
Total PAHs	127 FW	Humason and
BaP	3 FW	Gadbois 1982
Clam, Tridacna maxima		
Australia, 1980-1982, Great Barrier Reef		
Soft parts, total PAHs		
Pristine areas	<0.07 FW	Smith et al. 1984
Power boat areas	Up to 5 FW	
BaP		
Marine plankton		
Greenland	5 FW	Harrison et al. 1975
Italy	6-21 FW	
France	400 FW	
Worldwide	Up to 400 DW	Lee and Grant 1981
Mussel, Mytilus sp.		
Greenland		
Shell	60 FW	Harrison et al. 1975
Soft parts	18 FW	

Table 4. (Continued)

Taxonomic group, compound, and other variables	Concentration	Reference[a]
Italy		
Shell	11 FW	
Soft parts	130-540 FW	
Bivalve molluscs, 5 spp.		
Edible portions	6 (Max. 36) FW	Stegeman 1981
Decapod crustaceans, 4 spp.		
Edible portions	2 (Max. 8) FW	
Softshell clam, Mya arenaria, soft parts		
Coos Bay, Oregon		
1976-1978		
Near industrialized areas	6-20 FW	Mix and Schaffer 1983a
Remote areas	1-2 FW	
1978-1979		
Near industrialized areas	9 FW	
Remote areas	4 FW	
VERTEBRATES		
Fish, muscle		
Lake Ontario, 6 spp., total PAH	3-8 FW	Lawrence and Weber 1984a
Baltimore Canyon, east coast, USA, 5 spp.		
BaA	Max. 0.3 FW	Brown and Pancirov 1979
BaP	Max. <5 FW	
PYR	Max. <5 FW	
Smoked		
FL	3 FW	EPA 1980
PYR	2 FW	
Nonsmoked		
FL	Max. 1.8 FW	
PYR	Max. 1.4 FW	
Winter flounder, Pseudopleuronectes americanus		
Edible portions, 1980		
New York Bight		
Total PAHs	315 FW	Humason and Gadbois 1982
BaP	21 FW	
Long Island Sound		
Total PAHs	103 FW	
BaP	ND	

Table 4. (Continued)

Taxonomic group, compound, and other variables	Concentration	Reference[a]
Windowpane, Scopthalmus aquosus		
Edible portions, 1980		
New York Bight		
Total PAHs	536 FW	
BaP	4 FW	
Long Island Sound		
Total PAHs	86 FW	
BaP	ND	
Red hake, Urophycus chuss		
Edible portions, 1980		
New York Bight		
Total PAHs	412 FW	
BaP	22 FW	
Long Island Sound		
Total PAHs	124 FW	
BaP	5 FW	
BaP		
Fish		
Marine, edible portions		
9 spp.	Max. 3 FW	Stegeman 1981
Greenland	15 FW	Harrison et al. 1975
Italy	65 FW	
Steak, charcoal broiled	5-8 DW	Barnett 1976
Ribs, barbecued	11 DW	
INTEGRATED STUDIES		
Michigan, 1978, Hersey River		
Near wastewater treatment plant		
PHEN		
Sediments	4,097 FW	Black et al. 1981
Insects, whole	5,488 FW	
Crustaceans, muscle	447 FW	
Fish, muscle	28-15,313 FW	
BaA		
Sediments	3,504 FW	
Insects	2,893 FW	
Crustaceans	40 FW	
Fish	0.2-19 FW	

Table 4. (Concluded)

Taxonomic group, compound, and other variables	Concentration	Reference[a]
BaP		
Sediments	1,194 FW	
Insects	725 FW	
Crustaceans	8 FW	
Fish	0.07-1 FW	
Control location		
Sediments and biota		
PHEN	2-42 FW	
BaA	ND-6.7 FW	
BaP	0.04-1.2 FW	
Nova Scotia, 1980, total PAHs		
Near coking facility		
Sediments	2,830,000 DW	Sirota et al. 1983
American lobster, *Homarus americanus*		
Hepatopancreas	57,300-88,100 FW	
Tail muscle	1,910-2,670 FW	
Control area		
Sediments	<8,220 DW	
American lobster		
Hepatopancreas	1,185 FW	
Tail muscle	216 FW	
Black River, Ohio, contaminated area, total PAHs		
Sediments	6,700 DW	West et al. 1984
Brown bullhead, *Ictalurus nebulosus*	660 FW	
Water	153 FW	

[a]Each reference applies to data in the same row and in the rows that immediately follow for which no reference is indicated.

evident between accumulated levels of individual PAHs and total PAHs (Mix 1982). Elevated PAH concentrations, especially benz(a)anthracene, chrysene, fluorene, phenanthraene, and pyrene in oyster tissues and sediments were measured in samples from the vicinity of marinas, and were higher in oysters in cooler months, when lipids and glycogen were being stored preparatory to spawning (Marcus and Stokes 1985). In general, PAH concentrations in marine clams were highest in areas adjacent to industrialized bayfronts and lowest in clams inhabiting more remote areas; concentrations were lowest in autumn-winter, and highest during spring-summer (Mix and Schaffer 1983a). A similar pattern was observed in mussels, Mytilus edulis, with the more water soluble, lower molecular weight, PAHs bioconcentrated 10X to 100X above that of the higher molecular weight, less water soluble PAHs (Mix and Schaffer 1983b); PAH levels in mussels seemed to be independent of water salinity (Mix and Schaffer 1979). Clams contaminated with PAHs and removed to clean seawater for 24 hours showed significant depuration of unsubstituted 3- and 4-ring PAHs; in contrast, concentrations of all 5-, 6-, and 7-ring compounds, which includes most of the carcinogenic PAHs, were not significantly depurated (Mix 1982). A positive relation exists between PAH isomers in sediments, soft tissues of the mussel Mytilus edulis, and a seaweed (Fucus sp.) collected at Vancouver, British Columbia (Dunn 1980). For mussels, the general trend towards lower levels of higher molecular weight PAHs relative to levels in sediments suggests an uptake mechanism which involves the solution of PAHs in water; superimposed on this pattern is the more rapid turnover and shorter half-life of lower molecular weight PAHs in mussels (Dunn 1980).

PAH residues were higher than expected in American lobsters (Homarus americanus) collected offshore (mean weight 3.6 kg) when compared to smaller (0.6 kg) lobsters collected inshore (Sirota and Uthe 1981), suggesting that age or body size are important modifiers in PAH accumulation dynamics. PAH concentrations in sediments collected near a coking facility in Nova Scotia in 1980 contained up to 2,830 mg/kg dry weight, or more than 20X the levels recorded in Boston (Mass.) Harbor; concentrations in excess of 100 mg/kg dry weight sediment were recorded for phenanthrene, fluorene, pyrene, benz(a)anthracene, chrysene, benzo(e)pyrene, benzo(b)fluoranthene, and benzo(a)pyrene, and these seemed to reflect the elevated tissue levels in American lobsters collected from that locale (Sirota et al. 1983). PAH residues in digestive glands of American lobsters collected in 1979 in Nova Scotia from the vicinity of a major oil spill were higher than those from coastal control sites; however, PAH contents of edible muscle from control and oiled lobsters were similar (Sirota and Uthe 1981).

PAH levels in fish are usually low because this group rapidly metabolizes PAHs (Lawrence and Weber 1984a); furthermore, higher molecular weight PAHs, which include the largest class of chemical carcinogens, do not seem to accumulate in fish (West et al. 1984). Raw fish from unpolluted waters usually do not contain detectable amounts of PAHs, but smoked or cooked fish contain varying levels. The concentration of benzo(a)pyrene in skin of cooked

fish was much higher than in other tissues, suggesting that skin may serve as a barrier to the migration of PAHs in body tissues (EPA 1980).

Sediments and biota collected from the Hersey River, Michigan, in 1978, were heavily contaminated with phenanthrene, benz(a)anthracene, and benzo(a)pyrene when compared to a control site. Elevated PAH concentrations were recorded in sediments, whole insect larvae, crayfish muscle, and flesh of lampreys (family Petromyzontidae), brown trout (Salmo trutta), and white suckers (Catostomus commersoni), in that general order (Black et al. 1981). The polluted collection locale was the former site of a creosote wood preservation facility between 1902 and 1949, and, at the time of the study, received Reed City wastewater treatment plant effluent, described as an oily material with a naphthalene-like odor (Black et al. 1981). In many cases, aquatic organisms from PAH-contaminated environments have a higher incidence of tumors and hyperplastic diseases than those from nonpolluted environments. Carcinogenic PAHs have not been unequivocally identified as the causative agent for an increased incidence of cancer in any natural population of aquatic organisms, according to Neff (1982b). However, a growing body of evidence, mostly circumstantial, links PAHs to cancer in feral fish populations, especially bottom dwelling fish from areas with sediments heavily contaminated with PAHs (Baumann and Lech, in press).

TOXIC AND SUBLETHAL EFFECTS

GENERAL

A wide variety of PAH-caused adverse biological effects have been reported in numerous species of organisms under laboratory conditions, including effects on survival, growth, metabolism, and especially tumor formation. Inter- and intraspecies responses to carcinogenic PAHs were quite variable, and were significantly modified by many chemicals including other PAHs that are weakly carcinogenic or noncarcinogenic. Until these interaction effects are clarified, the results of single substance laboratory tests may be extremely difficult to apply to field situations of suspected PAH contamination.

FUNGI

Fungal degradation of PAHs may be important in the detoxification and elimination of PAHs in the environment. The fungus Cunninghamella elegans, for example, inhibited the mutagenic activity of benzo(a)pyrene, 3-methyl cholanthrene, benz(a)anthracene, and 7,12-dimethylbenz(a)anthracene, as judged by results of the Ames test using Salmonella typhimurium (Cerniglia et al. 1985). The rate of decrease in mutagenic activity in bacterial cultures incubated with PAHs was coincident with the rate of increase in fungal metabolism. C.elegans metabolized PAHs to dihydrodiols, phenols, quinones, and dihydrodiol epoxides, and to sulfate, glucuronide, and glucoside conjugates of these primary metabolites in a manner similar to that reported for mammalian enzyme systems, suggesting that this organism (and perhaps other fungi) is important in PAH metabolism and inactivation (Cerniglia et al. 1985).

TERRESTRIAL PLANTS

Biological effects of PAHs on terrestrial vegetation have been reviewed by EPA (1980), Lee and Grant (1981), Wang and Meresz (1982), Edwards (1983), and Sims and Overcash (1983). In general, these authorities agreed on several points. First, plants and vegetables can absorb PAHs from soils through their roots, and translocate them to other plant parts such as developing shoots. Uptake rates were governed, in part, by PAH concentration, PAH water solubility, soil type, and PAH physicochemical state (vapor or particulate). Lower molecular weight PAHs were absorbed by plants more readily than higher molecular weight PAHs. Under laboratory conditions, some plants concentrated selected PAHs above that of their immediate geophysical surroundings, but this has not been conclusively demonstrated in field-grown cultivated crops or other vegetation. Second, above-ground parts of vegetables, especially the outer shell or skin, contained more PAHs than underground parts, and this was attributed to airborne deposition and subsequent adsorption. Externally deposited PAHs in vegetables were difficult to remove with cold water washings; not more than 25% were removed from lettuce, kale, spinach, leeks, and tomatoes using these procedures. Third, PAH-induced phytotoxic effects were rare; however, the data base on this subject is small. Fourth, most higher plants can catabolize benzo(a)pyrene, and possibly other PAHs, but metabolic pathways have not been clearly defined. Finally, the biomagnification potential of vegetation in terrestrial and aquatic food chains needs to be measured; this work should be conducted with a variety of PAHs in both field and laboratory experiments.

Some plants contain chemicals known to protect against PAH effects. Certain green plants contain ellagic acid, a substance that can destroy the diol epoxide form of benzo(a)pyrene, inactivating its carcinogenic and mutagenic potential (Edwards 1983). PAHs synthesized by plants may act as plant growth hormones (Edwards 1983). Some vegetables, such as cabbage, brussel sprouts, and cauliflower, contain naturally occurring antineoplastic compounds including benzyl isothiocyanate and phenethyl isothiocyanate; these compounds are known to inhibit mammary cancers, stomach tumors, and pulmonary edemas induced in rats by benzo(a)pyrene and 7,12-dimethylbenz(a)anthracene (EPA 1980). Decreased activation of carcinogens has also been demonstrated in animals fed diets that were high in protein, low in carbohydrate, and containing adequate choline; the reverse was observed in diets high in carbohydrate, low in protein, or containing certain organophosphorus insecticides, piperonyl butoxide, carbon tetrachloride, nickel carbonyl, or tin (EPA 1980). In cases where dietary constituents can alter the metabolism of foreign agents, such as PAHs, the anticarcinogenic effect may result from an alteration of steady state levels of activated versus detoxified metabolites (EPA 1980). The implications of these observations to herbivorous wildlife are unknown at present.

AQUATIC BIOTA

PAHs vary substantially in their toxicity to aquatic organisms (Table 5). In general, toxicity increases as molecular weight increases (although high molecular weight PAHs have low acute toxicity, perhaps due to their low solubility in water) and with increasing alkyl substitution on the aromatic ring. Toxicity is most pronounced among crustaceans and least among teleosts (Neff 1979; Table 5). In all but a few cases, PAH concentrations that are acutely toxic to aquatic organisms are several orders of magnitude higher than concentrations found in even the most heavily polluted waters (Neff 1979). Sediments from polluted regions, however, may contain PAH concentrations similar to those which are acutely toxic, but their limited bioavailability would probably render them substantially less toxic than PAHs in solution (Neff 1979).

A growing literature exists on uptake, retention, and translocation of PAHs by aquatic plants and animals. Authorities generally agree that: most species of aquatic organisms studied to date rapidly accumulate (i.e., bioconcentrate) PAHs from low concentrations in the ambient medium; uptake of PAHs is highly species specific, being higher in algae, molluscs, and other species which are incapable of metabolizing PAHs; bioconcentration factors (BCF) tend to increase as the molecular weight of the PAH increases, with increasing octanol/water partition coefficient values, with time until approaching an apparent equilibrium level (sometimes within 24 hours), and with increases in dissolved organic matter in the medium, lipid content of organism, and a variety of endogenous and exogenous factors (Jackim and Lake 1978; Southworth et al. 1978; Lee and Grant 1981; Neff 1982a). BCF values have been determined for selected PAHs and aquatic organisms (Table 6); additional BCF data for aquatic biota are available for plants (Dobroski and Epifanio 1980; Boyle et al. 1984), crustaceans (Southworth 1979; Sirota and Uthe 1981; Fox and Rao 1982; Neff 1982a; Williams et al. 1985), tunicates (Baird et al. 1982), molluscs (Jackim and Wilson 1979; Dobroski and Epifanio 1980; Neff 1982a), and fishes (Southworth 1979; Neff 1982a; Stoker et al. 1984). Algal accumulation of benzo(a)pyrene increased linearly in a 24-hour exposure period, and correlated positively with surface area (Leversee et al. 1981), suggesting adsorption rather than absorption. Algae readily transform benzo(a)pyrene to oxides, peroxides (Kirso et al. 1983), and dihydrodiols (Warshawsky et al. 1983). Photosynthetic rates of algae, and presumably PAH accumulations, were significantly modified by light regimens. For reasons still unexplained, algae grown in "white" light (major energy in blue-green portion of the spectrum) were more sensitive to benzo(a)pyrene than were cultures grown in "gold" light (Warshawsky et al. 1983; Schoeny et al. 1984). Accumulation by oysters (Crassostrea virginica) and clams (Rangia cuneata) of naphthalene, phenanthrene, fluorene, and their methylated derivatives increased with increasing methylation and PAH molecular weight; uptake was more rapid under conditions of aontinuous flow than in static tests (Neff et al. 1976). When returned to PAH-free seawater, molluscs released PAHs to non-

Table 5. Toxicities of selected PAHs to aquatic organisms.

PAH compound, organism, and other variables	Concentration in medium (ug/l)	Effect[a]	Reference[b]
BENZ(a)ANTHRACENE			
Bluegill, *Lepomis macrochirus*	1,000	LC-87 (6 m)	EPA 1980
BENZO(a)PYRENE			
Sandworm, *Neanthes arenceodentata*	>1,000	LC-50 (96 h)	Neff 1979
CHRYSENE			
Sandworm	>1,000	LC-50 (96 h)	
7,12-DIMETHYLBENZ(a)ANTHRACENE			
Minnows, *Poeiciliopsis* spp.			
Juveniles	250	LC-0 (20 h)	Schultz
Juveniles	500	LC-100 (20 h)	and Schultz 1982
DIBENZ(a,h)ANTHRACENE			
Sandworm	>1,000	LC-50 (96 h)	Neff 1979
FLUORANTHENE			
Sandworm	500	LC-50 (96 h)	
FLUORENE			
Grass shrimp, *Palaemonetes pugio*	320	LC-50 (96 h)	
Bluegill	500	LC-12 (30 d)	Finger et al. 1985
Amphipod, *Gammarus pseudoliminaeus*	600	LC-50 (96 h)	
Rainbow trout, *Salmo gairdneri*	820	LC-50 (96 h)	
Bluegill	910	LC-50 (96 h)	
Sandworm	1,000	LC-50 (96 h)	Neff 1979

Table 5. (Continued)

PAH compound, organism, and other variables	Concentration in medium (ug/l)	Effect[a]	Reference[b]
Sheepshead minnow, *Cyprinodon variegatus*	1,680	LC-50 (96 h)	
Snail, *Mudalia potosensis*	5,600	LC-50 (96 h)	Finger et al. 1985
Mayfly, *Hexagenia bilineata*	5,800	LC-50 (120 h)	
Fathead minnow, *Pimephales promelas*	>100,000	LC-0 (96 h)	
NAPHTHALENE			
Copepod, *Eurytemora affinis*	50	LC-30 (10 d)	Neff 1979
Pink salmon, *Oncorhynchus gorbuscha*, fry	920	LC-50 (24 h)	
Dungeness crab, *Cancer magister*	2,000	LC-50 (96 h)	Neff 1985
Grass shrimp	2,400	LC-50 (96 h)	Neff 1979
Sheepshead minnow	2,400	LC-50 (24 h)	
Brown shrimp, *Penaeus aztecus*	2,500	LC-50 (24 h)	
Amphipod, *Elasmopus pectenicrus*	2,680	LC-50 (96 h)	
Coho salmon, *Oncorhyncus kisutch*, fry	3,200	LC-50 (96 h)	Neff 1985
Sandworm	3,800	LC-50 (96 h)	Neff 1979
Mosquitofish, *Gambusia affinis*	150,000	LC-50 (96 h)	
1-METHYLNAPHTHALENE			
Dungeness crab, *Cancer magister*	1,900	LC-50 (96 h)	
Sheepshead minnow	3,400	LC-50 (24 h)	
2-METHYLNAPHTHALENE			
Grass shrimp	1,100	LC-50 (96 h)	Neff 1985
Dungeness crab	1,300	LC-50 (96 h)	
Sheepshead minnow	2,000	LC-50 (24 h)	Neff 1979

Table 5. (Concluded)

PAH compound, organism, and other variables	Concentration in medium (ug/l)	Effect[a]	Reference[b]
TRIMETHYLNAPHTHALENES			
Copepod, Eurytemora affinis	320	LC-50 (24 h)	
Sandworm	2,000	LC-50 (96 h)	
PHENANTHRENE			
Grass shrimp	370	LC-50 (24 h)	
Sandworm	600	LC-50 (96 h)	EPA 1980
1-METHYLPHENANTHRENE			
Sandworm	300	LC-50 (96 h)	

[a] m = months, d = days, h = hours.

[b] Each reference applies to data in the same row and in the rows that immediately follow for which no reference is indicated.

detectable levels in about 60 days, with high molecular weight PAHs depurated more slowly than low molecular weight compounds; brown shrimp (Penaeus aztecus) and longnose killifish (Fundulus similis), which can metabolize PAHs, lost PAHs more quickly than clams and oysters, which apparently lack the detoxifying enzymes (Neff et al. 1976). Pink shrimp (Penaeus duorarum) exposed to 1.0 ug chrysene/l for 28 days and then transferred to unpolluted seawater for an additional 28 days contained concentrations of chrysene (91 ug/kg fresh weight in abdomen, 48 ug/kg in cephalothorax) that were considered potentially hazardous to human consumers over extended periods (Miller et al. 1982). Eggs of the sand sole (Psettichthys melanostictus) exposed to 0.1 ug benzo(a)pyrene /l for 5 days showed reduced and delayed hatch and, when compared to controls, produced larvae with high accumulations (2.1 mg/kg fresh weight) and gross abnormalities, such as twinning and tissue overgrowths, in 50% of the test larvae (Hose et al. 1982). Naphthalene and benzo(a)pyrene were rapidly accumulated from the medium by three species of California marine teleosts; loss was rapid, being >90% for naphthalene in 24 hours, and 20% (muscle) to 90% (gill) for benzo(a)pyrene in a similar period (Lee et al. 1972). Phenanthrene is metabolized by many species of aquatic organisms, including fish. A marine flounder, Platichthys flesus, given a single oral dose of 0.7 mg phenanthrene/kg body weight, contained elevated phenanthrene concentrations in lipids, melanin-rich tissues (such as skin), and the eye lens; most was eliminated within 2 weeks (Solbakken et al. 1984). Different rates of accumulation and depuration of benzo(a)pyrene and naphthalene in bluegill (Lepomis macrochirus) and Daphnia magna have been documented by McCarthy and Jimenez (1985) and McCarthy et al. (1985). Benzo(a)pyrene accumulations in bluegill, for example, were 10X greater than naphthalene, but benzo(a)pyrene is extensively metabolized, whereas naphthalene is not. Consequently, postexposure accumulations of naphthalene greatly exceeded that of the parent benzo(a)pyrene. Because the more hydrophobic PAHs, such as benzo(a)pyrene, show a high affinity for binding to dissolved humic materials and have comparatively rapid biotransformation rates, these interactions may lessen or negate bioaccumulation and food chain transfer of hydrophobic PAHs (McCarthy and Jimenez 1985; McCarthy et al. 1985).

Time to depurate or biotransform 50% of accumulated PAHs (Tb 1/2) varied widely. Tb 1/2 values for Daphnia pulex and all PAH compounds studied ranged between 0.4 and 0.5 hours (Southworth et al. 1978). For marine copepods and naphthalene, a Tb 1/2 of about 36 hours was recorded (Neff 1982a). For most marine bivalve molluscs, Tb 1/2 values ranged from 2 to 16 days. Some species, such as the hardshell clam (Mercenaria mercenaria), showed little or no depuration, while others, such as oysters, eliminated up to 90% of accumulated PAHs in 2 weeks--although the remaining 10% was released slowly, and traces may remain indefinitely (Jackim and Lake 1978). Percent loss of various PAHs in oysters (Crassostrea virginica), 7 days postexposure, ranged from no loss for benzo(a)pyrene to 98% for methylnaphthalene; intermediate were benz(a)anthracene (32%), fluoranthene (66%), anthracene (79%), dimethylnaphthalene (90%), and naphthalene (97%) (Neff 1982a). Teleosts and

Table 6. PAH bioconcentration factors (BCF) for selected species of aquatic organisms.

PAH compound, organism, and other variables	Exposure period[a]	BCF	Reference[b]
ANTHRACENE			
Cladoceran, Daphnia magna	60 m	200	EPA 1980
Fathead minnow, Pimephales promelas	2 to 3 d	485	Southworth 1979
Cladoceran, Daphnia pulex	24 h	760 to 1200	Southworth et al. 1978; Southworth 1979; EPA 1980; Neff 1985
Mayfly, Hexagenia sp.	28 h	3,500	EPA 1980
Rainbow trout, Salmo gairdneri	72 h	4,400 to 9,200	Linder et al. 1985
9-METHYLANTHRACENE			
Cladoceran, Daphnia pulex	24 h	4,583	Neff 1985
BENZ(a)ANTHRACENE			
Cladoceran, Daphnia pulex	24 h	10,109	Southworth et al. 1978
BENZO(a)PYRENE			
Teleosts, 3 spp., Muscle	1 h to 96 h	0.02 to 0.1	EPA 1980
Clam, Rangia cuneata	24 h	9 to 236	Neff 1979; EPA 1980
Bluegill, Lepomis macrochirus	4 h	12	Leversee et al. 1981
Atlantic salmon, Salmo salar Egg	168 h	71	Kuhnhold and Busch 1978
Midge, Chironomus riparius, larvae	8 h	166	Leversee et al. 1981
Rainbow trout, liver	10 d	182 to 920	Gerhart and Carlson 1978

Table 6. (Continued)

PAH compound, organism, and other variables	Exposure period[a]	BCF	Reference[b]
Oyster, Crassostrea virginica	14 d	242	EPA 1980
Northern pike Esox lucius			
Bile and gallbladder	3.3 h	3,974	Balk et al. 1984
"	19.2 h	36,656	
"	8.5 d	82,916	
"	23 d	53,014	
Liver	3.3 h	259	
"	19.2 h	578	
"	8.5 d	1,376	
"	23 d	619	
Gills	3.3 h	283	
"	19.2 h	382	
"	8.5 d	372	
"	23 d	213	
Kidney	3.3 h	192	
"	19.2 h	872	
"	8.5 d	1,603	
Other tissues	3.3 h to 23 d	<55	
Mosquitofish, Gambusia affinis	3 d	930	Lu et al. 1977
Bluegill			
No dissolved humic material (DHM)	48 h	2,657	McCarthy and Jimenez 1985
20 mg/l DHM	48 h	225	
Cladoceran, Daphnia magna	6 h	2,837	Leversee et al. 1981
Alga, Oedogonium cardiacum	3 d	5,258	Lu et al. 1977
Periphyton, mostly diatoms	24 h	9,600	Leversee et al. 1981
Mosquito, Culex pipiens quinquefasciatus	3 d	11,536	Lu et al. 1977
Sand sole, Psettichthys melanostictus			
Egg	6 d	21,000	Hose et al. 1982
Snail, Physa sp.	3 d	82,231	Lu et al. 1977
Cladoceran, Daphnia pulex	3 d	134,248	

Table 6. (Continued)

PAH compound, organism, and other variables	Exposure period[a]	BCF	Reference[b]
CHRYSENE			
Clam, *Rangia cuneata*	24 h	8	Neff 1979
Mangrove snapper, *Lutjanus griseus*			
Liver	4 d	83 to 104	Miller et al. 1982
Liver	20 d	258 to 367	
Pink shrimp, *Penaeus duorarum*			
Cephalothorax	28 d	248 to 361	
Cephalothorax	28 d + 28 d postexposure	21 to 48	
Abdomen	28 d	84 to 199	
Abdomen	28 d + 28 d postexposure	22 to 91	
FLUORANTHENE			
Rainbow trout, liver	21 d	379	Gerhart and Carlson 1978
FLUORENE			
Bluegill	30 d	200 to 1,800	Finger et al. 1985
NAPHTHALENE			
Clam, *Rangia cuneata*	24 h	6	Neff 1979
Sandworm, *Neanthes arenaceodenta*	3 to 24 h	40	Neff 1982a
Sandworm	24 h + 300 h post-treatment	not detectable	
Atlantic salmon, egg	168 h	44 to 83	Kuhnhold and Busch 1978
Cladoceran, *Daphnia pulex*	24 h	131	Neff 1985
Crustaceans, 3 spp.	72 h	195 to 404	Neff 1979
Bluegill, whole	24 h	310	McCarthy and Jimenez 1985

Table 6. (Concluded)

PAH compound, organism, and other variables	Exposure period[a]	BCF	Reference[b]
DIMETHYLNAPHTHALENES			
Crustaceans, 3 spp.	72 h	967 to 1,625	Neff 1979
PERYLENE			
Cladoceran, Daphnia pulex	24 h	7,191	Neff 1985
PHENANTHRENE			
Clam, Rangia cuneata	24 h	32	Neff 1979
Cladoceran, Daphnia pulex	24 h	325	Neff 1985
PYRENE			
Cladoceran Daphnia pulex	24 h	2,702	
Rainbow trout, liver	21 d	69	Gerhart and Carlson 1978

[a] m = minutes, h = hours, d = days.

[b] Each reference applies to the values in the same row and in the rows that follow for which no other reference is indicated.

arthropods usually had low Tb 1/2 values. In bluegill, 89% loss of benzo(a)pyrene was recorded 4 hours postexposure; for midge larvae it was 72% in 8 hours, and for daphnids it was 21% in 18 hours (Leversee et al. 1981).

The role of sediments in PAH uptake kinetics should not be discounted. Sediment-associated anthracene contributed about 77% of the steady state body burden of this compound in the amphipod Hyalella azteca (Landrum and Scavia 1983). For benzo(a)pyrene and the amphipod Pontoporeia hoyi, the sediment source (including interstitial water) accounted for 53% in amphipods collected at 60 m, but only 9% at 23 to 45 m (Landrum et al. 1984). Benthos from the Great Lakes, such as oligochaete worms (Limnodrilus sp., Stylodrilus sp.) and amphipods (Pontoporeia hoyi), obtain a substantial fraction of their PAH body content from the water when sediment PAH concentrations are low. However, when sediment PAH concentrations are elevated, benthos obtain a majority of their PAHs from that source through their ability to mobilize PAHs from the sediment/pore water matrix; the high concentrations of phenanthrene, fluorene, benzo(a)pyrene, and other PAHs measured in these organisms could provide a significant source of PAHs to predator fish (Eadie et al. 1983). Great Lakes benthos appear to contain as much PAHs as the fine grain fraction of the sediment which serves as their food, although overlying water or pore water appears to contribute a larger proportion of PAHs to the organism's body burden than does sediments (Eadie et al. 1984). Marine mussels (Mytilus edulis) and polychaete annelid worms (Nereis virens) exposed for 28 days to sediments heavily contaminated with various PAH compounds accumulated significant concentrations (up to 1,000X control levels) during the first 14 days of exposure, and little thereafter; during a 5-week postexposure period, depuration was rapid, with the more water soluble PAHs excreted most rapidly; PAH levels usually remained above control values to the end of the postexposure period (Lake et al. 1985). English sole (Parophrys vetulus), during exposure for 11 to 51 days to PAH-contaminated sediments, showed significant accumulations of naphthalenes in liver (up to 3.1 mg/kg dry weight) after 11 days, with concentrations declining markedly thereafter; uptake of phenanthrene, chrysene, and benzo(a)pyrene was negligible during the first 7 days (Neff 1982a).

Fluorene effects in freshwater pond ecosystems have recently been evaluated (Boyle et al. 1984, 1985; Finger et al. 1985). In ponds exposed to initial fluorene concentrations of 0.12 to 2.0 mg/l, Tb 1/2 values in water ranged from 6 to 11 days. Ten weeks after fluorene introduction, little degradation had occurred in the organic bottom sediments; fluorene residues were present in fish, invertebrates, and rooted submerged macrophytes. Studies with fingerling bluegills showed that 0.062 mg fluorene/l adversely affected their ability to capture chironomid prey, 0.12 mg/l reduced growth, and 1.0 mg fluorene/l increased their vulnerability to predation by largemouth bass (Micropterus salmoides). The authors concluded that fluorene, at concentrations well below its solubility and at levels that could realistically occur in the environment, represents a potential hazard to aquatic organisms.

Large interspecies differences in ability to absorb and assimilate PAHs from food have been reported. For example, crustaceans (Neff 1982a) and fish (Maccubbin et al. 1985; Malins et al. 1985a, 1985b) readily assimilated PAHs from contaminated food, whereas molluscs and polychaete annelids were limited (Neff 1982a). In all cases where assimilation of ingested PAHs was demonstrated, metabolism and excretion of PAHs were rapid (Neff 1982a). Thus, little potential exists for food chain biomagnification of PAHs (Southworth 1979; Dobroski and Epifanio 1980; Neff 1982a). In laboratory aquatic ecosystem studies, Lu et al. (1977) found that benzo(a)pyrene can be accumulated to high, and potentially hazardous, levels in fish and invertebrates. In the case of mosquitofish (Gambusia affinis), almost all of the accumulated benzo(a)pyrene was from its diet, with negligible accumulations from the medium. However, mosquitofish degraded benzo(a)pyrene about as rapidly as it was absorbed, in contrast to organisms such as snails (Physa sp.) which retained most (88%) of the accumulated benzo(a)pyrene for at least 3 days postexposure, presumably due to deficiencies in their mixed-function oxidase detoxication system (Lu et al. 1977). Benzo(a)pyrene, when administered to northern pike (Esox lucius) through the diet or the medium, followed similar pathways: entry via the gills or gastrointestinal system, metabolism in the liver, and excretion in the urine and bile (Balk et al. 1984). Benthic marine fishes exposed to naphthalene or benzo(a)pyrene, either in diet or through contaminated sediments, accumulated substantial concentrations in tissues and body fluids (Varanasi and Gmur 1981). The tendency of fish to metabolize PAHs extensively and rapidly may explain why benzo(a)pyrene, for example, is frequently undetected, or only detected in low concentrations in livers of fish from environments heavily contaminated with PAHs (Varanasi and Gmur 1980, 1981). Extensive metabolism of benzo(a)pyrene plus the presence of large proportions of polyhydroxy metabolites in liver of English sole indicates the formation of reactive intermediates such as diol epoxides and phenol epoxides of benzo(a)pyrene, both of which are implicated in mammalian mutagenesis and carcinogenesis (Varanasi and Gmur 1981).

Cytotoxic, mutagenic, and carcinogenic effects of many PAHs are generally believed to be mediated through active epoxides formed by interaction with microsomal monooxygenases. These highly active arene oxides can interact with macromolecular tissue components and can further be metabolized or rearranged to phenols or various conjugates. They can also be affected by epoxide hydrolase to form dihydrodiols, which are precursors of biologically active diol epoxides--a group that has been implicated as ultimate carcinogens. Investigators generally agree that marine and freshwater fishes are as well equipped as mammals with liver PAH-metabolizing enzymes; rapidly metabolize PAHs by liver mixed-function oxidases, with little evidence of accumulation; translocate conjugated PAH metabolites to the gall bladder prior to excretion in feces and urine; and have mixed-function oxidase degradation rates that are significantly modified by sex, age, diet, water temperature, dose-time relationships, and other variables. In addition, many species of fishes can convert PAHs, benzo(a)pyrene for example, to potent mutagenic metabolites, but because detection of the 7,8-dihydrodiol, 9,10-epoxide by analytical

methods is extremely difficult, most investigators must use biological assays, such as the Ames test, to detect mutagenic agents. At present, the interaction effects of PAHs with inorganic and other organic compounds are poorly understood. Specific examples of the above listed phenomena for PAH compounds and teleosts are documented for benzo(a)pyrene (Ahokas et al. 1975; Lu et al. 1977; Gerhart and Carlson 1978; Melius et al. 1980; Varanasi et al. 1980, 1984; Stegeman et al. 1982; Couch et al. 1983; Hendricks 1984; Melius 1984; Schoor 1984; Schoor and Srivastava 1984; Hendricks et al. 1985; Neff 1985; Fair 1986; von Hofe and Puffer 1986), 3-methylcholanthrene (Gerhart and Carlson 1978; Melius et al. 1980; Melius and Elam 1983; Schoor and Srivastava 1984; Neff 1985), benz(a)anthracene, chrysene, and pyrene (Gerhard and Carlson 1978), and 7,12-dimethylbenz(a)anthracene (Stegeman et al. 1982).

Baumann et al. (1982) summarized reports on increasing frequencies of liver tumors in wild populations of fish during the past decade, especially in brown bullhead (Ictalurus nebulosus) from the Fox River, Illinois (12% tumor frequency), in Atlantic hagfish (Myxine glutinosa) from Swedish estuaries (6%), in English sole from the Duwamish estuary, Washington (32%), and in tomcod (Microgadus tomcod) from the Hudson River, New York (25%). In all of these instances, significant levels of contaminants were present in the sediments, including PAHs. PAHs have been identified as genotoxic pollutants in sediments from the Black River, Ohio, where a high incidence of hepatoma and other tumors has been observed in ictalurid fishes (West et al. 1984, 1986). Reports of tumors in Great Lakes fish populations have been increasing. Tumors of thyroid, gonad, skin, and liver are reported, with tumor frequency greatest near areas contaminated by industrial effluents such as PAHs; liver tumors were common among brown bullhead populations at sites with large amounts of PAHs in sediments (Baumann 1984). A positive relationship was finally established between sediment PAH levels and prevalence of liver lesions in English sole in Puget Sound, Washington (Malins et al. 1984; Varanasi et al. 1984), and sediment levels and liver tumor frequency in brown bullheads from the Black River, Ohio (Baumann and Harshbarger 1985; Black et al. 1985). Sediment PAH levels in the Black River, Ohio, from the vicinity of a coke plant outfall, were up to 10,000 times greater than those from a control location: concentrations were greater than 100 mg/kg for pyrene, fluoranthene, and phenanthrene; between 50 and 100 mg/kg for benz(a)anthracene, chrysene, and benzofluoranthenes; and between 10 and 50 mg/kg for individual naphthalenes, benzo(e)pyrene, benzo(a)pyrene, perylene, indeno(1,2,3-cd)pyrene, benzo(g,h,i)perylene, and anthanthrene (Baumann et al. 1982). Brown bullheads from this location contained >1.0 mg/kg of acenapthalene (2.4), phenanthrene (5.7), fluoranthene (1.9), and pyrene (1.1), and lower concentrations of heavier molecular weight PAHs; bullheads also exhibited a high (33%) liver tumor frequency, which seemed to correspond to their PAH body burdens. Investigators concluded that the elevated frequency of liver neoplasia in Black River bullheads was chemically induced, and was the result of exposure to PAHs (Baumann et al. 1982; Baumann and Harshbarger 1985).

Neoplasms in several species of fishes have been produced experimentally with 3-methylcholanthrene, acetylaminofluorene, benzo(a)pyrene, and 7,12-dimethylbenz(a)anthracene, with tumors evident 3 to 12 months postexposure (Couch and Harshbarger 1985; Hendricks et al. 1985). Under laboratory conditions, liver neoplasms were induced in two species of minnows (Poeciliopsis spp.) by repeated short-term exposures (6 hours once a week, for 5 weeks) to an aqueous suspension of 5 mg/l of 7,12-dimethyl-benz(a)anthracene. About 44% of the fish surviving this treatment developed hepatocellular neoplasms 6 to 9 months postexposure (Schultz and Schultz 1982). Eastern mudminnows (Umbra pygmaea) kept in water containing up to 700 ug PAHs/l for 11 days showed increased frequencies of chromosomal aberrations in gills: 30% vs. 8% in controls (Prein et al. 1978). High dietary benzo(a)pyrene levels of 500 mg/kg produced significant elevations in hepatic mixed-function oxidase levels in rainbow trout after 9 weeks (Hendricks et al. 1985). Rainbow trout fed diets containing 1,000 mg benzo(a)pyrene/kg for 12 months developed liver tumors (Couch et al. 1983). About 25% of rainbow trout kept on diets containing 1,000 mg benzo(a)pyrene/kg for 18 months had histologically confirmed liver neoplasms as compared to 15% after 12 months, with no evidence of neoplasia in controls (Hendricks et al. 1985). Young English sole may activate and degrade carcinogenic PAHs, such as benzo(a)pyrene, to a greater extent than adults, but additional research is needed to determine if younger fish are at greater risk than older sole to PAH-induced toxicity (Varanasi et al. 1984). In English sole, a high significant positive correlation between PAH metabolites (1-and 3-hydroxy benzo(a)pyrene, hydroxy and dihydrodiol metabolites of pyrene and fluoranthene) in bile, and idiopathic liver lesions, prevalance of neoplasms, megalocytic hepatosis, and total number of hepatic lesions (Krahn et al. 1986) suggests that selected PAH metabolites and key organs or tissues may be the most effective monitors of PAH contamination in aquatic organisms.

In addition to those effects of PAHs emphasizing survival, uptake, depuration, and carcinogenesis previously listed, a wide variety of additional effects have been documented for aquatic organisms. These include: inhibited reproduction of daphnids and delayed emergence of larval midges by fluorene (Finger et al. 1985); decreased respiration and heart rate in mussels (Mytilus californianus) by benzo(a)pyrene (Sabourin and Tullis 1981); increased weight of liver, kidney, gall bladder, and spleen of sea catfish (Arius felis) by 3-methylcholanthrene, which was dose-related (Melius and Elam 1983); photosynthetic inhibition of algae and macrophytes by anthracene, naphthalene, phenanthrene, pyrene (Neff 1985), and fluorene (Finger et al. 1985); immobilization of the protozoan, Paramecium caudatum, by anthracene, with an EC-50 (60 min) of 0.1 ug/l (EPA 1980); perylene accumulation by algae (Stegeman 1981); accumulation without activation of benzo(a)pyrene and benzo(a)anthracene by a marine protozoan (Parauronema acutum), and biotransformation of various fluorenes by P. acutum to mutagenic metabolites (Lindmark 1981); interference by toluene and anthracene with benzo(a)pyrene uptake by freshwater amphipods (Landrum 1983); abnormal blood chemistry in oysters (Crassostrea virginica) exposed for one year to 5 ug 3-methyl-

cholanthrene/l (Couch et al. 1983); and enlarged livers in brown bullheads from a PAH-contaminated river (Fabacher and Baumann 1985).

AMPHIBIANS AND REPTILES

Limited data were available on biological effects of benzo(a)pyrene, 3-methylcholanthrene, and perylene to reptiles and amphibians (Balls 1964; Stegeman 1981; Anderson et al. 1982; Schwen and Mannering 1982a, 1982b; Couch et al. 1983).

Implantation of 1.5 mg of benzo(a)pyrene crystals into the abdominal cavity of adult South African clawed toads (Xenopus laevis) produced lymphosarcomas in 11 of the 13 toads (85%) after 86 to 288 days (Balls 1964). Immature toads were more resistant, with only 45% bearing lymphoid tumors of liver, kidney, spleen, or abdominal muscle 272 to 310 days after implantation of 1.5 mg of benzo(a)pyrene crystals in the dorsal lymph sac or abdominal cavity. Implantation of 3-methylcholanthrene crystals into X. laevis provokes development of lymphoid tumors similar to those occurring naturally in this species; moreover, these tumors are readily transplantable into other Xenopus or into the urodele species Triturus cristatus (Balls 1964). Intraperitoneal injection of perylene into tiger salamanders can result in hepatic tumors (Couch et al. 1983).

A critical point of interaction between PAHs and reptiles/amphibians involves the transformation of these compounds by cytochrome P-450-dependent monooxygenase systems (Stegeman 1981); in general, reaction rates in this group are considerably slower than those observed in hepatic microsomes from mammals (Schwen and Mannering 1982a). Mixed-function oxidation systems can be induced in liver and skin of tiger salamanders by perylene (Couch et al. 1983) and 3-methylcholanthrene (Anderson et al. 1982), and in liver of the leopard frog (Rana pipiens) and garter snake (Thamnophis sp.) by benzo(a)pyrene and 3-methylcholanthrene (Stegeman 1981; Schwen and Mannering 1982a, 1982b). A single dose of 40 mg/kg body weight of 3-methylcholanthrene was sufficient to induce mixed-function oxidase activity for several weeks in the leopard frog (Schwen and Mannering 1982b). Amphibians, including tiger salamanders, are quite resistant to PAH carcinogenesis when compared to mammals, according to Anderson et al. (1982). This conclusion was based on studies with Ambystoma hepatic microsomes and their inability to produce mutagenic metabolites of benzo(a)pyrene and perylene (as measured by bacterial Salmonella typhimurium strains used in the Ames test); however, rat liver preparations did produce mutagenic metabolites under these procedures (Anderson et al. 1982).

BIRDS

Only two articles were available on PAHs and avian wildlife, and both concerned mallards (Anas platyrhynchos). In one study, Patton and Dieter (1980) fed mallards diets that contained 4,000 mg PAHs/kg (mostly as naphthalenes, naphthenes, and phenanthrene) for a period of 7 months. No mortality or visible signs of toxicity were evident during exposure; however, liver weight increased 25% and blood flow to liver increased 30%, when compared to controls. In the second study, Hoffman and Gay (1981) measured embryotoxicity of various PAHs applied externally, in a comparatively innocuous synthetic petroleum mixture, to the surface of mallard eggs. The most embryotoxic PAH tested was 7,12-dimethylbenz(a)anthracene: approximately 0.002 ug/egg (equivalent to about 0.036 ug/kg fresh weight, based on an average weight of 55 g per egg) caused 26% mortality in 18 days, and, among survivors, produced significant reduction in embryonic growth and a significant increase in the percent of anomalies, e.g., incomplete skeletal ossification, defects in eye, brain, liver, feathers, and bill. At 0.01 ug 7,12-dimethylbenz(a)anthracene/egg, only 10% survived to day 18. Similar results were obtained with 0.015 ug (and higher) chrysene/egg. For benzo(a)pyrene, 0.002 ug/egg did not affect mallard survival, but did cause embryonic growth reduction and an increased incidence of abnormal survivors. At 0.01 ug benzo(a)pyrene/egg, 60% died in 18 days; at 0.05 ug/egg, 75% were dead within 3 days of treatment. Embryos may contain microsomal enzymes that can metabolize PAHs to more highly toxic intermediates than can adults, and avian embryos may have a greater capacity to metabolize PAHs in this manner than do mammalian embryos and fetuses (as quoted in Hoffman and Gay 1981); this observation warrants additional research. Several investigators have suggested that the presence of PAHs in petroleum, including benzo(a)pyrene, chrysene, and 7,12-dimethylbenz(a)anthracene, significantly enhances the overall embryotoxicity in avian species, and that the relatively small percent of the aromatic hydrocarbons contributed by PAHs in petroleum may confer much of the adverse biological effects reported after eggs have been exposed to microliter quantities of polluting oils (Hoffman and Gay 1981; Albers 1983).

MAMMALS

Numerous PAH compounds are distinct in their ability to produce tumors in skin and in most epithelial tissues of practically all animal species tested; malignancies were often induced by acute exposures to microgram quantities. In some cases, the latency period can be as short as 4 to 8 weeks, with the tumors resembling human carcinomas (EPA 1980). Certain carcinogenic PAHs are capable of passage across skin, lungs, and intestine, and can enter the rat fetus, for example, following intragastric or intravenous administration to pregnant dams (EPA 1980). In most cases, the process of carcinogenesis occurs

over a period of many months in experimental animals, and many years in man. The tissue affected is determined by the route of administration and species under investigation. Thus, 7,12-dimethylbenz(a)anthracene is a potent carcinogen for the mammary gland of young female rats after oral or intravenous administration; dietary benzo(a)pyrene leads to leukemia, lung adenoma, and stomach tumors in mice; and both PAH compounds can induce hepatomas in skin of male mice when injected shortly after birth (Dipple 1985). Acute and chronic exposure to various carcinogenic PAHs have resulted in destruction of hematopoietic and lymphoid tissues, ovotoxicity, antispermatogonic effects, adrenal necrosis, changes in the intestinal and respiratory epithelia, and other effects (Table 7; EPA 1980; Lee and Grant 1981). For the most part, however, tissue damage occurs at dose levels that would also be expected to induce carcinomas, and thus the threat of malignancy predominates in evaluating PAH toxicity. There is a scarcity of data available on the toxicological properties of PAHs which are not demonstrably carcinogenic to mammals (EPA 1980; Lee and Grant 1981).

Target organs for PAH toxic action are diverse, due partly to extensive distribution in the body and also to selective attack by these chemicals on proliferating cells (EPA 1980). Damage to the hematopoietic and lymphoid system in experimental animals is a particularly common observation (EPA 1980). In rats, the target organs for 7,12-dimethylbenz(a)anthracene are skin, small intestine, kidney, and mammary gland, whereas in fish the primary target organ is liver (Schultz and Schultz 1982). Application of carcinogenic PAHs to mouse skin leads to destruction of sebaceous glands and to hyperplasia, hyperkeratosis, and ulceration (EPA 1980). Tumors are induced in mouse skin by the repeated application of small doses of PAHs, by a single application of a large dose, or by the single application of a subcarcinogenic dose (initiation) followed by repeated application of certain noncarcinogenic agents (promotion) (Dipple 1985). Newborn mice were highly susceptible to 3-methylcholanthrene, with many mice dying from acute or chronic wasting disease following treatment; some strains of mice eventually developed thynomas, but other strains showed no evidence despite serious damage to the thymus (EPA 1980).

In general, PAH carcinogens transform cells through genetic injury involving metabolism of the parent compound to a reactive diol epoxide. This, in turn, can then form adducts with cellular molecules, such as DNA, RNA, and proteins, resulting in cell transformation (Dipple 1985; Ward et al. 1985). In the case of benzo(a)pyrene, one isomer of the 7,8-diol, 9,10-epoxide is an exceptionally potent carcinogen to newborn mice and is believed to be the ultimate carcinogenic metabolite of this PAH (Slaga et al. 1978). One of the most toxicologically significant processes involved in the response to PAH absorption is the interaction with drug metabolizing enzyme systems (Lee and Grant 1981). Increased production of mixed-function oxidase enzymes in various small mammals has been induced by halogenated napthalenes (Campbell et al. 1983), 3-methylcholanthrene (Miranda and Chhabra 1980), and numerous other PAHs (EPA 1980). PAH metabolites produced by microsomal enzymes in mammals

Table 7. Some effects of PAHs on selected laboratory animals.

Effect (units), organism, PAH compound	Concentration	Reference[a]
LD-50, ACUTE ORAL (mg/kg body weight)		
Rodents (*Rattus* spp., *Mus* spp.)		
Benzo(a)pyrene	50	Sims and Overcash 1978
Phenanthrene	700	
Naphthalene	1,780	
Fluoranthene	2,000	
CARCINOGENICITY, CHRONIC ORAL (mg/kg body weight)		
Rodents		
7,12-dimethylbenz(a)anthracene	0.00004-0.00025	Lo and Sandi 1978
Benzo(a)pyrene	0.002	Sims and Overcash 1983
Dibenz(a,h)anthracene	0.006	
Benz(a)anthracene	2.0	
Benzo(b)fluoranthene	40.0	
Benzo(k)fluoranthene	72.0	
Indeno(1,2,3-cd)pyrene	72.0	
Chrysene	99.0	
Anthracene	3,300.0	
CARCINOGENICITY, APPLIED EXTERNALLY AS TOPICAL (mg)		
Mice, *Mus* spp.		
Benzo(a)pyrene	0.001	Lo and Sandi 1978
Dibenz(a,c)anthracene	0.001	
7,12-dimethylbenz(a)anthracene	0.02	
Dibenz(a,j)anthracene	0.039	
Anthracene	0.08	
Benzo(g,h,i)perylene	0.8	
Benz(a)anthracene	1.0	
CARCINOGENICITY, SUBCUTANEOUS (mg)		
Mice		
Dibenz(a,h)anthracene		
Adults	>0.0002	

Table 7. (Continued)

Effect (units), organism, PAH compound	Concentration	Reference[a]
Newborn	>0.00008	
Dibenzo(a,i)pyrene		
In sesame oil	0.05	
In peanut oil	0.6	
Benzo(a)pyrene	0.06	
Dibenzo(a,e)pyrene	>0.6	
Benzo(b)fluoranthene	1.8	
Benz(a)anthracene	5.0	
Dibenzo(a,h)pyrene	6.0	
TESTICULAR DAMAGE (mg)		
Rat, Rattus spp.		
Benzo(a)pyrene, oral	100.0 (no effect)	EPA 1980
7,12-dimethylbenz(a)anthracene		
Intravenous		
Young rats	0.5 - 2.0	
Older rats	5.0	
Oral	20.0	
OOCYTE AND FOLLICLE DESTRUCTION, SINGLE INTRAPERITONEAL INJECTION (mg/kg body weight)		
Mice		
Benzo(a)pyrene	80.0	Mattison 1980
3-methylcholanthrene	80.0	
7,12-dimethylbenz(a)anthracene	80.0	
ALTERED BLOOD SERUM CHEMISTRY AND NEPHROTOXICITY, SINGLE INTRAPERITONEAL INJECTION (mg/kg body weight)		
Rat		
Phenanthrene	150.0	Yoshikawa et al. 1985
Pyrene	150.0	

Table 7. (Concluded)

Effect (units), organism, PAH compound	Concentration	Reference[a]
FOOD CONSUMPTION, DAILY FOR 5 DAYS (mg/kg body weight)		
Deer mice, Peromyscus maniculatus		
2-methoxynaphthalene		
30% reduction	825	Schafer and Bowles 1985
2-ethoxynaphthalene		
3% reduction	1,213	
House mice, Mus musculus		
2-methoxynaphthalene		
50% reduction	825	
2-ethyoxynaphthalene		
50% reduction	1,213	

[a]Each reference applies to the values in the same row, and in the rows that follow for which no other reference is indicated.

can be arbitrarily divided into water soluble groups, and organosoluble groups such as phenols, dihydrodiols, hydroxymethyl derivatives, quinones, and epoxides (EPA 1980). In the case of benzo(a)pyrene, the diol epoxides are usually considered as the ultimate carcinogens. Other microsomal enzymes convert epoxide metabolites to easily excretable water soluble compounds, with excretion primarily through feces and the hepatobiliary system (EPA 1980). Interspecies differences in sensivity to PAH-induced carcinogenesis are due largely to differences in levels of mixed function oxidase activities, and these will directly affect rates at which active metabolites are converted to less active products (Neff 1979).

Investigators agree that unsubstituted aromatic PAHs with less than 4 condensed rings have not shown tumorgenic activity; that many, but not all, 4-, 5-, and 6-ring PAH compounds are carcinogenic; and that only a few unsubstituted hydrocarbons with 7 rings or greater are tumorogenic or carcinogenic (Neff 1979; EPA 1980; Dipple 1985). Many PAH compounds containing 4 and 5 rings, and some containing 6 or more rings, provoke local tumors after repeated application to the dorsal skin of mice; the tumor incidence exhibited a significant dose-reponse relationship (Grimmer et al. 1985). Among unsubstituted PAHs containing a nonaromatic ring, e.g., cholanthrene and acenapthanthracene, all active carcinogens retained an intact phenanthrene segment (EPA 1980). The addition of alkyl substitutents in certain positions in the ring system of a fully aromatic PAH will often confer carcinogenic activity or dramatically enhance existing carcinogenic potency. For example, monomethyl substitution of benz(a)anthracene can lead to strong carcinogenicity in mice, with potency depending on the position of substitution in the order 7 > 6 > 8 = 12 > 9; a further enhancement of carcinogenic activity is produced by appropriate dimethyl substitution, with 7,12-dimethylbenz(a)anthracene among the most potent PAH carcinogens known. Alkyl substitution of partially aromatic condensed ring systems may also add considerable carcinogenic activity, as is the case with 3-methylcholanthrene. With alkyl substitutes longer than methyl, carcinogenicity levels decrease, possibly due to a decrease in transport through cell membranes (EPA 1980).

A good correlation exists between skin tumor initiating activities of various benzo(a)pyrene metabolites and their mutagenic activity in mammalian cell mutagenesis systems (Slaga et al. 1978), although variations in chromosome number and structure may accompany tumors induced by various carcinogenic PAHs in rats, mice, and hamsters (Bayer 1978; EPA 1980). Active PAH metabolites, e.g., dihydrodiols or diol epoxides, can produce sister chromatid exchanges in Chinese hamster ovary cell (Bayer 1978; EPA 1980; Pal 1984). When exchanges were induced by the diol epoxide, a close relationship exists between the frequency of sister chromatid exchanges and the levels of deoxyribnucleoside-diol-epoxide adduct formation (Pal 1984). In general, noncarcinogenic PAHs were not mutagenic (EPA 1980).

Laboratory studies with mice have shown that many carcinogenic PAHs adversely affect the immune system, thus directly impacting an organism's

general health, although noncarcinogenic analogues had no immunosuppressive effect; further, the more carcinogenic the PAH, the greater the immunosuppression (Ward et al. 1985).

Destruction of oocytes and follicles in mice ovary is documented following intraperitoneal injection of benzo(a)pyrene; 3-methylcholanthrene, and 7,12-dimethylbenz(a)anthracene; the rate of destruction was proportional to the activity of the ovarian cytochrome P-450 dependent monooxygenase, as well as the carcinogenicity of the PAH (Mattison 1980). However, no information is presently available to indicate whether PAHs present a hazard to reproductive success. In those cases where teratogenic effects are clearly evident, e.g., 7, 12-dimethylbenz(a)anthracene, the required doses were far in excess of realistic environmental exposures (Lee and Grant 1981).

Numerous studies show that unsubstituted PAHs do not accumulate in mammalian adipose tissues despite their high lipid solubility, probably because they tend to be rapidly and extensively metabolized (EPA 1980; Lee and Grant 1981).

Biological half-life (Tb 1/2) of PAHs is limited, as judged by rodent studies. In the case of benzo(a)pyrene and rat blood and liver, Tb 1/2 values of 5 to 10 minutes were recorded; the initial rapid elimination phase was followed by a slower disappearance phase lasting 6 hours or more (EPA 1980). Tb 1/2 values from the site of subcutaneous injection in mice were 1.75 weeks for benzo(a)pyrene, 3.5 weeks for 3-methylcholanthrene, and 12 weeks for dibenz(a,h)anthracene; the relative carcinogenicity of each compound was directly proportional to the time of retention at the injection site (Pucknat 1981).

Many chemicals are known to modify the action of carcinogenic PAHs in experimental animals, including other PAHs that are weakly carcinogenic or noncarcinogenic. The effects of these modifiers on PAH metabolism appear to fall into three major categories: those which alter the metabolism of the carcinogen, causing decreased activation or increased detoxification; those which scavenge active molecular species of carcinogens to prevent their reaching critical target sites in the cell; and those which exhibit competitive antagonism (DiGiovanni and Slaga 1981b). For example, benz(a)anthracene, a weak carcinogen, when applied simultaneously with dibenz(a,h)anthracene, inhibited the carcinogenic action of the latter in mouse skin; a similar case is made for benzo(e)pyrene or dibenz(a,c)anthracene applied to mouse skin shortly prior to initiation with 7,12-dimethylbenz(a)anthracene, or 3-methylcholanthrene (DiGiovanni and Slaga 1981a). Benzo(a)pyrene, a known carcinogen, interacts synergistically with cyclopenta(cd)pyrene, a moderately strong carcinogen found in automobile exhausts, according to results of mouse skin carcinogenicity studies (Rogan et al. 1983). Other PAH combinations were cocarcinogenic, such as benzo(e)pyrene, pyrene, and fluoranthene applied repeatedly with benzo(a)pyrene to the skins of mice (DiGiovanni and Slaga 1981a). Effective

inhibitors of PAH-induced tumor development include selenium, vitamin E, ascorbic acid, butylated hydroxytoluene, and hydroxyanisole (EPA 1980). In addition, protective effects against PAH-induced tumor formation have been reported for various naturally occurring compounds such as flavones, retenoids, and vitamin A (EPA 1980). Until these interaction effects are clarified, the results of single substance laboratory studies may be extremely difficult to apply to field situations of suspected PAH contamination. Additional work is also needed on PAH dose-response relationships, testing relevant environmental PAHs for carcinogenicity, and elucidating effects of PAH mixtures on tumor formation (Grimmer 1983).

RECOMMENDATIONS

At present, no criteria or standards have been promulgated for PAHs by any regulatory agency for the protection of sensitive species of aquatic organisms or wildlife. This observation is not unexpected in view of several factors: (1) the paucity of data on PAH background concentrations in wildlife and other natural resources; (2) the absence of information on results of chronic oral feeding studies of PAH mixtures and the lack of a representative PAH mixture for test purposes; and (3) the demonstrable--and as yet, poorly understood--effects of biological modifiers, such as sex, age, and diet, and interaction effects of PAHs with inorganic and other organic compounds, including other PAHs.

Nevertheless, the growing data base for aquatic life indicates a number of generalizations: (1) many PAHs are acutely toxic at concentrations between 50 and 1,000 ug/l; (2) deleterious sublethal responses are sometimes observed at concentrations in the range of 0.1 to 5.0 ug/l; (3) uptake can be substantial, but depuration is usually rapid except in some species of invertebrates; and (4) whole body burdens in excess of 300 ug benzo(a)pyrene/kg (and presumably other PAHs) in certain teleosts would be accompanied by a rise in the activity of detoxifying enzymes.

Current aquatic research has focused on PAHs because of their known relationship with carcinogenesis and mutagenesis. Many reports exist of high incidences of cancer-like growths and developmental anomalies in natural populations of aquatic animals and plants, but none conclusively demonstrate the induction of cancer by exposure of aquatic animals to environmentally realistic levels of carcinogenic PAHs in the water column, diet, or sediments (Neff 1982b, 1985). However, recent studies by Baumann, Malins, Black, Varanasi and their coworkers, among others, have now established that sediments heavily contaminated with PAHs from industrial sources were the direct cause of elevated PAH body burdens and elevated frequencies of liver neoplasia in fishes from these locales. At present, only a few sites containing high PAH concentrations in sediments have been identified (Couch and Harshbarger 1985), suggesting an urgent need to identify and to evaluate other PAH-contaminated aquatic sites. Most fishery products consumed by upper trophic levels, including man, contain PAH concentrations similar to those in green vegetables and smoked and charcoal-broiled meats, and would probably represent a minor source of PAH toxicity; however, consumption of aquatic

organisms, especially filter-feeding bivalve molluscs, from regions severely contaminated with petroleum or PAH-containing industrial wastes, should be avoided (Jackim and Lake 1978; Neff 1982b). Neff (1982b) suggested that repeated consumption of PAH-contaminated shellfish may pose a cancer risk to humans. If true, this needs to be evaluated using seabirds, pinnipeds, and other wildlife groups which feed extensively on molluscs that are capable of accumulating high burdens of carcinogenic PAHs, in order to determine if similar risks exist.

For avian wildlife, data are missing on PAH background concentrations and on acute and chronic toxicity; these data should be collected posthaste. Studies with mallard embryos and PAHs applied to the egg surface showed toxic and adverse sublethal effects at concentrations between 0.036 and 0.18 ug PAH/kg whole egg (Hoffman and Gay 1981). Additional research is needed on petroleum-derived PAHs and their effects on developing embryos of seabirds and other waterfowl.

PAH criteria for human health protection (Table 8) were derived from tests with small laboratory mammals, primarily rodents. Accordingly, these proposed criteria should become interim guidelines for protection of nonhuman mammalian resources pending acquisition of more definitive data. The proposed PAH criteria are controversial. Pucknat (1981) states that there is no way at present to quantify the potential human health risks incurred by the interaction of any PAH with other PAHs or with other agents in the environment, including tumor initiators, promotors, and inhibitors. The problem arises primarily from the diversity of test systems and bioassay conditions used for determining carcinogenic potential of individual PAHs in experimental animals, and is confounded by the lack of a representative PAH mixture for test purposes, the absence of data for animal and human chronic oral exposures to PAH mixtures, and the reliance on data derived from studies with benzo(a)pyrene to produce generalizations concerning environmental effects of PAHs--generalizations which may not be scientifically sound--according to Pucknat (1981). EPA (1980) emphasizes that only a small percentage of PAH compounds are known to be carcinogenic, and that measurements of total PAHs (i.e., the sum of all multiple fused-ring hydrocarbons having no heteroatoms) can not be equated with carcinogenic potential; furthermore, when the term "total PAHs" is used, the compounds being considered should be specified for each case. Lee and Grant (1981) state that an analysis of dose-response relationships for PAH-induced tumors in animals shows, in some cases, deviation from linearity in dose-response curves, especially at low doses, suggesting a two-stage model consistent with a linear nonthreshold pattern. Because overt tumor induction follows a dose-response relationship consistent with a multihit promotion process, the multihit component of carcinogenesis may be supplied by environmental stimuli not necessarily linked or related to PAH exposure.

The well-documented existence of carcinogenic and anticarcinogenic agents strongly suggests that a time assessment of carcinogenic risk for a particular

Table 8. Proposed PAH criteria for human health protection (modified from EPA 1980; Lee and Grant 1981; Pucknat 1981).

Criterion, PAH group, and units	Concentration
DRINKING WATER	
Total PAHs	
ug/l[a]	0.0135-0.2
Daily intake, ug[a]	0.027-0.4
Yearly intake, ug	4.0
Benzo(a)pyrene	
ug/l	0.00055
Daily intake, ug	0.0011
Carcinogenic PAHs	
ug/l[b]	0.0021
Daily intake, ug[b]	0.0042
ug/l[c]	
Cancer risk 10^{-5}	0.028
Cancer risk 10^{-6}	0.0028
Cancer risk 10^{-7}	0.00028
Daily intake, ug[c]	
Cancer risk 10^{-5}	0.056
Cancer risk 10^{-6}	0.0056
Cancer risk 10^{-7}	0.00056
FOOD	
Total PAHs	
Daily intake, ug[d]	1.6-16.0
Yearly intake, ug	4,150.0
Benzo(a)pyrene	
Daily intake, ug[e]	0.16-1.6
AIR	
Total PAHs	
ug/m^3	0.0109
Daily intake, ug[f]	0.164-0.251
Cyclohexane extractable fractions	
Coke oven emissions, coal tar products, ug/m^3, 8 to 10 hour-weighted average	100.0-150.0

Table 8. (Concluded)

Criterion, PAH group, and units	Concentration
Benzene soluble fractions	
Coal tar pitch volatiles, ug/m^3, 8-hour, time-weighted average	200.0
Benzo(a)pyrene	
ug/m^3	0.0005
Daily intake, ug[f]	0.005-0.0115
Carcinogenic PAHs	
ug/m^3	0.002
Daily intake, ug[f]	0.03-0.046
ALL SOURCES	
Total PAHs	
Daily, ug	1.79-16.6
Benzo(a)pyrene	
Daily intake, ug	0.166-1.61
Daily allowable limit, ug[g]	0.048
Carcinogenic PAHs (except diet)	
Daily intake, ug[b]	0.086-0.102

[a]Total of 6 PAHs: fluoranthene, benzo(a)pyrene, benzo(g,h,i)perylene, benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene.

[b]Total of 3 PAHs: benzo(a)pyrene, benzo(j)fluoranthene, and indeno(1,2,3-cd)pyrene.

[c]Based on all carcinogenic PAHs.

[d]Assuming 1,600 g food daily, 70 kg adult, 1 to 10 ug total PAHs/diet.

[e]As above, except 0.1 to 1.0 ug benzo(a)pyrene/diet.

[f]Assuming average of 15 to 23 m^3 of air inhaled daily.

[g]From Wang and Meresz (1982).

PAH can be evaluated only through a multifactorial analysis (Lee and Grant 1981). One of the most toxicologically significant processes involved in the response to PAH absorption is the interaction with drug metabolizing enzyme systems. The induction of this enzyme activity in various body tissues by PAHs and other xenobiotics is probably critical to the generation of reactive PAH metabolites at the target site for tumor induction. At present, wide variations occur in human and animal carcinogen-metabolizing capacity. Moreover, it has not yet been possible to definitely correlate enzyme activity with susceptibility to carcinogenesis. The obligatory coupling of metabolic activation with PAH-induced neoplasia in animals indicates that the modulation of drug metabolizing enzymes is central to carcinogenesis (Lee and Grant 1981).

PAHs from drinking water contribute only a small proportion of the average total human intake (Harrison et al. 1975). The drinking water quality criterion for carcinogenic PAH compounds is based on the assumption that each compound is as potent as benzo(a)pyrene, and that the carcinogenic effect of the compounds is proportional to the sum of their concentrations (EPA 1980). Based on an oral feeding study of benzo(a)pyrene in mice, the concentration of this compound estimated to result in additional risk of one additional case for every 100,000 individuals exposed (i.e, 10^{-5}) is 0.028 ug/l. Therefore, with this assumption, the sum of the concentrations of all carcinogenic PAH compounds should be less than 0.028 ug/l in order to keep the lifetime cancer risk below 10^{-5} (EPA 1980). The corresponding recommended criteria which may result in an incremental cancer risk of 10^{-6} and 10^{-7} over the lifetime are 0.0028 and 0.00028 ug/l, respectively (Table 8). If the above estimates are made for consumption of aquatic organisms only, the levels are 0.311 (10^{-5}), 0.031(10^{-6}), and 0.003(10^{-7}) ug/kg, respectively (EPA 1980). The use of contaminated water for irrigation can also spread PAHs into other vegetable foodstuffs (EPA 1980). When vegetables grown in a PAH-polluted area are thoroughly washed and peeled, their contribution to total PAH intake in humans is not significant (Wang and Meresz 1982). Herbivorous wildlife, however, may ingest significant quantities of various PAHs from contaminated vegetables--but no data were available on this subject.

PAHs are widely distributed in the environment as evidenced by their detection in sediments, soils, air, surface waters, and plant and animal tissues. However, the ecological impact of PAHs is uncertain. PAHs show little tendency for bioconcentration, despite their high lipid solubility (Pucknat 1981), probably because most PAHs are rapidly metabolized. Sims and Overcash (1983) list a variety of research needs regarding PAHs in soil-plant systems. Specifically, research is needed to establish: the rates of PAH decomposition in soils; the soil PAH levels above which PAH constituents adversely affects the food chain; and enhancement factors that increase degradation rates of PAHs, especially PAHs with more than 3 rings. Once these factors have been determined, PAH disposal into soils may become feasible at environmentally nonhazardous levels.

Diet is the major source of PAHs to humans. Authorities agree that most foods contain 1 to 10 ug total PAHs/kg fresh weight, that smoking or barbecuing fish and meats increases total PAH content up to 100X, that contaminated molluscs and crustaceans may contribute significantly to PAH intake, and that PAH carcinogenic risk to humans has existed at least since man began to cook his food (Barnett 1976; EPA 1980; Lee and Grant 1981; Lawrence and Weber 1984a). A total of 22 PAHs has been identified in foods, of which 11 have been found to be carcinogenic in experimental animals. Of these, only 5 (benzo(a)pyrene, benz(a)anthracene, 3-methylcholanthrene, dibenz(a,h)anthracene, and 7,12-dimethylbenz(a)anthracene) have been demonstrated to induce tumors following oral administration to rats and mice, and only 3 of the 11 exhibited positive dose-response relationships in chronic studies with mice (Lo and Sandi 1978). At the present time, there is no evidence that any of the 11 known carcinogenic PAHs or their combinations can cause cancer in human beings via the oral route, especially in quantities likely to be present in foods (Lo and Sandi 1978).

In view of the carcinogenic characteristics of many PAH compounds, their increasing concentrations in the environment should be considered alarming, and efforts should be made to reduce or eliminate them wherever possible (Suess 1976).

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This report synthesizes technical literature on ecological and toxicological aspects of polycyclic aromatic hydrocarbons (PAH) in the environment, with special reference to fisheries and wildlife resources. Subtopics include: chemical properties, sources, and fate; background concentrations in biological and nonbiological samples; toxic and sublethal effects of PAH to flora and fauna; and proposed criteria and research needs for the protection of sensitive, nonhuman organisms.

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