# 5. POTENTIAL FOR HUMAN EXPOSURE

## 5.1 OVERVIEW

This chapter provides a discussion of the environmental fate and potential for human exposure to 17 PAHs. For the purposes of describing environmental fate, these PAHs have been grouped into low, medium, and high molecular weight classes (see Section 5.3.1, Transport and Partitioning). In general, chemicals within each class have similar environmental fates. When available, data are provided for the individual PAHs that are the subject of the profile. When data on each compound are not available, data on members of the weight class are provided. Data regarding total PAHs or generalizations about PAHs are also used to provide insight into the behavior of the compounds cbvered in this profile.

PAHs are released to the environment through natural and synthetic sources with emissions largely to the atmosphere. Natural sources include emissions from volcanoes and forest fires. Synthetic sources provide a much greater release volume than natural sources; the largest single source is the burning of wood in homes. Automobile and truck emissions are also major sources of PAHs. Environmental tobacco smoke, unvented radiant and convective kerosene space heaters, and gas cooking and heating appliances may be significant sources of PAHs in indoor air. Hazardous waste sites can be a concentrated sources of PAHs on a local scale. Examples of such sites are abandoned wood-treatment plants (sources of creosote) and former manufactured-gas sites (sources of coal tar). PAHs can enter surface water through atmospheric deposition and from discharges of industrial effluents (including wood-treatment plants), municipal waste water, and improper disposal of used motor oil. Several of the PAHs have been detected at hazardous waste sites at elevated levels. In air, PAHs are found sorbed to particulates and as gases. Particle-bound PAHs can be transported long distances and are removed from the atmosphere through precipitation and dry deposition. PAHs are transported from surface waters by volatilization and sorption to settling particles. The compounds are transformed in surface waters by photooxidation, chemical oxidation, and microbial metabolism. In soil and sediments, microbial metabolism is the major process for degradation of PAHs. Although PAHs are accumulated in terrestrial and aquatic plants, fish, and invertebrates, many animals are able to metabolize and eliminate these compounds. Bioconcentration factors (BCFs), which express the concentration in tissues compared to concentration in media, for fish and crustaceans are frequently in

the 10-10,000 range. Food chain uptake does not appear to be a major source of exposure to PAHs for aquatic animals.

The greatest sources of exposure to PAHs for most of the United States population are active or passive inhalation of the compounds in tobacco smoke, wood smoke, and contaminated air, and ingestion of the compounds in foodstuffs. The general population may also be exposed to PAHs in drinking water and through skin contact with soot and tars. Higher than background levels of PAHs are found in foods that are grilled or smoked. Estimates of human exposures to PAHs vary. The average total daily intake of PAHs by a member of the general population has been estimated to be 0.207 µg from air, 0.027 µg from water, and 0.16-l .6 µg from food. The total potential exposure to carcinogenic PAHs for adult males in the United States was estimated to be 3 µg/day. Smokers of unfiltered cigarettes may experience exposures twice as high as these estimates. Persons living in the vicinity of hazardous waste sites where PAHs above background levels have been detected may also be exposed to higher levels.

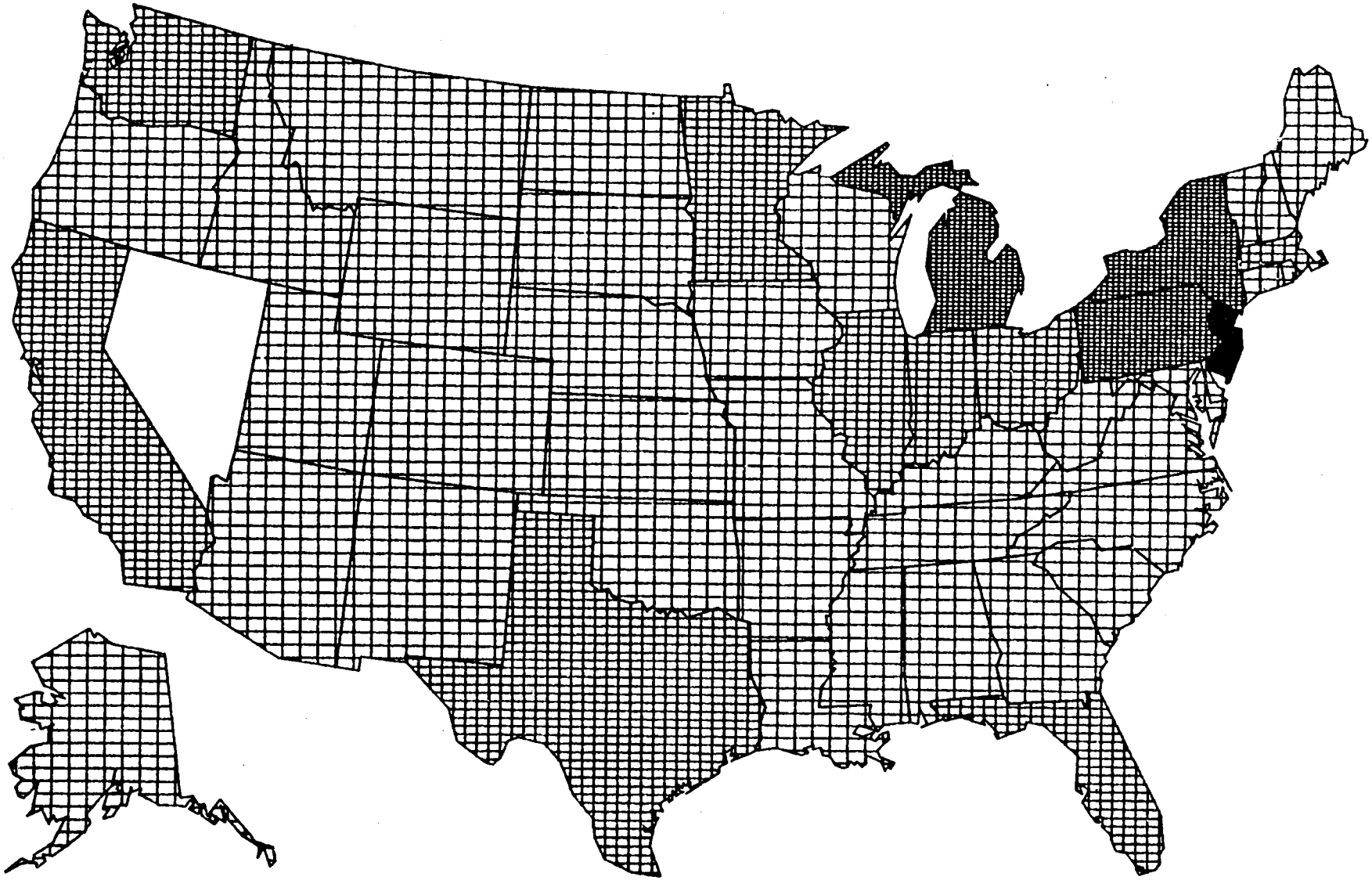
PAHs have been identified in at least 600 of the 1,408 hazardous waste sites that have been proposed for inclusion in the EPA National Priorities List (NPL) (HazDat 1994). However, the number of sites evaluated for PAHs is not known. The frequencies of these sites can be seen in Figure 5-1.

## 5.2 RELEASES TO THE ENVIRONMENT

### 5.2.1 Air

Most of the direct releases of PAHs to the environment are to the atmosphere from both natural and anthropogenic sources, with emissions from human activities predominating. PAHs in the atmosphere are mostly associated with particulate matter; however, the compounds are also found in the gaseous phase (NRC 1983; Yang et al. 1991). The primary natural sources of airborne PAHs are forest fires and volcanoes (Baek et al. 1991; NRC 1983). The residential burning of wood is the largest source of atmospheric PAHs (Peters et al. 1991; Ramdahl et al. 1982); releases are primarily the result of inefficient combustion and uncontrolled emissions (Freeman and Cattell 1990; NRC 1983; Tan et al. 1992). Other important stationary anthropogenic sources include industrial power generation, incineration (Shane et al. 1990; Wild et al. 1992); the production of coal tar, coke, and asphalt; and petroleum catalytic cracking (Baek et al. 1991; Guerin 1978; Perwak et al. 1982; Santodonato 1981).

## FIGURE 5-1. FREQUENCY OF NPL SITES WITH PAHs CONTAMINATION*




*Derived from HazDat 1994

Environmental tobacco smoke, unvented radiant and convective kerosene space heaters, and gas cooking and heating appliances may be important sources of PAHs in indoor air (Chuang et al. 1991; Hoffmann and Hoffmann 1993; Mumford et al. 1991; NRC 1986; Traynor et al. 1990). Stationary sources account for about 80% of total annual PAH emissions; the rest are from mobile sources. The most important mobile sources of PAHs are vehicular exhaust from gasoline and diesel-powered engines (Baek et al. 1991; Johnson 1988; Yang et al. 1991). Mobile sources are often the major contributors to PAH releases to the atmosphere in urban or suburban areas (Baek et al. 1991). The amount of anthracene released to the atmosphere in 1992 by U.S. industrial facilities sorted by state is given in Table 5-l (TRI92 1994). The TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list. TRI92 (1994) data were not available for other PAHs included in this profile.

The U.S. annual emissions (from early to mid 1970s) of polycyclic organic matter (a term generally used to describe PAHs, their nitrogen-containing analogs, and their quinone degradation products [Santodonato et al. 1981]) were estimated by NRC (1983) as follows: open buming 4,024 metric tons (39%), residential heating-3,956 metric tons (38%), automobiles and trucks-2,266 metric tons (22%), and industrial boilers-74 metric tons (1%). NRC (1983) estimated that the total amount of benzo[a]pyrene produced in the United States is between 300 and 1,300 metric tons annually. Peters et al. (1981) estimated that a total of 11,031 metric tons of PAHs were released to the atmosphere in the United States on an annual basis, with 36% of the total coming from residential heating, 6% from industrial processes, 1% from incineration, 36% from open burning, 1% from power generation, and 21% from mobile sources. This estimate can be compared to that of Ramdahl et al. (1982), who reported that a total of 8,598 tons of PAHs were emitted to the atmosphere annually from the following sources: (1) residential heating-16%; (2) industrial processes 41%; (3) incineration-l % (4) open burning-13%; (5) power generation-5%; and (6) mobile sources-25%.

The composition of PAH emissions varies with the combustion source. For example, emissions from residential wood combustion contain more acenaphthylene than other PAHs (Perwak et al. 1982), whereas auto emissions contain more benzo[g,h,i]perylene and pyrene (Rogge et al. 1993a; Santodonato et al. 1981). PAHs in diesel exhaust particulates are dominated by three- and four-ring compounds, primarily fluoranthene, phenanthrene, and pyrene (Kelly et al. 1993; Rogge et al. 1993a; Westerholm and Li 1994). Diesel exhaust vapor emissions are dominated by phenanthrene and anthracene (Westerholm and Li 1994). Acenaphthene, fluorene, and phenanthrene have been found to

## Table 5-1. Releases to the Environment from Facilities that Manufacture or Process Anthracene

| State[b] | Number of facilities | Range of reported amounts released in pounds per year[a] | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | Air | Water | Land | Underground Injection | Total Environment[c] | POTW Transfer | Off-site Waste Transfer |
| AL | 7 | 2-3500 | 0-5 | 0 | 0 | 2-3505 | 0 | 0-465000 |
| AR | 1 | 327-327 | 0 | 0 | 0 | 327-327 | 0 | 0 |
| CA | 1 | 1-1 | 0 | 0 | 0 | 1-1 | 0 | 0 |
| FL | 2 | 0-702 | 0 | 0 | 0 | 0-702 | 0 | 0-250 |
| IL | 5 | 126-1000 | 0 | 0 | 0 | 126-1000 | 0-250 | 0-10273 |
| IN | 5 | 0-11090 | 0 | 0 | 0 | 0-11090 | 0-88 | 0 |
| KY | 2 | 20-94 | 0-78 | 0 | 0 | 20-172 | 0 | 0-680 |
| LA | 7 | 0-125 | 0-9 | 0 | 0 | 0-125 | 0 | 0-7370 |
| MI | 2 | 85-4085 | 0 | 0 | 0 | 85-4085 | 0-5 | 0-1190 |
| MN | 1 | 160-160 | 0 | 0 | 0 | 160-160 | 0 | 0 |
| MO | 1 | 10-10 | 5-5 | 0 | 0 | 15-15 | 0 | 0 |
| MS | 2 | 0-250 | 108-250 | 0-250 | 0 | 108-750 | 0 | 0 |
| NJ | 3 | 0-272 | 0-5 | 0-7 | 0 | 0-272 | 0 | 2-250 |
| NY | 2 | 0-910 | 0 | 0 | 0 | 0-910 | 0 | 0 |
| OH | 12 | 0-3700 | 0-250 | 0-720 | 0 | 0-4425 | 0-5 | 0-14112 |
| OK | 1 | 0-0 | 0 | 1-1 | 0 | 1-1 | 0 | 5-5 |
| PA | 7 | 0-997 | 0-37 | 0-560 | 0 | 0-1594 | 0 | 0-7900 |
| SC | 2 | 0-255 | 0 | 0 | 0 | 0-255 | 0-250 | 0-145 |
| TX | 13 | 0-2780 | 0 | 0-1532 | 0 | 0-2780 | 0 | 0-493911 |
| UT | 2 | 2-4 | 0 | 0 | 0 | 2-4 | 0 | 0 |
| VI | 1 | 3879-3879 | 15-15 | 0 | 0 | 3894-3894 | 0 | 0 |
| WA | 2 | 34-5395 | 11-250 | 0 | 0 | 45-5645 | 0 | 0-6650 |
| WV | 3 | 10-1500 | 0 | 0 | 0 | 10-1500 | 0 | 0-250 |

Source: TRI92 1994

be predominant in total (particle- and vapor-phase) diesel emissions (Lowenthal et al. 1994). Phenanthrene was the most abundant and frequently detected PAH in samples of fly ash and bottom ash collected from municipal refuse incinerators in the United States (Shane et al. 1990), whereas benzo[g,h,i]perylene was the most abundant and frequently detected PAH in fly ash samples collected from municipal solid waste incinerators in the United Kingdom (Wild et al. 1992). Fluoranthene, benzo[a]fluoranthene, benzo[g,h,i]perylene, indeno[ 1,2,3-c,d]pyrene, phenanthrene, and chrysene were predominant in emission particle samples collected from a municipal waste incinerator, whereas benzo[g,h,i]perylene and benz[a]anthracene were predominant in emission particle samples collected from a municipal and medical/pathological waste incinerator (Williams et al. 1994). Emission particle samples from a pilot scale rotary kiln incinerator charged with polyethylene contained predominantly benz[a]anthracene and phenanthrene when an afterburner was used, whereas pyrene, fluoranthene, and phenanthrene were predominant without an afterburner; total PAH concentrations were reduced by a factor greater than 100 by the use of an afterburner (Williams et al. 1994). In coal tar pitch emissions, concentrations of phenanthrene and pyrene have been reported to be 20-80 times greater than the concentrations of benzo[a]pyrene and benzo[g,h,i]perylene (Sawicki 1962). Chrysene/triphenylene, pyrene, and fluoranthene were dominant among the PAHs found in fine particle emissions from natural gas home appliances (Rogge et al. 1993b). Cigarette mainstream smoke contains a wide variety of PAHs with reported concentrations of benzo[a]pyrene ranging from approximately 5-80 ng/cigarette; sidestream smoke concentrations are significantly higher with sidestream/mainstream concentration ratios for benzo[a]pyrene ranging from 2.5 to 20 (Hoffmann and Hoffmann 1993; IARC 1983).

### 5.2.2 Water

Important sources of PAHs in surface waters include deposition of airborne PAHs (Jensen 1984), municipal waste water discharge (Barrick 1982), urban storm water runoff (MacKenzie and Hunter 1979), runoff from coal storage areas (Stahl et al. 1984; Wachter and Blackwood 1979), effluents .from wood treatment plants and other industries (DeLeon et al. 1986; Snider and Manning 1982; USDA 1980), oil spills (Giger and Blumer 1974), and petroleum pressing (Guerin 1978). Brown and Weiss (1978) estimated that l-2 tons of benzo[a]pyrene were released from municipal sewage effluents and 0.1-0.4 tons of benzo[a]pyrene were released from petroleum refinery waste waters in the United States in 1977.

Most of the PAHs in surface waters are believed to result from atmospheric deposition (Santodonato et al. 1981). However, for any given body of water, the major source of PAHs could vary. Jensen (1984) studied benzo[a]pyrene loading in a marine coastal area and determined that atmospheric deposition was indeed the major source of benzo[a]pyrene, with lesser amounts contributed by refinery effluent, municipal waste water, urban runoff, and rivers. Prahl et al. (1984) found that combustion-derived PAHs adsorbed to suspended sediments in rivers accounted for the major portion of PAHs in the waters of a Washington coastal area, and other studies have identified industrial effluents, road runoff, and oil spills as the major contributors in specific bodies of water (DeLeon et al. 1986; Santodonato et al. 1981).

The amount of anthracene released to surface water and publicly owned treatment works (POTWs) in 1992 by U.S. industrial facilities sorted by state is shown in Table 5-l (TRI92 1994). The TRI data should be used with caution since only certain facilities are required to report. This is not an exhaustive list. TRI92 (1994) data were not available for other PAHs included in this profile. Because most of the PAHs released to aquatic environments tend to remain near the sites of deposition, lakes, rivers, estuaries, and coastal marine environments near centers of human populations and industrial activity tend to be the major repositories of aquatic PAHs (Neff 1979).

### 5.2.3 Soil

Most of the PAHs in soil are believed to result from atmospheric deposition after local and long-range transport. The presence of PAHs in the soil of regions remote from any industrial activity supports this contention (Thomas 1986). Other potential sources of PAHs in soil include sludge disposal from public sewage treatment plants, automotive exhaust, irrigation with coke oven effluent, leachate from bituminous coal storage sites, and use of soil compost and fertilizers (Perwak et al. 1982; Santodonato et al. 1981; Stahl et al. 1984; White and Lee 1980). The principal sources of PAHs in soils along highways and roads are vehicular exhausts and emissions from wearing of tires and asphalt. PAHs may also be released to soils at concentrations above background and landfill sites (Black et al. 1989) and industrial sites, including creosote production (Ellis et al. 1991), wood-preserving (Mueller et al. 1991; Weissenfels et al. 1990), and coking plants (Weissenfels et al. 1990; Werner et al. 1988). Soils at the sites of former manufactured gas plants are also heavily contaminated with PAHs (Bewley et al. 1989; Tumey and Goerlitz 1990).

The amount of anthracene released to surface water and publicly owned treatment works (POTWs) in 1992 by U.S. industrial facilities sorted by state is shown in Table 5-l (TRI92 1994). Based on data in Table 5-1, only relatively small amounts of anthracene were discharged in hazardous waste sites from U.S. industrial facilities in 1992. However, some of the anthracene wastes transferred off-site (see Table 5-l) ultimately may be disposed of on land. The TRI data should be used with caution since only certain facilities are required to report. This is not an exhaustive list. TRI92 (1994) data were not available for other PAHs included in this profile.

## 5.3 ENVIRONMENTAL FATE

### 5.3.1 Transport and Partitioning

The global movement of PAHs can be summarized as follows: PAHs released to the atmosphere are subject to short- and long-range transport and are removed by wet and dry deposition onto soil, water, and vegetation. In surface water, PAHs can volatilize, photolyze, oxidize, biodegrade, bind to suspended particles or sediments, or accumulate in aquatic organisms (with bioconcentration factors often in the 10-10,000 range). In sediments, PAHs can biodegrade or accumulate in aquatic organisms. PAHs in soil can volatilize, undergo abiotic degradation (photolysis and oxidation), biodegrade, or accumulate in plants. PAHs in soil can also enter groundwater and be transported within an aquifer.

Transport and partitioning of PAHs in the environment are determined to a large extent by physicochemical properties such as water solubility, vapor pressure, Henry's law constant, octanol-water partition coefficient ($K_{ow}$), and organic carbon partition coefficient ($K_{oc}$). In general, PAHs have low water solubilities. The Henry's law constant is the partition coefficient that expresses the ratio of the chemical's concentrations in air and water at equilibrium and is used as an indicator of a chemical's potential to volatilize. The $K_{oc}$ indicates the chemical's potential to bind to organic carbon in soil and sediment. The $K_{ow}$ is used to estimate the potential for an organic chemical to move from water into lipid and has been correlated with bioconcentration in aquatic organisms. Some of the transport and partitioning characteristics (e.g., Henry's law constant, $K_{oc}$ values, and $K_{ow}$, values) of the 17 PAHs are roughly correlated to their molecular weights. These properties are discussed by grouping these PAHs as follows:

- Low molecular weight compounds (152-l 78 g/mol)-acenaphthene, acenaphthylene, anthracene, fluorene, and phenanthrene;
- Medium molecular weight compounds (202 g/mol)-fluoranthene and pyrene; and
- High molecular weight compounds (228-278 g/mol)-benz[a]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, benzo[a]pyrene, benzo[e]pyrene, chrysene, dibenz[a,h]anthracene, and indeno[ 1,2,3-c,d]pyrene.

As an example, Hattemer-Frey and Travis (1991) found that the low solubility, low vapor pressure and high $K_{ow}$ of benzo[a]pyrene result in its partitioning mainly between soil (82%) and sediment (17%), with ≈1% partitioning into water and <1% into air, suspended sediment and biota.

PAHs are present in the atmosphere in the gaseous phase or sorbed to particulates. The phase distribution of PAHs in the atmosphere is important in determining their fate because of the difference in rates of chemical reactions and transport between the two phases. The phase distribution of any PAH depends on the vapor pressure of the PAH, the atmospheric temperature, the PAH concentration, the affinity of the PAH for the atmospheric suspended particles ($K_{oc}$), and the nature and concentrations of the particles (Baek et al. 1991). In general, PAHs having two to three rings (naphthalene, acenaphthene, acenaphthylene, anthracene, fluorene, phenanthrene) are present in air predominantly in the vapor phase. PAHs that have four rings (fluoranthene, pyrene, chrysene, benz[a]anthracene) exist both in the vapor and particulate phase, and PAHs having five or more rings (benzo[a]pyrene, benzo[g,h,i]perylene) are found predominantly in the particle phase (Baek et al. 1991; Jones et al. 1992). The ratio of particulate to gaseous PAHs in air samples collected in Antwerp, Belgium, was 0.03 for anthracene, 0.49 for pyrene, 3.15 for summed benz[a]anthracene and chrysene, and 11.5 for summed benzo[a]fluoranthene and benzo[b]fluoranthene (NRC 1983).

Using field data from Osaka, Japan, Pankow et al. (1993) examined the effects of relative humidity. (RH) on measured gas/particle partition coefficients over the range 42% ≥ 95%. They found that for seven PAHs or groups of PAHs (including phenanthrene + anthracene, fluoranthene, and pyrene) sorption decreased with increasing RH.

Atmospheric residence time and transport distance depend on the size of the particles to which PAHs are sorbed and on climatic conditions which will determine rates of wet and dry deposition. About 90-95% of particulate PAHs are associated with particle diameters <3.3 μm, and the peak distributions

are localized between 0.4 and 1.1 μm (Baek et al: 1991). Both coarse particles with aerodynamic diameters >3-5 μm and nucleic particles with diameters <0.1 μm have limited atmospheric residence times. The coarse particles are removed from the atmosphere by wet and dry deposition, while the nucleic particles are removed mainly by coagulation with other nucleic particles or with larger particles, followed by wet and dry deposition. Particles with a diameter range of 0.1-3.0 μm, with which airborne PAHs are principally associated, remain airborne for a few days or longer, due to slower dry deposition and less efficient wet deposition (Baek et al. 1991). Therefore, airborne particulate PAHs in this size range can transport long distances (Lunde and Bjorseth 1977). Larger particles emitted from urban sources tend to settle onto streets and become part of urban runoff. However, PAHs in urban air are primarily associated with submicrometer-diameter soot particles that have residence times of weeks and are subject to long-range transport (Butler and Crossley 1981). Long-range transport of PAHs was examined by Lunde and Bjorseth (1977), Bjorseth et al. (1978a), and Bjorseth and Olufsen (1983) who found that PAHs originating in Great Britain had been transported as far as Norway and Sweden.

The relative importance of wet and dry deposition in removing PAHs from the atmosphere varies with the individual PAH. For example, Perwak et al. (1982) estimated that a total of 23% of benzo[a]pyrene released to the atmosphere is deposited on soil and water surfaces. Dry deposition of benzo[a]pyrene adsorbed to atmospheric aerosols accounts for most of the removal; wet deposition is less significant by a factor of 3-5. In a mass balance study of the atmospheric deposition of PAHs to Siskiwit Lake, which is located on a wilderness island in northern Lake Superior, dry aerosol deposition of particulate phase PAHs was found to be the predominant form of input to surface waters by an average ratio of 9:l over wet deposition (McVeety and Hites 1988).

PAH compounds tend to be removed from the water column by volatilization to the atmosphere, by binding to suspended particles or sediments, or by being accumulated by or sorbed onto aquatic biota. The transport of PAHs from water to the atmosphere via volatilization will depend on the Henry's law constants (Hs) for these compounds. The low molecular weight PAHs have Henry's law constants in the range of $10^{-3}$-$10^{-5}$ atm-$m^3$/mol; medium molecular weight PAHs have constants in the $10^{-6}$ range; and high molecular weight PAHs have values in the range of $10^{-5}$-$10^{-8}$. Compounds with values ranging from $10^{-3}$ to $10^{-5}$ are associated with significant volatilization, while compounds with values less than $10^{-5}$ volatilize from water only to a limited extent (Lyman et al. 1982). Half-lives for volatilization of benz[a]anthracene and benzo[a]pyrene (high molecular weight PAHs) from water have

been estimated to be greater than 100 hours (Southworth 1979). Southworth et al. (1978) stated that lower molecular weight PAHs could be substantially removed by volatilization if suitable conditions (high temperature, low depth, high wind) were present. Southworth (1979) estimated half-lives for volatilization of anthracene (a low molecular weight PAH) of 18 hours in a stream with moderate current and wind, versus about 300 hours in a body of water with a depth of 1 meter and no current. Even for PAHs susceptible to volatilization, other processes, such as adsorption, photolysis or biodegradation (see Section 5.3.2.2) may become more important than volatilization in slow-moving, deep waters.

Because of their low solubility and high affinity for organic carbon, PAHs in aquatic systems are primarily found sorbed to particles that either have settled to the bottom or are suspended in the water column. It has been estimated that two-thirds of PAHs in aquatic systems are associated with particles and only about one-third are present in dissolved form (Eisler 1987). In an estuary, volatilization and adsorption to suspended sediments with subsequent deposition are the primary removal processes for medium and high molecular weight PAHs, whereas volatilization and biodegradation (see Section 5.3.2.2) are the major removal processes for low molecular weight compounds (Readman et al. 1982). In an enclosed marine ecosystem study, less than 1% of the original amount of radiolabeled benz[a]anthracene added to the system remained in the water column after 30 days; losses were attributed to adsorption to settling particles and to a lesser extent to photodegradation (Hinga and Pilson 1987).

Baker et al. (1991) found that several PAHs were significantly recycled in the water column of Lake Superior. Fluorene and phenanthrene were rapidly removed from surface waters and settled through the water column to the sediment-water interface where a large fraction of the recently settled contaminants were released back into the water column. Higher molecular weight PAHs were found to have lower settling fluxes, but these compounds were efficiently buried in the surficial sediments with little recycling. Settling particles were found to be greatly enriched in hydrophobic organic chemicals.

The $K_{oc}$ of a chemical is an indication of its potential to bind to organic carbon in soil and sediment. The low molecular weight PAHs have $K_{oc}$ values in the range of $10^3$-$10^4$, which indicates a moderate potential to be adsorbed to organic carbon in the soil and sediments. The medium molecular weight compounds have $K_{oc}$ values in the lo4 range. High molecular weight PAHs have $K_{oc}$ values in the

range of $10^5$-$10^6$, which indicates stronger tendencies to adsorb to organic carbon (Southworth 1979). PAHs from lands cleared by slash and bum methods have been found to be deposited in charred litter and to move into soils by partitioning and leaching (Sullivan and Mix 1985). Phenanthrene and fluoranthene (low and medium molecular weight PAHs, respectively) from these areas were incorporated into soil to a greater extent (i.e., less strongly adsorbed to organic carbon in the charred litter) than high molecular weight PAHs such as benzo[g,h,i]pyrene and indeno[ 1,2,3-c,d]pyrene.

Because mobile colloids may enhance the mobility in porous medias of hydrophobic pollutants such as PAHs, Jenkins and Lion (1993) tested bacterial isolates from soil and subsurface environments for their ability to enhance transport of phenanthrene in aquifer sand. The most mobile isolates tested significantly enhanced the transport of phenanthrene, as a model PAH, in sand.

Sorption of PAHs to soil and sediments increases with increasing organic carbon content and with increasing surface area of the sorbent particles. Karickhoff et al. (1979) reported adsorption coefficients for sorption of pyrene to sediments as follows: sand-9.4-68; silt-1,500-3,600; and clay-l ,400-3,800. Gardner et al. (1979) found that from three to four times more anthracene and about two times more fluoranthene, benz[a]anthracene, and benzo[a]pyrene were retained by marsh sediment than by sand.

PAHs may also volatilize from soil. Volatilization of acenaphthene, acenaphthylene, anthracene, fluorene, and phenanthrene (low molecular weight PAHs) from soil may be substantial (Coover and Sims 1987; Southworth 1979; Wild and Jones 1993). However, of 14 PAHs studied in two soils, volatilization was found to account for about 20% of the loss of 1-methylnaphthalene and 30% of the loss of naphthalene; volatilization was not an important loss mechanism for anthracene, phenanthrene, fiuoranthene, pyrene, chrysene, benz[a]anthracene, benzo[b]fluoranthene, dibenz[a,h]anthracene, benzo[a]pyrene, and indeno[l,2,3-c,d]pyrene (Park et al. 1990).

Physicochemical properties of several phenanthrene and anthracene metabolites [ 1-hydroxy-2-napthoic acid (lH2NA); 2,3-dihydroxy naphthalene (23DHN); 2-carboxy benzaldehyde (2CBA); and 3,4-dihydroxy benzoic acid (34DHBA)] were experimentally measured and/or estimated and used with a Fugacity Level 1 model to estimate the distribution of the metabolites and their parent compounds in a contaminated soil (Ginn et al. 1994). The volumes of the air, water and soil phases were assumed to be 20%, 30%, and 48%, respectively. A volume of 2% was assumed for nonaqueous phase liquid

(NAPL) phase. The parent compounds, anthracene and phenanthrene, had the greatest tendency to be associated with the NAPL and soil phases. The polar metabolites of phenanthrene, lH2NA and 34DHBA, were associated more with the water phase of the subsurface. The metabolites 2CBA and 23DHN had a stronger affinity for the NAPL phase than for the water phase.

PAHs have been detected in groundwater either as a result of migration directly from contaminated surface waters or through the soil (Ehrlich et al. 1982; Wilson et al. 1986). Fluorene from an abandoned creosote pit was found to migrate through sand and clay into groundwater (Wilson et al. 1986). PAHs have also been shown to be transported laterally within contaminated aquifers (Ehrlich et al. 1982).

PAHs can be accumulated in aquatic organisms from water, sediments, and food. Bioconcentration factors (BCFs) for several species of aquatic organisms are listed in Table 5-2. In fish and crustaceans BCFs have generally been reported in the range of 10-10,000 (Eisler 1987). In general, bioconcentration was greater for the higher molecular weight compounds than for the lower molecular weight compounds. Bioconcentration experiments performed with radiolabeled compounds may overestimate the BCFs of some PAHs. For example, Spacie et al. (1983) estimated BCFs of 900 for anthracene and 4,900 for benzo[a]pyrene in bluegills (whole body) based on total radiolabeled carbon ($^{14}C$) activity. However, the estimated BCFs based only on the parent compounds were 675 and 490, respectively, indicating that biotransformation of the parent compounds occurred in addition to bioconcentration. Biotransformation by the mixed function oxidase (MFO) system in the fish liver can result in the formation of carcinogenic and mutagenic intermediates; exposure to PAHs has been linked to the development of tumors in fish (Eisler 1987). The ability of fish to metabolize PAHs may explain why benzo[a]pyrene frequently is not detected or found only at very low levels in fish from environments heavily contaminated with PAHs (Varanasi and Gmur 1980, 1981). The breakdown products (polyhydroxy compounds) are eliminated in feces (via bile) and urine. Although fish and most crustaceans evaluated to date have the MFO system required for biotransformation of PAHs, some molluscs and other aquatic invertebrates are unable to metabolize PAHs efficiently (Varanasi et al. 1985). Varanasi et al. (1985) ranked the extent of benzo[a]pyrene metabolism by aquatic organisms as follows: fish > shrimp > amphipod crustaceans > clams. Half-lives for elimination of PAHs in fish ranged from >2 days to 9 days (Niimi 1987).

## TABLE 5-2. Polycyclic Aromatic Hydrocarbon (PAHs) Bioconcentration Factors (BCFs) for Selected Species of Aquatic Organisms[a]

| PAH compound and organism | Exposure period[b] | BCF |
|---|---|---|
| **ANTHRACENE** | | |
| Mayfly, *Hexagenia* sp. | 28 h | 3,500 |
| Cladoceran, *Daphnia pulex* | 24 h | 760–1,200 |
| Cladoceran, *Daphnia magna* | 60 m | 200 |
| Fathead minnow, *Pimephales promelas* | 2–3 d | 485 |
| Rainbow trout, *Salmo gairdneri* | 72 h | 4,400–9,200 |
| **BENZ(*a*)ANTHRACENE** | | |
| Cladoceran, *D. pulex* | 24 h | 10,109 |
| **BENZO(*a*)PYRENE** | | |
| Midge, *Chironomus riparius*, larvae | 8 h | 166 |
| Mosquito, *Culex pipiens quinquefasciatus* | 3 d | 11,536 |
| Alga, *Oedogonium cardiacum* | 3 d | 5,258 |
| Periphyton, mostly diatoms | 24 h | 9,600 |
| Cladoceran, *D. pulex* | 3 d | 134,248 |
| Cladoceran, *D. magna* | 6 h | 2,837 |
| Snail, *Physa* sp. | 3 d | 82,231 |
| Clam, *Rangia cuneata* | 24 h | 9–236 |
| Oyster, *Crassostrea virginica* | 14 d | 242 |
| Northern pike *Esox lucius* | 3.3 h–23 d | <55 |
| Mosquitofish, *Gambusia affinis* | 3 d | 930 |
| Bluegill, *Lepomis macrochirus* | 4 h | 12 |
| Bluegill, *L. macrochirus* | | |
| No dissolved humic material (DHM) | 48 h | 2,657 |
| 20 mg/L DHM | 48 h | 225 |
| **CHRYSENE** | | |
| Clam, *Rangia cuneata* | 24 h | 8 |
| Pink shrimp, *Penaeus duorarum* | | |
| Cephalothorax | 28 d | 248–361 |
| Cephalothorax | 28 d + 28 d postexposure | 21–48 |
| Abdomen | 28 d | 84–199 |
| Abdomen | 28 d + 28 d postexposure | 22–91 |

## TABLE 5-2. Polycyclic Aromatic Hydrocarbon (PAHs) Bioconcentration Factors (BCFs) for Selected Species of Aquatic Organisms[a] (continued)

| PAH compound and organism | Exposure period[b] | BCF |
|---|---|---|
| **FLUORENE** | | |
| Bluegill | 30 d | 200–1,800 |
| **PHENANTHRENE** | | |
| Cladoceran, *D. pulex* | 24 h | 325 |
| Clam, *R. cuneata* | 24 h | 32 |
| **PYRENE** | | |
| Cladoceran, *D. pulex* | 24 h | 2,702 |

[a]Data summary from multiple studies; adapted from Eislen (1987)

[b]m = minutes; h = hours; d = days

Mollusks also eliminate accumulated PAHs. Neff (1982) reported that oysters (*Crassostrea gigas*) eliminated the following percentages of accumulated PAHs during a 7-day elimination period: benzo[a]pyrene-0%; benz[a]anthracene-32%; fluoranthene-66%; and anthracene-79%.

Fish and crustaceans readily assimilate PAHs from contaminated food, whereas mollusks and polychaete worms have limited assimilation (Eisler 1987). Biomagnification (a systematic increase in tissue concentrations moving up a food chain) has not been reported because of the tendency of many aquatic organisms to eliminate these compounds rapidly (Eisler 1987). In general, PAHs obtained from the diet contribute to total tissue concentrations only to a limited extent. For example, food chain uptake of anthracene by fathead minnows *(Pimephales promelas)* consuming water fleas *(Daphnia pulex)* was estimated to be about 15% of the amount accumulated from the water (Southworth 1979). In a simple aquatic food chain involving seston (i.e., organic and inorganic particulate matter >0.45 μm), blue mussels, and the common eider duck, significant changes were observed in the composition of 19 PAHs moving through the trophic levels. Decreasing PAH concentrations were found with increasing trophic level, probably as a result of the selective biotransformation capacity of the organisms for different PAHs. The high theoretical flux of PAHs through the food chain did not result in increasing concentrations with increasing trophic level (i.e., biomagnification was not observed), indicating rapid biotransformation of the compounds (Broman et al. 1990).

Sediment-associated PAHs can be accumulated by bottom-dwelling invertebrates and fish (Eisler 1987). For example, Great Lakes sediments containing elevated levels of PAHs were reported by Eadie et al. (1983) to be the source of the body burdens of the compounds in bottom-dwelling invertebrates. Varanasi et al. (1985) found that benzo[a]pyrene was accumulated in fish, amphipod crustaceans, shrimp, and clams when estuarine sediment was the source of the compound. Approximate tissue to sediment ratios were 0.6-1.2 for amphipods, 0.1 for clams, and 0.05 for fish and shrimp.

Some terrestrial plants can take up PAHs from soil via the roots or from air via the foliage; uptake rates are dependent on the concentration, solubility, and molecular weight of the PAH and on the plant species (Edwards 1983). Mosses and lichens have been used to monitor atmospheric deposition of PAHs (Thomas et al. 1984). About 30-70% of atmospheric PAHs (indeno[l,2,3-c,d]pyrene, fluoranthene, and benzo[a]pyrene) deposited on a forest were sorbed onto tree foliage (i.e., leaves and

needles) and then deposited as falling litter (Matzner 1984). Vaughan (1984) stated that atmospheric deposition on leaves often greatly exceeds uptake from soil by roots as a route of PAH accumulation.

The uptake of PAHs from soil to plants and the subsequent biomagnification is generally quite low (Sims and Overcash 1983). Ratios of PAH concentrations in vegetation to those in soil have been reported to range from 0.001 to 0.18 for total PAHs and from 0.002 to 0.33 for benzo[a]pyrene (Edwards 1983). ,In a study of PAH uptake from cropland soils conducted in the United Kingdom, elevated concentrations of PAHs in soils were not correlated with concentrations in plant tissues (Wild et al. 1992). The cropland soils had received repeated applications of PAHs in sewage sludge that was applied to the soils over a number of years. PAH content of the soils substantially increased as a result of the sludge amendments, and residues of some PAHs persisted in the soils for years. Tissues from plants grown in the treated soils were relatively enriched with low molecular weight PAHs (e.g., acenaphthene, fluorene, phenanthrene), but increased PAH concentrations (relative to tissues from plants grown in control plots that did not receive sludge amendments) were not consistently detected. The PAH concentrations in aboveground plant parts were not strongly related to soil PAH levels but were probably the result of atmospheric deposition. The presence of PAHs in root crop tissues was probably due to adsorption of the compounds to root surfaces. In a similar study, Wild and Jones (1993) used carrots. (Daucus carotu) as a test crop to investigate the potential for PAHs to move from sewage sludge amended soil into the human food chain. Due to the over-riding influence of atmospheric delivery of PAHs, there was no evidence that sludge application increased the PAH concentration of the foliage. Low molecular weight PAHs such as fluoranthene and pyrene were relatively enriched in the peel, probably because of their greater bioavailability. Transfer of PAHs from the root peel to the core appeared to be minimal. This again suggests that simple adsorption onto the peel maybe an important process.

Simonich and Hites (1994a) studied the partitioning of PAHs between vegetation and the atmosphere throughout the growing season and under natural conditions. They found the partitioning process to be dependent primarily upon the atmospheric gas-phase PAH concentration and the ambient temperature. During the spring and fall, when ambient temperatures are low, gas-phase PAHs partition into vegetation. In the summer, some PAHs volatilize and return to the atmosphere. They also developed a mass-balance model for PAHs in the northeastern United States and published values for PAH concentrations and fluxes in air, water, sediments, and soils (Simonich and Hites 1994b). Their model showed that 44 ± 18% of PAHs emitted into the atmosphere from sources in the region studied

were removed by vegetation. They further hypothesized that most of the PAHs absorbed by vegetation at the end of the growing season are incorporated into the soil and permanently removed from the atmosphere.

PAHs may accumulate in terrestrial animals through the food chain or by ingestion of soil. The environmental fate of creosote coal tar distillate (which contained 21% phenanthrene and 9% acenaphthene) was studied in a terrestrial microcosm containing soil, rye grass, insects, snails, mealworm larvae, and earthworms by Gile et al. (1982). Two gray-tailed voles (*Microtus canicaudus)* were added 54 days after the start of the experiment, which continued for 19-26 more days. Average surface soil concentrations (measured on an unspecified day) were 0.60 ppm (phenanthrene) and 1.19 ppm (acenaphthene). During the last 3 days of the experiment, the following phenanthrene concentrations were measured: snail-3.27 ppm; pill bugs-l .72 ppm; and earthworm-l 8.30 ppm. The acenaphthene concentrations measured were as follows: snail-11.2 ppm (day 37); pill bugs-0.99 ppm (day 75); and earthworm-71.9 ppm (days 72-75). The whole body concentration in the vole analyzed for phenanthrene was 7.20 ppm; in the vole analyzed for acenaphthene it was 37.00 ppm. The authors found that these compounds were not metabolized in this system. Whole body concentrations in the vole exceeded soil concentrations by a factor of 12 for phenanthrene and 31 for acenaphthene; however, most of the radiolabeled acenaphthene was found as bound residues in the gastrointestinal tract of the animal and, therefore, was not accumulated.

### 5.3.2 Transformation and Degradation

#### 5.3.2.1 Air

The processes that transform and degrade PAHs in the atmosphere include photolysis and reaction with $NO_x$, $N_2O_5$, OH, ozone, sulfur dioxide, and peroxyacetyl nitrate (Baek et al. 1991; NRC 1983). Possible atmospheric reaction products are oxy-, hydroxy-, nitro- and hydroxynitro-PAH derivatives (Baek et al. 1991). Photochemical oxidation of a number of PAHs has been reported with the formation of nitrated PAHs, quinones, phenols, and dihydrodiols (Holloway et al. 1987; Kamens et al. 1986). Some of these breakdown products are mutagenic (Gibson et al. 1978). Reaction with ozone or peroxyacetylnitrate yields diones; nitrogen oxide reactions yield intro and dinitro PAHs. Sulfonic acids have also been formed from reaction with sulfur dioxide.

The rates of homogeneous vapor phase chemical reactions are usually faster than heterogeneous chemical reactions of particulate PAHs with sunlight and oxidants in the atmosphere, particularly due to light shielding and stabilizing (toward both oxidation and photolysis) effects in the adsorbed state (Behymer and Hites 1988).

PAHs have a wide range of volatilities and therefore are distributed in the atmosphere between the gas and particle phases. The 24 ring PAHs exist, at least partially, in the gas phase. Atkinson et al. (1991) calculated atmospheric lifetimes (1.44 times the half-life) of several gas-phase PAHs due to reactions with measured or estimated ambient concentrations of OH radicals, $NO_3$ radicals, $N_2O_5$, and $O_3$. Their laboratory studies showed that, for PAHs not containing cyclopenta-fused rings, the major gas-phase process resulting in atmospheric loss will be reaction with the OH radical. Calculated atmospheric lifetimes for acenaphthene, acenaphthylene, phenanthrene, and anthracene were on the order of a few hours. Nighttime reaction with $N_2O_5$ was estimated to be a minor source of atmospheric loss. The reactions of PAHs, including fluoranthene and pyrene, with the OH radical (in the presence of $NO_x$) and with $N_2O_5$ led to the formation of nitroarenes that have been identified in the ambient air. As a class of compounds, the nitrated PAHs have been found to be much more mutagenic than their parent PAHs (Kamens et al. 1993).

Most PAHs in the atmosphere .are associated with particulates (Baek et al.. 1991). Vu-Due and Huynh (1991) describe two types of chemical reactions that appear to be the predominant mode of transformation of these PAHs: (1) reactions between PAHs adsorbed on the particle surfaces and oxidant gases like $NO_2$, $O_3$, and $SO_3$ that do not appear to be influenced by exposure to UV irradiation and (2) photooxidation of PAHs irradiated either under solar radiation or simulated sunlight which produces a variety of oxidized derivatives such as quinones, ketones, or acids. Kamens et al. (1990) estimate that, even in highly polluted air, photolysis is the most important factor in the decay of particle-sorbed PAHs in the atmosphere, followed by reaction with $NO_2$, $N_2O_5$, and $HNO_3$.

The National Research Council (NRC 1983) noted that compounds adsorbed to soot are more resistant to photochemical reactions than pure compounds. Butler and Crossley (1981) estimated half-lives for degradation of the following PAHs adsorbed to soot particles and exposed to sunlight in air containing 10 ppm nitrogen oxides: benzo[a]pyrene-7 days; benzo[g,h,i]perylene-8 days; benz[a]anthracene-11 days; pyrene-14 days; chrysene-26 days; fluoranthene-27 days; and phenanthrene-30 days. However, Thomas et al. (1968) reported that benzo[a]pyrene adsorbed on

soot was readily photooxidized, with 60% of the compound destroyed within the first 40 minutes of exposure to sunlight. The effect of substrate on PAH photolytic half-lives was investigated by Behymer and Hites (1988). Photolysis of 18 PAHs adsorbed to low-carbon fly ash produced a wjde range of half-lives that indicated a relationship between structure and photochemical reactivity. Photolysis of the same compounds adsorbed to fly ash samples containing >5% carbon produced similar half-lives, indicating that for these fly ash samples, photolysis is dependent on the physical and chemical structure of the adsorbent and independent of PAH structure. The investigators postulated that dark (i.e., high carbon content) substrates stabilize PAHs to photolytic breakdown since they absorb more light, making less light available for photolysis. McDow et al. (1993) hypothesized that PAHs in atmospheric particles may be either dissolved in a liquid organic phase or adsorbed at an organic phase-solid elementai carbon interface. Therefore, the reactivity of PAHs might depend, not only on the surface characteristics of the particle's solid core, but also on the chemical composition of the organic phase that surrounds the core. Experiments revealed that photodegradation of PAHs (including benz [a]anthracene, chrysene, benzo[a]pyrene, benzo[b]fluoranthene, and benzo[k]fluoranthene) in a mixture of methoxyphenols, based on relative amounts collected in actual samples from hardwood burning, was 10-30 times faster than in hexane. Their results demonstrated that variations in chemical composition of different types of particles such as diesel exhaust and wood smoke might strongly affect the reactivity of PAHs. Eisenberg and Cunningham (1985) found that the photochemical reaction products of PAHs (anthracene, phenanthrene, fluoranthene, benz[a]anthracene, chrysene, and benzo[a]pyrene) adsorbed on particulates include singlet oxygen, which may be implicated in the formation of mutagenic compounds.

Some PAHs are degraded by oxidation reactions that have been measured in the dark (to eliminate the possibility of photodegradation). Korfmacher et al. (1980) found that, while fhrorene was completely oxidized, fluoranthene and phenanthrene were not oxidized, and benzo[a]pyrene and anthracene underwent minimal oxidation. These compounds were tested adsorbed to coal fly ash; the authors stated that the form of the compound (adsorbed or pure) and the nature of the adsorbent greatly affected the rate and extent of oxidation.

Several studies have been carried out to investigate the reaction of PAHs with ozone at ambient concentrations (Baek et al. 1991). Alebic-Juretic et al. (1990) found degradation of PAHs on particle surfaces by ozone to be an important pathway for their removal from the atmosphere. Half-lives of PAHs obtained under laboratory conditions were used to predict lifetimes in an atmosphere containing

a constant ozone concentration of 0.05 ppm. The predicted lifetimes were 3 hours for benzo[a]pyrene, 12 hours for pyrene, and 6 days for fluoranthene. Lane and Katz (1977) investigated the kinetics of the dark reaction of several PAHs with ozone and found the reaction to be extremely fast under simulated atmospheric conditions, with a reported half-life of 0.62 hours for benzo[a]pyrene exposed to 190 ppb of ozone.

In an attempt to determine the atmospheric oxidation processes that would result in an arene oxide functional group in PAHs, Murray and Kong (1994) studied the reaction of particle-bound PAHs with oxidants derived from the reactions of ozone with alkenes. Phenanthrene and pyrene were converted to arene oxides under these simulated atmospheric conditions. Control experiments indicated that the oxidant responsible for the transformation was not ozone, but a product of the reaction of ozone with tetramethylethylene (TME), probably the carbonyl oxide or the dioxirane derived from TME.

#### 5.3.2.2 Water

The most important processes contributing to the degradation of PAHs in water are photooxidation, chemical oxidation, and biodegradation by aquatic microorganisms (Neff 1979). Hydrolysis is not considered to be an important degradation process for PAHs (Radding et al. 1976). The contribution of the individual processes,to the overall fate of a PAH will depend largely on the temperature, depth, pollution status, flow rate, and oxygen content of the water. As a result, a process that is a major loss/degradation process for a particular PAH in a certain surface water may not be so in another surface water with different water quality.

The rate and extent of photodegradation vary widely among the PAHs (Neff 1979). Unfortunately, there is no easily defined trend in the rates of photolysis that could be correlated with the chemical structure of PAHs. For example, the rate of aquatic photolysis of naphthalene containing two benzene rings is much slower than anthracene which contains three benzene rings (Anderson et al. 1986). Based on half-life data, photolysis in water may be an important fate determining process for acenaphthene, acenaphthylene, anthracene, pyrene, benzo[a]pyrene, and benz[a]anthracene relative to the other PAHs discussed in this document (Behymer and Hites 1988; Anderson et al. 1986; Zepp and Schlotzhauer 1983). A study by Nagata and Kondo (1977) reported that anthracene, phenanthrene, and benz[a]anthracene were susceptible to photodegradation, and that benzo[a]pyrene, chrysene, fluorene, and pyrene were resistant to photodegradation. In the photooxidation of PAHs, the most common

reactions result in the formation of peroxides, quinones, and diones (NAS 1972). The major photoproducts of anthracene, phenanthrene, and benz[a]anthracene are anthraquinone, 9,10-phenanthrequinone, and 7,12-benz[a]anthraquinone, respectively (David and Boule 1993).

The rate of photolysis is accelerated by the presence of certain sensitizers (Zepp and Schlotzhauer 1983). Conversely, the rate of photolysis is decreased by the presence of certain quenchers in water (e.g., certain carbonyl compounds). The importance of photolysis will also decrease with the increase of depth in a body of water, particularly in turbid water, because of light attenuation and scattering (Zepp and Schlotzhauer 1979).

Generally, oxidation with singlet oxygen and peroxy radicals are the two important oxidative processes for environmental pollutants in water. The rate constants for reactions of PAHs with singlet oxygen and peroxy radicals (Mabey et al. 1981) and the typical concentrations of the two oxidants in environmental waters (Mill and Mabey 1985) suggest that these reactions may not be important in controlling the overall fate of PAHs in water.

PAHs in water can be chemically oxidized by chlorination and ozonation. A high efficiency of PAH degradation from chlorination has been reported by Harrison et al. (1976a, 1976b) for both laboratory and waste-water treatment plant conditions. Pyrene was the most rapidly degraded PAH. Benz[a]anthracene, benzo[a]pyrene, and perylene were also highly degraded. Indeno[ 1,2,3-c,d]pyrene and benzo[g,h,i]pyrene were intermediate with respect to relative degradation. Benzo[k]fluoranthene and fluoranthene were the most slowly degraded of the compounds tested.

The PAH-related by-products resulting from chlorination are not fully known (Neff 1979). Oyler et al. (1978) identified the following products resulting from the chlorination of PAHs: anthraquinone, a chlorohydrin of fluoranthene; and monochloro derivatives of fluorene, phenanthrene, l-methyl-phenanthrene, and 1-methylnaphthalene. Mori et al. (1993) found that treatment of aqueous benz[a]anthracene (B[a]A) solution with chlorine in both the presence and absence of bromide ion produced a variety of halogenated compounds. The main product was the oxygenated compound, B[a]A-7, l2-dione. A variety of mutagenic halogen substituted and halogen additive (polar) compounds also were produced. The oxidation reaction with chlorine of B[a]A in water was accelerated in the presence of bromide ion.

In water, ozonation is generally slower and less efficient than chlorination in degrading PAHs (Neff 1979). Reaction pathways for ozonation of some PAHs include benz[a]anthracene to 7,12-quinone; benzo[a]pyrene to 3,6-, 1,6-, and 4,5diones; and fluorene to fluorenone (NAS 1972).
In general, PAHs can be significantly metabolized by microbes under oxygenated conditions. However, under anoxic conditions, degradation will be extremely slow (Neff 1979). Concentrations of dissolved oxygen >0.7 mg/L is adequate for biotransformation and the presence of a minimal concentration of PAH is required for biodegradation to proceed (Borden et al. 1989). The minimum total PAH concentration below which biotransformation may be inhibited under ambient nutrient conditions may be 30-70 µg/L (Borden et al. 1989). Some other factors that increase the rates of PAH biodegradation are higher water temperature (summer versus winter) and the presence of adapted microorganisms (Aamand et al. 1989; Anderson et al. 1986; Lee and Ryan 1983). Some PAHs are partially or completely degraded by some species of aquatic bacteria and fungi. The bacterial degradation pathway includes an initial dioxygenase attack to form cis-dihydrodiols (via dioxetane intermediates) that are further oxidized to dihydroxy products. In fungi and mammalian systems (which, unlike bacteria, have cytochrome P-450 enzyme systems), truns-dihydrodiol is produced via an arene oxide intermediate (Anderson et al. 1986; Cemiglia and Heitkamp 1989; Neff 1979). This is significant since the arene oxides have been linked to the carcinogenicity of PAHs. Algae were found to transform benzo[a]pyrene to oxides, peroxides, and dihydrodiols (Kirso et al. 1983; Warshawsky et al. 1983).

Microorganisms in stored groundwater samples completely degraded acenaphthene and acenaphthylene within 3 days (Ogawa et al. 1982). When these reactions occurred under aerobic conditions, there was no evidence of anaerobic degradation of PAHs within the aquifer from which the samples were obtained.

Information on the biodegradation of PAHs by fungi is limited compared to the information that is available about bacteria. However, the fungus *Cunninghamella elegans* has been reported to be capable of metabolizing naphthalene (Cemiglia and Gibson 1979), anthracene, benzo[a]pyrene (Cemiglia and Heitkamp 1989), and fluorene (Pothuluri et al. 1993).

No correlation between biodegradability and molecular weight is evident in three- to four-ring PAHs. For example, phenanthrene with three benzene rings biodegraded in an estuarine water from Savannah,

Georgia, with a half-life of 19 days in August, but anthracene, containing the same number of benzene rings, did not biodegrade at all (Lee and Ryan 1983). Based on estimated reaction rates or half-lives, acenaphthylene, acenaphthylene, and fluorene, the three PAHs that have lower molecular weights than phenanthrene, may not readily biodegrade in water (Lee and Ryan 1983; Mabey et al. 1981). While both naphthalene and phenanthrene biodegraded in water, other PAHs, such as anthracene, benz[a]anthracene, chrysene, and fluorene, did not readily biodegrade in water, but degraded readily in sediment water slurries (Lee and Ryan 1983). On the other hand, PAHs with five or more benzene rings, such as benzo[a]pyrene, dibenz[a,h]anthracene, and benzo[g,h,i]perylene, may not biodegrade readily even in sediment-water slurries (Lee and Ryan 1983; Mabey et al. 1981).

Based on theoretical modeling, photolysis would account for 5% and biodegradation 91% of the transformation/removal of anthracene from deep, slow moving, and somewhat turbid water. The corresponding values in a very shallow, fast-moving, clear water were 47 and 12%, respectively (Southworth 1979).

#### 5.3.2.3 Sediment and Soil

Microbial metabolism is the major process for degradation of PAHs in soil environments. Photolysis, hydrolysis, and oxidation generally are not considered to be important processes for the degradation of PAHs in soils (Sims and Overcash 1983). However, in a study of PAH losses from four surface soils amended with PAHs in sewage sludge, losses due to volatilization and photolysis from sterilized soils were considered to be important for PAHs composed of less than four aromatic rings, whereas abiotic losses were insignificant for PAHs containing four or more aromatic rings (Wild and Jones 1993). Another study that assessed the fate of several PAHs, which included naphthalene, anthracene, phenanthrene, fluoranthene, pyrene, chrysene, benz[a]anthracene, benzo[b]fluoranthene, dibenz[a,h]anthracene, benzo[a]pyrene, dibenzo[a,i]pyrene and indeno[l,2,3-c,d]pyrene, in two soils concluded that abiotic degradation (photolysis and oxidation) accounted for mean losses of 13, 8.3, and 15.8% loss in case of naphthalene, anthracene, and phenanthrene, respectively. No significant abiotic loss was observed for the other PAHs (Park et al. 1990).

The rate and extent of biodegradation of PAHs in soil are affected by environmental factors; the organic content; structure and particle size of the soil; characteristics of the microbial population; the presence of contaminants such as metals and cyanides that are toxic to microorganisms; and the

physical and chemical properties of the PAHs (Wilson and Jones 1993). Based on experimental results, the estimated half-lives (days) of the PAHs in soil were: naphthalene, 2.1-2.2; anthracene, 50-l34; phenanthrene, 16-35; fluoranthene, 268-377; pyrene, 199-260; chrysene, 371-387; benz[a]anthracene, 162-261; benzo[b]fluoranthene, 211-294; benzo[a]pyrene, 229-309; dibenz[a,h]anthracene, 361-420; dibenzo(a,i)pyrene, 232-361; and indeno[l,2,3-c,d]pyrene, 288-289 (Park et al. 1990). Although there are differences in the biodegradation half-life values estimated by different investigators (Park et al. 1990; Wild and Jones 1993; Symons et al. 1988), their results suggest that the biodegradation half-lives of PAH with more than three rings will be considerably longer (>20 days to hundreds of days) than the PAHs with three or fewer rings. Environmental factors that may influence the rate of PAH degradation in soil include temperature, Ph, oxygen concentration, PAH concentrations and contamination history of soil, soil type, moisture, nutrients, and other substances that may act as substrate co-metabolites (Sims and Overcash 1983). The size and composition of microbial populations in turn can be affected by these factors. For example, in low-Ph soils, fungi are dominant over bacteria, and thereby control microbial degradation in these environments. Sorption of PAHs to organic matter and soil particulates also influences bioavailability, and hence, biotransformation potential. Sorption of PAHs by soil organic matter may limit biodegradation of compounds that would otherwise rapidly undergo metabolism (Manila1 and Alexander 1991; Weissenfels et al. 1992).

Although the pathways of microbial degradation are well known for anthracene, benzo[a]pyrene, and phenanthrene, degradation pathways for other PAHs are largely unknown (Sims and Overcash 1983). Metabolism of PAHs by bacteria includes the formation of cis-dihydrodiols through dioxetane intermediates, whereas in fungi (and mammalian systems) trans-dihydrodiols are produced through arene oxide intermediates (Sims and Overcash 1983). MacGillivray and Shiaris (1994) estimated the relative contribution of prokaryotic (bacteria) and eukaryotic (yeast, fungi) microorganisms to PAH biotransformation using phenanthrene as a model compound. They found that the relative contribution of eukaryotic microorganisms to phenanthrene transformation in inoculated sterile sediment was less than 3% of the total activity.

In laboratory studies, Sims et al. (1988) demonstrated extensive degradation of two-ring PAHs in sandy soils, with half-lives of approximately 2 days. The three-ring PAHs, anthracene, and phenanthrene had half-lives of 16 and 134 days, respectively. Four- to six-ring PAHs generally had half-lives >200 days. Anthracene and fluoranthene showed slightly higher biodegradation rates than

benz[a]anthracene or benzo[a]pyrene in a study with fine and medium sands and marsh sediments (Gardner et al. 1979). Degradation rates expressed as a percentage of the mass removed per week for the four compounds were anthracene-2.0-3.0%, fluoranthene-1.9-2.4%, benz[a]anthracene-1.4-l .8%, and benzo[a]pyrene-0.84-1.4%. The ranges of half-lives of phenanthrene and benzo[g,h,i]perylene in four soils amended with PAHs in sewage sludge were 83-193 days and 282-535 days, respectively. Mean half-lives were found to be positively correlated with log $K_{ow}$ and inversely correlated with log water solubility. Previous exposure of the test soils to PAHs enhanced the rate of biodegradation of low molecular weight PAHs but had little effect on the loss of higher molecular weight compounds (Wild and Jones 1993).

Herbes and Schwall (1978) investigated the rates of microbial transformation of PAHs in freshwater sediments from both pristine and oil-contaminated streams. They found that turnover times (l/k) in the uncontaminated sediment were 10-400 times greater than in contaminated sediment. Absolute rates of PAH transformation (micrograms of PAH per gram of sediment per hour) were 3,000-125,000 times greater in the contaminated sediment. Turnover times in the oil-contaminated sediment increased 30-l00-fold per additional ring from naphthalene through benz[a]anthracene; naphthalene was broken down in hours while the turnover times for benz[a]anthracene and benz[a]pyrene were ~400 days and >3.3 years, respectively. Therefore, four- and five-ring PAHs, including the carcinogenic benz[a]anthracene and benz[a]pyrene, may persist even in sediments that have received chronic PAH inputs.

The rate of biodegradation may be altered by the degree of contamination. At hazardous waste sites, half-lives may be longer since other contaminants at the site may be toxic to degrading . microorganisms. Bossert and Bartha (1986) reported reduced biodegradation of PAHs in soil containing a chemical toxic to microorganisms.

Efroymson and Alexander (1994) investigated the effects of nonaqueous phase-liquids (NAPLs) on the biodegradation of hydrophobic compounds, including phenanthrene, in soil and subsoil. Mineralization of phenanthrene in the subsoil was reduced if the compound was dissolved in a NAPL. However, the suppression of the mineralization of phenanthrene in soil by NAPLs was short-lived, suggesting growth of organisms capable of using phenanthrene.

## 5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

### 5.4.1 Air

There is a relatively large body of data characterizing PAH air levels at a variety of U.S. sites. Caution must be used in interpreting and comparing results of different studies, however, because of the different sampling methods used. PAHs occur in the atmosphere in both the particle phase and the vapor phase, as discussed in Section 5.2.1. Three-ring PAH compounds are found in the atmosphere primarily in the gaseous phase, whereas, five- and six-ring PAHs are found mainly in the particle phase; four-ring PAH compounds are found in both phases. To fully characterize atmospheric PAH levels, both particle- and vapor-phase samples must be collected. Many of the earlier monitoring studies used filter sampling methods, which provided information on particle-phase PAH concentrations only, and which did not account for losses of some of the lower molecular weight PAHs by volatilization. As a result, the early use of particulate samples may have resulted in an underestimation of total PAH concentrations. More recent monitoring studies often use sampling methods that collect both particle- and vapor-phase PAHs and that prevent or minimize volatilization losses, thus providing more reliable characterization of total atmospheric PAH concentrations (Baek et al. 1991).

Several monitoring studies indicate that there are higher concentrations of PAHs in urban air than in rural air. Pucknat (1981) summarized 1970 data from the U.S. National Air Surveillance Network and reported that benzo[a]pyrene concentrations in 120 U.S. cities were between 0.2 and 19.3 ng/$m^3$. Ambient benzo[a]pyrene concentrations in nonurban areas ranged between 0.1 and 1.2 ng/$m^3$. More recently, Greenberg et al. (1985) evaluated atmospheric concentrations of particulate phase PAHs at four New Jersey sites (three urban and one rural) over two summer and winter seasons during 198l-82. Urban PAH concentrations were approximately 3-5 times higher than those at the rural site; in addition, winter concentrations were approximately 5-10 times higher than summer concentrations. Geometric mean concentrations of ten PAHs (benzo[a]pyrene, benzo[e]pyrene, benzo[b]fluoranthene, benzofi]fluoranthene, benzo[k]fluoranthene, benz[a]anthracene, indeno[l,2,3-c,d]pyrene, benzo[g,h,i]perylene, pyrene, and chrysene) ranged from 0.03 to 0.62 ng/$m^3$ in urban areas and from 0.01 to 0.12 ng/$m^3$ in the rural area during the summer seasons. During the winter seasons, geometric mean concentrations of these PAHs ranged from 0.40 to 11.15 ng/$m^3$ in urban areas and from 0.08 to 1.32 ng/$m^3$ in the rural area. Geometric mean concentrations of benzo[a]pyrene ranged from 0.11 to

0.23 ng/$m^3$ (urban) and 0.04 to 0.06 ng/$m^3$ (rural) during the summer seasons, and from 0.69 to 1.63 ng/$m^3$ (urban) and 0.17 to 0.32 (rural) during the winter seasons. A more extensive study by Harkov and Greenberg (1985) of atmospheric benzo[a]pyrene concentrations at 27 New Jersey sites indicated similar differences in mean urban (0.6 ng/$m^3$) and rural (0.3 ng/$m^3$) concentrations. Significant seasonal trends were also observed, with mean benzo[a]pyrene concentrations during the winter more than an order of magnitude greater than during the summer.

Several other studies provide evidence that atmospheric concentrations of particle-phase PAHs are higher in winter than in summer. In a 1981-82 study conducted in the Los Angeles area; atmospheric concentrations of 10 PAHs (anthracene, fluoranthene, pyrene, chrysene, benz[a]anthracene, combined benzo[e]pyrene and perylene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, and combined benzo[g,h,i]perylene, and indeno[l,2,3-c,d]pyrene) ranged from 0.14 to 1.45 ng/$m^3$ (with an average of 0.43 ng/$m^3$) during the summer (August-September), and from 0.40 to 4.46 ng/$m^3$ (with an average of 1.28 ng/$m^3$) during the winter (February-March) (Grosjean 1983). A similar seasonal variation in particle-phase PAH concentrations in the Los Angeles atmosphere was seen in an earlier 1974-75 study (Gordon 1976). Quarterly geometric mean concentrations of 11 PAHs (pyrene, fluoranthene, benz[a]anthracene, chrysene, benzo[a]pyrene, benzo[e]pyrene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, and indeno[ 1,2,3-c,d]pyrene) ranged from 0.06 to 2.71 ng/$m^3$ (with an average of 0.45 ng/$m^3$) during the May-October period, and from 0.26 to 8.25 ng/$m^3$ (with an average of 1.46 ng/$m^3$) during the November-April period. The highest and lowest concentrations were observed during the fourth (November-January) and second (May-July) quarters, respectively. Ratios of fourth quarterly and second quarterly geometric mean concentrations ranged from 3.9 for indeno[l,2,3-c,d]pyrene to 7.5 for benzo[a]pyrene and 9.8 for benz[a]anthracene. Possible factors contributing to these seasonal variations in PAH levels include the following: changes in emission patterns; changes in meteorological conditions (i.e., daylight hours and temperature); and changes in space heating emissions, volatilization, and photochemical activity.

Certain monitoring data suggest that ambient levels of some PAHs may be decreasing. Faoro and Manning (1981) analyzed a limited sample of U.S. National Air Surveillance Network data updated through 1977, which indicated that benzo[a]pyrene concentrations have shown consistent, sizable declines during the period from 1967 to 1977 at 26 urban sites and 3 background sites studied (data not provided).

Over the past two decades, the ambient air levels of PAHs in a number of major cities have been characterized. Although data from studies in different areas cannot be used to indicate definitive temporal trends in PAH air levels, a comparison of the results of these studies yields no strong suggestion that the ambient air levels of PAHs may be decreasing, except in traffic tunnels.

In a 1981-82 study that characterized air levels of 13 PAHs in Los Angeles, Grosjean (1983) reported mean ambient particle-phase PAH concentrations ranging from 0.32 ng/$m^3$ for benzo[k]fluoranthene to 3.04 ng/$m^3$ for combined benzo[g,h,i]perylene and indeno[ 1,2,3-c,d]pyrene. Mean concentrations of anthracene, fluoranthene, pyrene, chrysene, benz[a]anthracene, combined perylene and benzo[e]pyrene, benzo[b]fluoranthene, and benzo[a]pyrene were 0.54; 0.94, 1.62, 0.97, 0.48, 0.43, 0.94, and 0.64 ng/$m^3$, respectively. Similar results were obtained in an earlier (1974-1975) study of atmospheric particle-phase PAHs in the Los Angeles area, where ambient annual geometric mean concentrations ranged from 0.17 ng/$m^3$ for benzo[j]fluoranthene to 3.27 ng/$m^3$ for benzo[g,h,i]perylene (Gordon 1976). The annual geometric mean concentration of benzo[a]pyrene was 0.46 ng/$m^3$; most individual PAHs had annual geometric mean concentrations of <0.6 ng/$m^3$. The relatively high levels of benzo[g,h,i]perylene found in these studies.have been attributed to high levels of automobile emissions, which are known to contain high levels of benzo[g,h,i]perylene relative to other PAHs (Santodonato et al. 1981). During the same time period, Fox and Staley (1976) reported somewhat higher ambient average concentrations of particle-phase PAHs in College Park, Maryland, ranging from 3.2 ng/$m^3$ for benzo[a]pyrene to 5.2 ng/$m^3$ for pyrene.

In a 1985-86 study, reported average ambient concentrations (combined particle- and vapor-phase) of eight PAHs in Denver ranged between 0.83 ng/$m^3$ for benzo[k]fluoranthene and 39 ng/$m^3$ for phenanthrene (Foreman and Bidleman 1990). In a study conducted in Hamilton, Ontario, between May 1990 and June 1991, the concentrations of PAHs in respirable air particulate samples were found to range from 0.6 ng/$m^3$ for phenanthrene to 4.3 ng/$m^3$ for benzo[g,h,i]perylene, and 5.1 ng/$m^3$ for combined benzo[b,j,k]fluoranthenes (Legzdins et al. 1994). In a recent limited study, mean concentrations of particle-phase PAHs in New York City air were reported to range from 0.11 ng/$m^3$ for anthracene to 4.05 ng/$m^3$ for benzo[g,h,i]perylene (Tan and Ku 1994).

Atmospheric PAH concentrations have been found to be significantly elevated in areas of enclosed traffic tunnels. In a 1985-86 study in the Baltimore Harbor Tunnel the average concentrations of particle-phase PAHs ranged from 2.9 ng/$m^3$ for anthracene to 27 ng/$m^3$ for pyrene (Benner and

Gordon, 1989). These values are up to an order of magnitude lower than those obtained in 1975 by Fox and Staley (1976), which ranged from 66 ng/$m^3$ for benzo[a]pyrene to 120 ng/$m^3$ for pyrene. Benner and Gordon (1989) postulated that the observed decrease in PAH concentrations over the 1975-85 decade resulted from the increasing use of catalytic converters in U.S. automobiles over that period, These authors also reported concentrations of PAHs in a typical vapor-phase sample from the Boston Harbor Tunnel for four PAHs included in this profile: anthracene (32.3 ng/$m^3$), fluoranthene (25.6 ng/$m^3$), phenanthrene (184 ng/$m^3$), and pyrene (28.3 ng/$m^3$). They emphasized that the vapor-phase samples included PAHs inherently present in the vapor phase as well as the more volatile 3- and 4-ring PAHs that may be desorbed from particles during sampling. These results underscore the need to evaluate both particle- and vapor-phase samples to obtain more reliable estimates of total atmospheric PAH concentrations.

### 5.4.2 Water

PAHs have been detected in surface waters of the United States. In an assessment of STORET data covering the period 1980-82, Staples et al. (1985) reported median concentrations in ambient water of <10 µg/L for 15 PAHs (acenaphthene, acenaphthylene, anthracene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, benzo[a]pyrene, chrysene, fluoranthene, fluorene, indeno[ 1,2,3-c,d]pyrene, naphthalene, phenanthrene,. and pyrene). The number of samples ranged from 630 (naphthalene) to 926 (fluoranthene); the percentage of samples in which these PAHs were detected ranged from 1.0 (benzo[g,h,i]perylene) to 5.0 (phenanthrene) and 7.0 (naphthalene).

Basu and Saxena (1978a) reported concentrations of selected PAHs in surface waters used as drinking water sources in four U.S. cities (Huntington, West Virginia; Buffalo, New York; and Pittsburgh and Philadelphia, Pennsylvania). Total concentrations of PAHs ranged from 4.7 ng/L in Buffalo to 600 ng/L in Pittsburgh. Mean concentrations of benzo[a]pyrene in the Great Lakes have been detected at levels between 0.03 and 0.7 ppt (ng/L) (Environment Canada 1991).

DeLeon et al. (1986) analyzed surface water from 11 locations in the Mississippi River. Seventeen PAHs were identified in the samples at levels ranging from 1 ng/L for 6 compounds to a high of 34 ng/L for phenanthrene. The highest concentration of phenanthrene was detected in a sample

collected near New Orleans, Louisiana, near an industrial area, implicating industrial effluent or surface runoff from this area as a possible source.

During April and May 1990, Hall et al. (1993) analyzed 48-hour composite samples from three locations in the Potomac River and three locations in the upper Chesapeake Bay for eight PAHs: perylene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benz[a]anthracene, and chrysene. Pyrene was the.only PAH found (0.42 µg/L) in these samples; it was detected in only one of nine Chesapeake Bay samples and not detected in any of the Potomac River samples (detection limit, 0.04 µg/L).

In a more recent study by Pham et al. (1993), raw water samples from 5 areas in the St. Lawrence River and its tributaries were analyzed for 12 PAHs. The highest mean total PAH concentrations were observed in samples collected in the spring (27.3 ng/L) and autumn (21.03 ng/L), which was attributed to snow melt and increased runoff during these respective seasons. The lowest mean total PAH concentration was observed in summer (14.63 ng/L). High molecular weight PAHs were detected more frequently in the spring and autumn samples. Phenanthrene, benzo[b]fluoranthene, fluoranthene, and pyrene were predominant, comprising on average 33.8%, 17.4%, 17.1%, and 12.8% of the total PAHs, respectively: With the exception of anthracene and benzo[b]fluoranthene, a general decrease in concentration with increasing molecular weight was observed.

PAHs have been detected in urban runoff generally at concentrations much higher than those reported for surface water. Data collected as part of the Nationwide Urban Runoff Program indicate concentrations of individual PAHs in the range of, 300-10,000 ng/L, with the concentrations of most PAHs above 1,000 ng/L (Cole et al. 1984). In a recent study by Pitt et al. (1993) which involved the collection and analysis of approximately 140 urban runoff samples from a number of different source areas in Birmingham, Alabama, and under various rain conditions, fluoranthene was one of two organic compounds detected most frequently (23% of samples). The highest frequencies of detection occurred in roof runoff, urban creeks, and combined sewer overflow samples. The maximum reported concentration of fluoranthene in these samples was 130 µg/L.

Industrial effluents also have elevated PAH levels. Morselli and Zappoli (1988) reported elevated PAH levels in refinery waste waters, with concentrations for most PAHs in the range of 400 ng/L (benzo[b]fluoranthene) to 16,000 ng/L (phenanthrene). In an analysis of STORET data covering the

period 1980-88, Staples et al. (1985) reported median concentrations in industrial effluents of <10 µg/L (10,000 ng/L) for 15 PAHs. The number of samples ranged from 1,182 (benzo-[blfluoranthene) to 1,288 (phenanthrene); the percentage of samples in which PAHs were detected ranged from 1.5 (benzo[g,h,i]perylene) to 7.0 (fluoranthene).

Few data are available on the concentrations of PAHs in U.S. groundwater. Basu and Saxena (1978b) reported total PAH concentrations in groundwater from three sites in Illinois, Indiana, and Ohio to be in the range of 3-20 ng/L. Groundwater levels of PAHs near a coal and oil gasification plant and U.S. wood treatment facilities have been found to be elevated. Groundwater samples from the site of a Seattle coal and oil gasification plant which ceased operation in 1956 were found to contain acenaphthylene, acenaphthene, fluorene, phenanthrene, fluoranthene, pyrene, and chrysene at concentrations ranging from not detected (detection limit 0.005 mg/L) to 0.25, 0.18, 0.14, 0.13, 0.05, 0.08, and 0.01 mg/L, respectively (Tumey and Goerlitz 1990). Individual PAHs in the groundwater from 5 U.S. wood treatment facilities were reported at average concentrations of 57 ppb (0.057 mg/L) for benzo[a]pyrene to 1,825 ppb (1.8 mg/L) for phenanthrene (Rosenfeld and Plumb 1991).

An evaluation of the analytical data from 358 hazardous waste sites with over 5,000 wells indicated that anthracene, fluoranthene, and naphthalene were detected (practical quantitation limit, 10-200 µg/L) in groundwater from at least 0.1% of the sites in three of the ten EPA Regions into which the United States is divided (Garman et al. 1987). A review of groundwater monitoring data from 479 waste disposal sites (178 CERCLA or Super-fund sites, 173 RCRA sites, and 128 sanitary/municipal landfill sites) located throughout the United States indicated that 14 of the PAHs included in this profile were detected at frequencies ranging from 2 detections at one site in one EPA Region for indeno[l,2,3-c,d]pyrene, to 85 detections at 16 sites in 4 EPA Regions for fluorene (Plumb 1991). Benzo[a]pyrene was detected 13 times at 6 sites in 6 EPA Regions. Concentrations were not reported.

Data summarized by Sorrel et al. (1980) indicate low levels of PAHs in finished drinking waters of the United States. Reported maximum concentrations for total PAHs (based on measurement of 15 PAHs) in the drinking water of 10 cities ranged from 4 to 24 ng/L; concentrations in untreated water ranged from 6 to 125 ng/L. The low concentrations of PAHs in finished drinking water were attributed to efficient water treatment processes. Shiraishi et al. (1985) found PAHs in tap water at concentrations of 0.1-1.0 ng/L, primarily as chlorinated derivatives of naphthalene, phenanthrene, fluorene, and fluoranthene. The significance to human health of these compounds is not known (Eisler 1987).

### 5.4.3 Sediment and Soil

PAHs are ubiquitous in soil. Because anthropogenic combustion processes are a major source of PAHs in soils, soil concentrations have tended to increase over the last 100-150 years, especially in urban areas (Jones et al. 1989a, 1989b). Background concentrations for rural, agricultural, and urban soils (from the United States and other countries) are given in Table 5-3. In general, concentrations ranked as follows: urban > agricultural > rural. Evidence of the global distribution of PAHs was given by Thomas (1986) who detected benzo[g,h,i]perylene and fluoranthene at concentrations above 150 µg/kg in arctic soils. Soil samples collected from remote wooded areas of Wyoming contained total PAH concentrations of up to 210 µg/kg.

Recent data on PAH concentrations in soil at contaminated sites are summarized in Table 5-4. Because of the different sampling methods and locations at each site, this tabulation does not provide a reliable inter-site comparison. Additional studies indicate significantly elevated concentrations of PAHs at contaminated sites. Soil samples collected from the Fountain Avenue Landfill in New York City contained PAH concentrations ranging from 400 to 10,000 µg/kg (Black et al. 1989). In a 1988 study at a hazardous waste land treatment site for refinery process wastes, which had been operative since 1958, average PAH concentrations in surface soils (0-30 cm) ranged from not detected (detection limits 0.1-2.0 mg/kg dry weight) for acenaphthylene, acenaphthene, anthracene, benz[a]anthracene, and benzo[k]fluoranthene to 340 mg/kg dry weight for dibenz[a,h]anthracene (Loehr et al. 1993). In addition to dibenz[a,h]anthracene, the three most prevalent compounds at this depth were benzo[a]pyrene (204 mg/kg), benzo[b]fluoranthene (130 mg/kg), and chrysene (100 mg/kg). PAH concentrations decreased with increasing depth and the majority of PAHs were not detected at depths below 60 cm. At 90-135 cm, only phenanthrene (1.4 mg/kg), pyrene (4.0 mg/kg), chrysene (0.9 mg/kg), and dibenz[a,h]anthracene (0.8 mg/kg) were found.

Sediments are major sinks for PAHs, primarily because of the low solubility of these compounds and their strong affinity for organic carbon in particulate matter. PAH concentrations in sediment are generally much higher than those detected in surface water, i.e., in the range of µg/kg (ppb) rather than ng/kg (ppt) .

In an assessment of STORET data covering the period 1980-1982, Staples et al. (1985) reported median concentrations in sediment of ≤500 µg/kg dry weight for 15 PAHs (acenaphthene,

## TABLE 5-3. Background Soil Concentrations of Polycyclic Aromatic Hydrocarbons (PAHs)

| Compound | Concentrations (μg/kg) | | |
|---|---|---|---|
| | Rural soil | Agricultural Soil | Urban Soil |
| Acenaphthene | 1.7 | 6 | |
| Acenaphthylene | | 5 | |
| Anthracene | | 11–13 | |
| Benzo(a)anthracene | 5–20 | 56–110 | 169–59,000 |
| Benzo(a)pyrene | 2–1,300 | 4.6–900 | 165–220 |
| Benzo(b)fluoranthene | 20–30 | 58–220 | 15,000–62,000 |
| Benzo(e)pyrene | | 53–130 | 60–14,000 |
| Benzo(g,h,i)perylene | 10–70 | 66 | 900–47,000 |
| Benzo(k)fluoranthene | 10–110 | 58–250 | 300–26,000 |
| Chrysene | 38.3 | 78–120 | 251–640 |
| Fluoranthene | 0.3–40 | 120–210 | 200–166,000 |
| Fluorene | | 9.7 | |
| Ideno(1,2,3-c,d)pyrene | 10–15 | 63–100 | 8,000–61,000 |
| Phenanthrene | 30.0 | 48–140 | |
| Pyrene | 1–19.7 | 99–150 | 145–147,000 |

[a]Derived from:

IARC 1973
White and Vanderslice 1980
Windsor and Hites 1979
Edwards 1983
Butler et al. 1984
Vogt et al. 1987
Jones et al. 1987

## TABLE 5-4. Soil Concentrations (mg/kg dry weight) Polycyclic Aromatic Hydrocarbons (PAHs) at Contaminated Sites[a]

| Compound | Wood-preserving[b] | | Creosote production[c] | | Wood treatment[d] | Coking plant[d] | Coking plant[e] | Gas works[f] | | Gas works[g] |
|---|---|---|---|---|---|---|---|---|---|---|
| | Surface-soil | Subsoil | mean | range | | | | mean | range | range |
| Acenaphthene | 7 | 1,368 | | | | | 29 | 2 | 0–11 | nd–3.0 |
| Acenaphthylene | 5 | 49 | 33 | 6–77 | | | 187 | | | nd–3.0 |
| Anthracene | 10 | 3,037 | 334 | 15–693 | 766 | 6 | 130 | 156 | 57–295 | nd–3.1 |
| Benz(*a*)anthracene | 12 | 171 | | | 356 | 16 | 200 | 317 | 155–397 | nd–8.6 |
| Benzo(*a*)pyrene | 28 | 82 | | | 94 | 14 | | 92 | 45–159 | nd–15 |
| Benzo(*e*)pyrene | | | | | | | | | | nd–12[i] |
| Benzo(*b*)fluoranthene | 38 | 140 | | | | | | 260 | 108–552 | nd–19 |
| Benzo(*k*)fluoranthene | | | | | | | | 238 | 152–446 | |
| Benzo(*j*)fluoranthene | | | | | | | | | | nd–1.2[i] |
| Benzo(*g,h,i*)perylene | | | | | | | | | | nd–16 |
| Chrysene | 38 | 481 | 614 | 8–1,586 | 321 | 11 | 135 | 345 | 183–597 | nd–12 |
| Dibenz(*a,h*)anthracene | | | | | 101 | 2 | | 2,451 | 950–3,836 | nd–2.0 |
| Fluoranthene | 35 | 1,629 | 682 | 21–1,464 | 1,350 | 34 | | 2,174 | 614–3,664 | nd–2.6 |
| Fluorene | 3 | 1,792 | 650 | 49–1,294 | 620 | 7 | 245 | 225 | 113–233 | nd–6.5 |
| Indeno(1,2,3-*c,d*)pyrene | 10 | 23 | | | | | | 207 | 121–316 | nd–13 |
| Naphthalene | 1 | 3,925 | 1,313 | <1–5,769 | 92 | 56 | 59 | | | nd[h]–46 |
| Phenanthrene | 11 | 4,434 | 1,595 | 76–3,402 | 1,440 | 27 | 277 | 379 | 150–716 | nd–26 |
| Pyrene | 49 | 1,016 | 642 | 19–1,303 | 983 | 28 | 285 | 491 | 170–833 | nd–4.3 |

[a]Modified from Wilson and Jones 1993
[b]Mueller et al. 1991—composite samples
[c]Ellis et al. 1991—samples are 1·5 m or 3·5 m
[d]Weissenfels et al. 1990a—no range or sampling details provided
[e]Werner et al. 1988—no range or sampling details provided
[f]Bewley et al. 1989—samples taken from prototype treatment bed
[g]Turney and Goerlitz 1990—samples taken in 1986 from plant inoperative since 1956.
[h]nd = not detected (no detection limit given)
[i]estimated value

acenaphthylene, anthracene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, benzo[a]pyrene, chrysene, fluoranthene, fluorene, indenopyrene, naphthalene, phenanthrene, and pyrene). The number of samples ranged from 236 (anthracene) to 360 (benzo-[alpyrene, fluoranthene); the percentage of samples in which these PAHs were detected ranged from 6.0 (acenaphthene, benzo[b]fluoranthene, benzo[k]fluoranthene, indeno[ 1,2,3-c,d]pyrene) to 22.0 (fluoranthene, pyrene).

Eadie et al. (1982) analyzed surficial sediments in southwestern Lake Erie near a large coal-fired power plant. Sediment concentrations for total PAHs were generally in the range of 530-700 µg/kg, although concentrations in river and near-shore sediments reached nearly 4,000 µg/kg (4 ppm). Heit et al. (1981) reported total concentrations of PAHs (3-7 ring PAHs) from two lakes in the Adirondack acid lake region of 2,660 µg/kg and 770 µg/kg (calculated from data presented). Average concentrations of total PAHs in sediments from three coastal South Carolina marinas were reported to range from 35.6 to 352.3 µg/kg (Marcus et al. 1988). Benzo[a]pyrene levels in bottom sediments of the Great Lakes have been reported to range from 34 to 490 ppb (µg/kg) (Environment Canada 1991). Concentrations of PAHs in sediments from Cape Cod and Buzzards Bay in Massachusetts and the Gulf of Maine have been reported to be in the range of 540-1,300 µg/kg (Hites et al. 1980). Concentrations of low molecular weight PAHs (naphthalene, acenaphthylene, fluorene, phenanthrene, anthracene, and 2-methylnaphthalene) and high molecular weight PAHs (fluoranthene, pyrene, benz[a]anthracene, chrysene, benzofluoranthenes, benzo[a]pyrene, indeno[l,2,3-c,d]pyrene, dibenz[a,h]anthracene, and benzo[g,h,i]perylene) in sediment from the highly polluted Boston Harbor have been reported to range from approximately 100 to 11,000 µg/kg dry wt, and 800 to 23,000 µg/kg dry wt, respectively (Demuth et al. 1993).

Total PAH concentrations in bottom sediments from the main stem of the Chesapeake Bay were reported to range from 45 to 8,920 µg/kg for samples collected from 16 stations in 1986 (Huggett et al. 1988). At least 14 PAHs were found to be dominant among pollutants of surface sediments from the Elizabeth River, a subestuary of the James River in Virginia, with a maximum total PAH concentration of 170,000 µg/g (ppm) observed in one sample from a site of two large wood preservative spills (Bieri et al. 1986). In a more recent study, surface sediment samples from the highly contaminated Elizabeth River were found to contain total concentrations of 14 PAHs ranging from 1.5 to 4,230 µg/g (ppm) dry weight (Halbrook et al. 1992). Significantly lower concentrations,

ranging from 0.34 to 0.95 µg/g (340-950 µg/kg) dry weight, were found in sediment samples from the nearby Nansemond River which served as a clean reference site.

Two-thirds of 105 sediment samples collected throughout Florida during the summers of 1989 and 1990 from sites known or suspected to be contaminated with priority pollutants were found to contain at least one of 15 PAH target analytes (Jacobs et al. 1993). Pyrene was detected most frequently (61% of samples); dibenz[a,h]anthracene and naphthalene were detected least frequently (4% of samples). Total PAH concentrations ranged from below the detection limit to 1,090 mg/kg. Mean concentrations for individual PAHs ranged from 0.87 mg/kg (dibenz[a,h]anthracene and naphthalene) to 30.8 mg/kg (acenaphthene).

Drainage stream sediments from a wood-preserving facility near Pensacola, Florida, were found to be highly contaminated with creosote-derived PAHs, with maximum concentrations from two sampling sites ranging from 300 µg/kg for naphthalene to 12,000 µg/kg for phenanthrene and 140,000 µg/kg for anthracene (Elder and Dresler 1988). Fluoranthene, pyrene, benz[a]anthracene, chrysene, acenaphthene, and fluorene were other dominant PAHs. PAHs were not detected in water samples from the drainage stream. Furthermore, no significant PAH contamination was found in surface sediments from estuarine sites adjacent to the drainage stream; PAHs were detected in sediment samples from only one of seven estuarine sites at concentrations ranging from 75 µg/kg for benz[a]anthracene to 190 µg/kg for fluoranthene.

In 1991, Kennicutt et al. (1994) found that sediment samples from Casco Bay in Maine contained total PAH concentrations ranging from 16 to 20,800 µg/kg dry weight. PAHs were found at all 65 locations sampled. PAHs with four or more rings accounted for more than 60% of Casco Bay sedimentary PAHs. The predominance of PAHs with highly condensed ring structures with few alkylations indicated a pyrogenic or combustion source as the major contributor.

Mean total PAH concentrations of sediments collected in 1985-87 from Moss Landing Harbor, Elkhom Slough, and nearshore Monterey Bay, California, were found to range from 1,470 to 3,080, 157 to 375, and 24 to 114 µg/kg dry weight, respectively (Rice et al. 1993). The Moss Landing Harbor and nearshore Monterey Bay ecosystems are subject to PAH contamination from various local industries, harbor-related activities, power generation, municipal waste treatment, and agricultural runoff. The largest Pacific Coast fossil-fueled power plant is located at Moss Landing. Elkhom

Slough is a seasonal estuary which receives freshwater runoff. Combustion PAHs (i.e., benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, benzo[a]pyrene, benzo[e]pyrene, chrysene, fluoranthene, and pyrene) were predominant, with mean total concentration at these 3 sites ranging from 1,250 to 2,710, to 335, and 11 to 59 ug/kg dry weight, respectively.

Median concentrations of PAHs in sediment coves collected in 1991 from three northern New Jersey waterways (Arthur Kill, Hackensack River, and Passaic River) highly contaminated with petroleum hydrocarbons ranged from 0.47 mg (470 ug/kg) (acenaphthylene) to 5.10 mg (5,100 µg/kg) (pyrene) (Huntley et al. 1993). In addition to pyrene, fluoranthene, chrysene, and benzo[a]pyrene were the most frequently detected PAHs, with median concentrations at the three sites ranging from 2.40 to 4.10, 1.35 to 2.85, and 0.86 to 2.30 mg/kg, respectively. Mean total PAH concentrations at the 29 sampling stations ranged from 0 to 161 mg/kg. A mean total PAH concentration of 139 mg/kg was found at a sampling station downstream from a chemical control Superftmd site. At most sampling stations, PAH concentrations increased with sample depth up to approximately 45-50 cm, indicating a decline in recent loadings relative to historic inputs.

### 5.4.4 Other Environmental Media

PAHs have been detected in many food products including cereal, potatoes, grain, flour, bread, vegetables, fruits, oils, and smoked or broiled meat and fish. The concentrations in uncooked foods largely depend on the source of the food. For example, vegetables and fruits obtained from a polluted environment may contain higher PAH concentrations than those obtained from nonpolluted environments. Benzo[a]pyrene, dibenz[a,h]anthracene, and chrysene have been detected in vegetables grown near a heavily traveled road (Wang and Meresz 1982). The method of cooking can also influence the PAH content of food; the time of cooking, the distance from the heat source, and the drainage of fat during cooking (e.g., cooking in a pan versus on a grill) all influence PAH content. For example, charcoal broiling increases the amounts of PAHs in meat. In a composite sample characterized to be typical of the U.S. diet, Howard (1979) found that PAH concentrations in all food groups were less than 2 ppb (µg/g). The following ranges of benzo[a]pyrene concentrations (wet or dry weight not specified) were summarized by Santodonato et al. (1981) from studies conducted in many countries:

- cooking oils: 0.5-S ppb (µg/g)
- margarine: 0.2-6.8 ppb

- smoked fish: trace-6.6 ppb
- smoked or broiled meats: trace-105 ppb
- grains and cereals: not detected-60 ppb
- fruits: not detected-29.7 ppb
- vegetables: not detected-24.3 ppb

These data include samples from areas identified as “polluted.”

Gomaa et al. (1993) recently reported the results of a study to screen smoked foods, including turkey, pork, chicken, beef, and fish products, for carcinogenic and noncarcinogenic PAHs. Eighteen commercially available liquid smoke seasonings and flavorings were also evaluated. All smoked meat products and liquid smoke seasonings were purchased from local supermarkets in Michigan. Total PAH concentrations in smoked red meat products ranged from 2.6 μg/kg in cooked ham to 29.8 μg/kg in.grilled pork chops, while those in smoked poultry products ranged from 2.8 μg/kg in smoked turkey breast to 22.4 μg/kg in barbecued chicken wings. Total PAH concentrations in smoked fish products ranged from 9.3 μg/kg in smoked shrimp to 86.6 μg/kg in smoked salmon. Total concentrations of carcinogenic PAHs (benz[a]anthracene, benzo[b]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, and indeno[l,2,3,-c,d]pyrene) ranged from not detected in several red meat products to 7.4 μg/kg in grilled pork chops; from not detected in several poultry products to 5.5 μg/kg in barbecued chicken wings; and from 0.2 μg/kg in smoked trout and shrimp to 14.9 and 16.1 μg/kg in smoked oysters and salmon, respectively. Total PAH concentrations in liquid smoke flavorings and seasonings ranged from 6.3 to 43.7 μg/kg, while total carcinogenic PAH concentrations ranged from 0.3 to 10.2 μg/kg. Smoked meat products processed with natural wood smoke had higher total PAH and total carcinogenic PAH concentrations than those processed with liquid smoke flavorings. Carcinogenic PAHs were not detected in 10% of the smoked food samples and 24% of the samples had concentrations of carcinogenic PAHs <I μg/kg. Benzo[a]pyrene was not detected in 31% of the samples; 45% of the samples had concentrations <l μg/kg. Benzo[a]pyrene was found at concentrations >l μg/kg in 24% of the samples, which included pork sausage (1.8-2.3 μg/kg), grilled pork chops (2.5 μg/kg), whole ham (1.1 μg/kg), beef sausage (1.1 μg/kg), salmon (3.9 μg/kg), and oysters (3.0 μg/kg). Benzo[a]pyrene was detected in 92% (12/13) of liquid smoke flavorings and seasoning samples, with concentrations ranging from 0.1 to 3.4 μg/kg.

Similar results have been obtained in recent investigations of benzo[a]pyrene concentrations in smoked foods in other countries. In Brazil, benzo[a]pyrene was detected in 52% (23/44) of smoked meat

samples; concentrations ranged from 0.1 to 5.9 µg/kg and were generally <1.0 µg/kg (Yabiku et al. 1993). In France, benzo[a]pyrene concentrations in smoked fish, poultry, and pork products were found to range from <0.2 to 1.9, 0.3 to 1.9, and <0.2 to 7.2 µg/kg, respectively; 36% (26/71) of the samples analyzed had benzo[a]pyrene concentrations >1 µg/kg (Moll et al. 1993). Because many imported food products are included in the U.S. food supply, these data may be relevant to estimating dietary PAH exposures of the general U.S. population.

In data summarized by Edwards (1983) the maximum total concentration of PAHs in vegetation near a source was 25,000 ppb (25 µg/g) (dry weight), while concentrations in nonsource areas ranged from 20 to 1,000 ppb (0.02-l .0 µg/g). In general, concentrations in leaves, stems, and fruits were higher than those in roots. Fluoranthene, pyrene, and chrysene/triphenylene were found in concentrations of 1.2, 2.0, and 2.9 µg/g, respectively, in composite samples of green leaves from 62 plant species in the Los Angeles area; corresponding values for dried leaf samples were 0.47, 1.l, and 1.9 µg/g (Rogge et al. 1993d). Edwards (1983) reported that washing removed a maximum of 25% of PAHs on the leaves of plants.

PAHs have been found in the tissues of aquatic organisms. In an assessment of STORET data covering the period 1980-1982, Staples et al. (1985) reported median concentrations in biota of <2.0 mg/kg (ppm) wet weight for 8 PAHs (acenaphthene, acenaphthylene, benz[a]anthracene, benzo[a]pyrene, chrysene, fluoranthene, fluorene, and pyrene) and <2.5 mg/kg wet weight for seven PAHs (anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, indenopyrene, naphthalene, and phenanthrene). The number of samples ranged from 83 (naphthalene) to 140 (acenaphthylene); only benzo[g,h,i]perylene (1 sample, 0.8%) and indenopyrene (1 sample, 0.8%) were found in detectable concentrations.

In summary of data on tissue contamination in mussels and oysters from the first 3 years (1986-1988) of the National Oceanic and Atmospheric Administration (NOAA) Mussel Watch Project, which involved the analysis of samples from 177 coastal and estuarine U.S. sites, overall mean concentrations of low molecular weight PAHs ranged from not detected (detection limits 3.3-67 ng/g dry weight) to 4,200 ng/g dry weight (NOAA 1989). Mean concentrations of low molecular weight PAHs for individual years 1986, 1987, and 1988 ranged up to 9,600, 3,200, and 4,300 ng/g dry weight, respectively. Overall mean concentrations of high molecular weight PAHs ranged from not detected (detection limits 3.9-47 ng/g dry weight) to 11,000 ng/g dry weight. Mean concentrations of high

molecular weight PAHs for 1986, 1987, and 1988 ranged up to 15,000, 10,000, and 11,000 ng/g dry weight, respectively. States with sites ranked among the highest five for concentrations of low molecular weight PAHs in 1986, 1987, 1988, and overall in 1986-1988, were California, Florida, Hawaii, Massachusetts, Mississippi, New York, Oregon, and Washington; for high molecular weight PAHs these states were California, Florida, Hawaii, Massachusetts, New York, and Washington. No consistent trends over the 1986-88 period were observed in the data; significant increases and decreases in concentrations of low and high molecular weight PAHs were observed with almost equal frequency. Low molecular weight PAH concentrations showed significant increasing trends at a single site each in New York, New Jersey, Florida, Texas, California, and Oregon; significant decreasing trends were observed at a single site each in Massachusetts, Maryland, and Mississippi, and at two sites in Texas. High molecular weight PAH concentrations showed significant increasing trends at a single site each in New York and Washington, and at two sites in Florida; significant decreasing trends were observed at a single site each in Connecticut, Maryland, and Florida.

Concentrations of phenanthrene and total PAHs ranged from 2 to 296 and 63 to 2,328 µg/kg (ng/g) wet weight, respectively, in caged mussels *(Elliptio complanata)* after 3 weeks' exposure at various locations in St. Mary's River, which is heavily contaminated from industrial and municipal discharges in the Sault Ste. Marie, Ontario, area (Kauss 1991). PAH concentrations ranging from approximately 50 ng/g wet weight for acenaphthylene to 4,660 nglg wet weight for fluoranthene were found in the digestive glands of the American lobster *(Homerus americancus)* collected in the proximity of a coal-coking plant that had been closed for a decade (King et al. 1993). Benzo[a]pyrene concentration was reported to be 720 nglg wet weight.

In a study to evaluate the concentrations of PAHs in various fish and shellfish species from Prince William Sound, Alaska, following the 1989 Exxon Valdez spill of more than 10 million gallons of crude oil, PAHs were not detected in 18% (72/402) of the samples; trace levels were found in 78% (312/402) of the samples; and individual PAH concentrations ranging from 5 to 12 µg/kg (wet or dry weight not specified) were found in 4% (18/402) of the samples. There was no apparent difference between PAH concentrations in salmon collected from impacted areas and those collected from control areas; however, there was a suggestion that contamination may be increasing with time. No PAHs were detected in 14% (31/221) of samples collected in 1989, trace levels were found in 85% of these samples, and only 1% (3 samples) had individual PAH concentration >5 µg/kg; whereas in the 1990

samples, PAHs were detected in all of the 41 samples, trace levels were found in 87% of the samples, and 13% (6 samples) had individual PAH concentrations >5 µg/kg.

PAHs are present at 1–2 weight percent in crude oils (Guerin 1978). Actual PAH concentrations in crude oil depend on the geological source of the oil (IARC 1989). For example, the NRC (1985) has reported concentrations of seven individual carcinogenic PAHs ranging from 1.2 µg/g for benzo[a]pyrene to 23 µg/g for chrysene in a South Louisiana crude oil and from 0.5 µg/g for benzo[e]pyrene to 6.9 µg/g for chrysene in a Kuwaiti crude oil. PAHs are also found in refined petroleum products including gasoline, kerosene, diesel fuel, some heating oils, and motor oil (Guerin 1978).

PAHs have also been detected in used motor oils. The following concentrations of benzo[a]pyrene and benz[a]anthracene measured in 1,071 samples of used motor oils were reported by Franklin Associates (1984):

| Positive | Samples(%) | Mean (mg/kg) | Median (mg/kg) | Range (mg/kg) |
|---|---|---|---|---|
| Benzo[a]pyrene | 58 | 24.5 | 10 | <1–405 |
| Benz[a]anthracene | 74 | 71.3 | 12 | <5–660 |

The levels, either in concentration or percent weight, in which several PAHs appear in various other substances are given below. A coal tar sample has been found to contain approximately 0.007 mg/kg benz[a]anthracene, 3 mg/kg benzo[b]fluoranthene, 4 g/kg chrysene, and 30 mg/kg benzo[a]pyrene (Perwak et al. 1982). High-temperature coal tar contains 1,000 mg/kg dibenz[a,h]anthracene (IARC 1985). A sample of coal tar pitch was found to contain <10 mg/kg benz[a]anthracene, <10 mg/kg chrysene, and approximately 10 mg/kg benzo[a]pyrene; creosote oil contains <3 mg/kg benz[a]anthracene, <1 mg/kg chrysene, and <10 mg/kg benzo[a]pyrene (Perwak et al. 1982). Creosote has been reported to contain 21% phenanthrene, 10% fluorene, 10% fluoranthene, 9% acenaphthene, 8.5% pyrene, 3% chrysene, 3% naphthalene, and 2% anthracene (Lorenz and Gjovik 1972).

PAHs have also been reported to occur in chewing tobacco, snuff, and in mainstream and sidestream tobacco smoke. Reported concentrations of some PAHs in various types of tobacco smoke are shown in Table 5-5 (IARC 1983). These data show concentrations of benzo[a]pyrene in cigarette mainstream smoke ranging between 5 and 78 ng/cigarette (IARC 1983). Other studies indicate that concentrations of carcinogenic PAHs in mainstream smoke from unfiltered cigarettes may range from 0.1 to 0.25 µg

per cigarette (Hoffmann and Hecht 1990). Concentrations of PAHs in sidestream smoke are significantly higher than in mainstream smoke with sidestreammainstream concentration ratios for benzo[a]pyrene ranging from 2.5 to 20 (Adams et al. 1987; Evans et al. 1993; Grimmer et al. 1987; Hoffmann and Hoffmann 1993; IARC 1983). Benzo[a]pyrene concentrations of 0.42-63 ppb (ng/g) have been reported in snuff (Brunnemann et al. 1986).

PAH concentrations in a variety of other media have been evaluated. PAH concentrations in fly ash and bottom ash samples from domestic municipal incinerators ranged from not detected to 7,400 µg/kg, with phenanthrene the most abundant and frequently detected compound (Shane et al. 1990). Machado et al. (1993) reported the total concentrations of 16 PAHs (all PAHs in this profile except benzo[e]pyrene) in asphalt and coal tar pitch to be 50, 122 and 294, 300 ppm µg/g), respectively; benzo[a]pyrene concentrations were <6 and 18,100 ppm, respectively. The concentrations of benzo[a]pyrene (250-480 ppm) and several other PAHs in coal tar fumes were higher than those in asphalt fumes by two to three orders of magnitude. The PAH content of asphalt and coal tar pitch fumes increased with increasing generation temperature.

Tire wear particles, brake lining particles, and paved road dust from a residential area had total PAH concentrations of 226.1, 16.2, and 58.7 µg/g, with maximum concentrations of individual PAHs of 54.1 µg/g (pyrene), 2.6 µg/g (benzo[g,h,i]perylene), and 9.4 µg/g (pyrene), respectively (Rogge et al. 1993c). Benzo[a]pyrene concentrations in these media were 3.9, 0.74, and 2.3 µg/g, respectively. Combined particle- and vapor-phase emissions from scrap tire fires have been reported to contain average total PAH concentrations of 3.2 mg/$m^3$, with average benzo[a]pyrene concentrations ranging from 0.07 to 0.08 mg/$m^3$ (Lemieux and Ryan 1993). Tire pyrolysis oil, which may be used as a fuel, contains high levels of PAHs, with average total PAH concentrations ranging from 14,540 ppm (µg/g) to over 100,000 ppm (10%); benzo[a]pyrene concentrations ranged from <10 to 600 ppm (Williams and Taylor 1993).

## 5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

The greatest sources of exposure to PAHs for most of the U.S. population are active or passive inhalation of the compounds in tobacco smoke, wood smoke, and contaminated air, and ingestion of these compounds in foodstuffs. Smoking one pack of cigarettes a day has been estimated to result in exposure to carcinogenic PAHs of up to 5 µg/day (Menzie et al. 1992) and in exposure to

## TABLE 5-6. Average Indoor Concentrations (μg/m$^3$) of Polycyclic Aromatic Hydrocarbons (PAHs) in Different Categories of Sample Homes Occupied by Smokers and Non-smokers[a]

| | E/E | | Home-type G/E | | G/G | | |
|---|---|---|---|---|---|---|---|
| Compound | Smokers | Non-smokers | Smokers | Non-smokers | Smokers | Non-smokers | Outdoor Air |
| Acenaphthylene | 18 | 11 | 71 | 15 | 33 | 17 | 4.2 |
| Anthracene | 3.5 | 1.8 | 8.3 | 3.4 | 8.9 | 2.2 | 0.96 |
| Benz[a]anthracene | 0.32 | 0.25 | 1.7 | 0.34 | 1.1 | 0.55 | 0.42 |
| Benzo[a]pyrene | 0.37 | 0.30 | 1.7 | 0.27 | 0.96 | 0.58 | 0.23 |
| Benzo[e]pyrene | 1.4 | 0.67 | 5.5 | 0.52 | 2.4 | 1.0 | 0.46 |
| Benzofluoranthenes | 0.79 | 0.68 | 1.1 | 0.97 | 2.0 | 2.0 | 1.1 |
| Benzo[g,h,i]perylene | 0.53 | 0.44 | 1.4 | 0.59 | 1.2 | 0.75 | 0.50 |
| Chrysene | 0.91 | 0.76 | 3.6 | 0.81 | 2.3 | 1.6 | 1.1 |
| Fluorothene | 7.2 | 7.7 | 13 | 7.5 | 13 | 16 | 5.6 |
| Indeno[1,2,3-c,d]pyrene | 0.35 | 0.28 | 1.1 | 0.40 | 0.84 | 0.64 | 0.35 |
| Phenanthrene | 79 | 57 | 130 | 63 | 130 | 110 | 31 |
| Pyrene | 4.3 | 4.6 | 11 | 4.8 | 5.0 | 9.3 | 4.4 |

[a]Adapted from Chuang et al. (1991)
[b]E/E denotes electric heating and cooking systems; G/E denotes gas heating and electric cooking systems; G/G denotes gas heating and cooking systems

benzo[a]pyrene of 0.4 µg/day (Santodonato et al. 1981). Other potential routes of human exposure are ingestion of contaminated drinking water and food products, and skin contact with soot and tars. PAHs are ubiquitous in the environment, resulting from the incomplete combustion of organic materials, whether natural (forest fires or volcanoes) or synthetic (combustion of fuels for heating and transportation). The amount of PAHs found in food products depends as much on the method of preparation (especially grilling or smoking) as on the origin of the food. Disinfection of public water supplies with chlorine can result in the presence of chlorinated and oxygen substituted PAHs (Shiraishi et al. 1985). Coal tar preparations have been used in the clinical treatment of skin disorders. Contamination of the ambient air can be derived from industrial and construction sources. Eldridge et al. (1983) measured substantial levels of PAHs emitted from freshly laid petroleum road asphalt.

Estimates of general population exposure to total PAHs (µg/day) and carcinogenic PAHs in comparison to readily measured benzo[a]pyrene concentrations were presented by Santodonato et al. (1981), as follows:

| Source | Benzo[a]pyrene | Select Carcinogenic PAHs[a] | Total PAHs |
|---|---|---|---|
| Air | 0.0095–0.0435 | 0.038 | 0.207 |
| Water | 0.0011 | 0.0042 | 0.027 |
| Food | 0.16–1.6 | – | 1.6–16 |

[a]Total of benzo[a]pyrene, benzo[j]fluoranthene, and indeno[1,2,3-c,d]pyrene.

The most noteworthy of these estimates are the relatively high exposures from ingestion of contaminate food; however, it was noted that because of the lack of reliable monitoring data for PAHs in food, the uncertainty of the food estimates was greater than the uncertainty of the estimates for air or water. Nevertheless, the authors concluded that estimates from all three exposure sources were probably accurate within one order of magnitude, so that food was predominant among the sources of exposure.

More recent estimates of the potential exposures of American adult males to carcinogenic PAHs were provided by Menzie et al. (1992). The estimates provided by these investigators do not include potential exposures experienced in occupational settings or those resulting from use of consumer products (e.g., cosmetics or asphaltic materials added to roofs or driveways). From the average American diet, the intake of carcinogenic PAHs was estimated to be 1–5 µg/day, mostly from

ingestion of unprocessed grains and cooked meats. This dietary intake estimate was increased to 6-9 µg/day for individuals consuming diets with a large meat content as a result of the additional contribution from charcoal-cooked or smoked meats and fish. Exposure via inhalation of ambient air was estimated to be 0.16 µg/day (median), with a range of 0.02-3 µg/day, assuming an inhalation rate of 20 $m^3$/day. Smoking one pack of unfiltered cigarettes per day increases this estimate by an additional 2-5 µg/day; chain smokers consuming three packs per day increase their exposure by an estimated 6-15 µg/day. Exposure to carcinogenic PAHs for the typical adult male from ingestion of drinking water and incidental ingestion of soil is minor compared to other potential routes of exposure. Drinking water exposure was estimated to be 0.006 µg/day (median), with a range of 0.0002-0.12 µg/day, assuming a consumption rate of 2 L/day. Assuming incidental ingestion of 50 mg soil/day, which may be more typical for small children than for most adults, the estimated median soil intake of carcinogenic PAHs was 0.06 µg/day (range, 0.003-0.3 µg/day). Therefore, the total potential exposure of carcinogenic PAHs for adult males was estimated to be 3 µg/day (median), with a maximum value of 15 µg/day. Smokers of nonfiltered cigarettes may experience exposures twice as high as these estimates. Ingestion of food appears to be the main source of exposure to PAHs for nonsmokers, although inhalation of ambient air is also an important route.

In a Dutch market-basket survey conducted from 1984 to 1986, the mean daily dietary intake of PAHs by 18-year-old males in composites of 221 different foods from 23 commodity groups was estimated to range from 5 to 17 µg/day. The most frequently detected PAHs were benzo[b]fluoranthene (59% of samples), fluoranthene (48%), and benzo[k]fluoranthene (46%). The largest contribution of PAHs to the total diet came from the sugar and sweets, cereal products, and oils, fats, and nuts commodity groups (de Vos et al. 1990).

Consumption of Great Lakes fish is not expected to contribute significantly to dietary intake unless the fish are smoked (Environment Canada 1991). The estimated exposure from consuming 114 g fish containing 50 ppt (ng/g) benzo[a]pyrene once a week would be 5.7 ng/person/week, or 11.6 pg/kg of body weight per day for a 70-kg individual. However, in some areas of the United States, fish consumption advisories have been issued based on elevated concentrations of PAHs found in locally caught fish or shell fish (see Section 5.6) (RTI 1993).

The average intake of benzo[g,h,i]perylene by adults from drinking water sources has been estimated to be 2 ng/day (assuming a drinking water ingestion rate of 2 L/day), inhalation exposure to the

compound has been estimated to be 10 ng/day (assuming an air intake rate of 20 $m^3$/day) (EPA 1989a).

Indoor air can be an important source of human exposure to PAHs. Potential indoor combustion sources of PAHs include tobacco smoke, unvented space heaters, and food preparation (Lioy and Greenberg et al. 1990). PAHs are among the major carcinogenic agents in environmental tobacco smoke (ETS), which is comprised primarily of diluted sidestream smoke, with a much smaller contribution from exhaled mainstream smoke (Hoffmann and Hoffmann 1993). Exposure to ETS is of particular concern because it has recently been declared a human lung carcinogen by the U.S. EPA. Concentrations of some PAHs in cigarette smoke-polluted environments are listed in Table 5-5. Chuang et al. (1991) monitored the concentrations of PAHs in the indoor and outdoor air of eight homes in Columbus, Ohio, in the winter of 1986-87 and obtained the indoor air results shown in Table 5-6.

Environmental tobacco smoke was the most significant influence on indoor air PAH levels; homes occupied by smokers had higher average concentrations of most PAHs than homes occupied by nonsmokers. In homes occupied by nonsmokers, the.highest average concentrations of most PAHs were found in homes that had gas cooking and heating appliances, followed by homes with gas heating and electric cooking appliances. Homes equipped with electric cooking and heating had the lowest average concentrations of most PAHs.

The Total Human Environmental Exposure Study (THEES), a multimedia study of human exposure to benzo[a]pyrene, was conducted in a rural town, Phillipsburg, New Jersey, where the major industry was a grey-iron pipe manufacturing plant that contributed to high levels of benzo[a]pyrene in the ambient atmosphere (Butler et al. 1993; Lioy 1990; Lioy et al. 1988; Waldeman et al. 1991). Benzo[a]pyrene concentrations in respirable particulate personal samples from 10 homes in areas near the foundry were measured in the range of 0.1 to 8.1 ng/$m^3$, depending on personal habits (whether the windows were kept open, how frequently the doors were opened, cooking methods, hobbies, whether home improvements were being made) and sources of home heating. The mean outdoor air concentration of benzo[a]pyrene was 0.9 ng/$m^3$. In samples of food collected from family meals over a 2-week period, the concentration level of benzo[a]pyrene ranged from 0.004 to 1.2 ng/g (wet weight)., No detectable amounts of benzo[a]pyrene were observed in the drinking water supply (detection limit, 0.1 ng/L). In comparing the inhalation and ingestion pathways in each home, Lioy et

## TABLE 5-5. Concentrations of Some Polycyclic Aromatic Hydrocarbons (PAHs) in Tobacco Smoke[a]

| Compound | Cigarette main stream smoke (µg/100 cigarettes) | Cigarette side stream smoke (µg/100 cigarettes) | Cigarette smoke-polluted environments (µg/m$^3$) | Cigar smoke (µg/100 g) | Pipe smoke (µg/100 g) |
|---|---|---|---|---|---|
| Anthracene | 2.3–23.5 | | | 11.9 | 110.0 |
| Benz(*a*)anthracene | 0.4–7.6 | 4–20 | 0.1–100 | 2.5–3.9 | |
| Benzo(*b*)fluoranthene | 0.4–2.2 | | 0.1–35[b] | | |
| Benzo(*j*)fluoranthene | 0.6–2.1 | | | | |
| Benzo(*k*)fluoranthene | 0.6–1.2 | | | | |
| Benzo(*g,h,i*)fluoranthene | 0.1–0.4 | | | | |
| Benzo(*g,h,i*)perylene | 0.3–3.9 | 9.8 | 0.4–17 | | |
| Benzo(*a*)pyrene | 0.5–7.8 | 2.5–19.9 | 0.4–760 | 1.8–5.1 | 8.5 |
| Benzo(*e*)pyrene | 0.2–2.5 | 13.5 | 0.4–18 | | |
| Chrysene | 0.6–9.6 | | 2.6–16 | | |
| Dibenz(*a,h*)anthracene | 0.4 | | <0.1–13 | | |
| Fluoranthene | 1–27.2 | 126 | 0.2–99 | 20.1 | |
| Fluorene | present | | | | |
| Indeno(1,2,3-*c,d*)pyrene | 0.4–2.0 | | 0.6–1 | | |
| Phenanthrene | 8.5–62.4 | | 4–87 | 115 | |
| Pyrene | 5–27 | 39–101 | 0.8–66 | 17.6 | 75.5 |

[a]Adapted from a tabulation of data from several studies in IARC (1983) and Guerin et al. (1992)
[b]Benzofluoranthenes

al. (1988) found that potential intake could be similar in each medium. Of the 20 weeks of exposure (10 homes over a 2-week exposure period), 10 had higher food benzo[a]pyrene exposures and 10 had higher inhalation benzo[a]pyrene exposures. The range of estimated food exposures (l0-4,005 ng/week) was much greater than the range of estimated air exposures (78-385 ng/week). The dominance of one pathway or the other seemed to depend on indoor combustion sources (e.g., cigarette smoke or coal-burning stoves) and personal eating habits. For smokers, inhalation of tobacco smoke was the main source of benzo[a]pyrene exposure; intake from this source was much higher than inhalation of ambient and indoor air or ingestion of food. Smokers also had higher exposure through food intake than nonsmokers. For the average nonsmoker, ingestion of food was the most important route of exposure (Lioy 1990).

Occupational exposures to PAHs can result from processes such as petroleum refining, metalworking, the production of coke, the manufacture of anodes, and the production of aluminum. In reviewing the available data on occupational exposures, it is important to understand that it cannot be implied that the results of a study at one industrial site would be valid for another site or for the same site at another time.

Occupational exposures to PAHs are possible in all operations involved in extraction and processing of crude oil, including drilling, pumping and treating, transport, storage, and refinement (Suess et al. 1985). The main route of exposure is inhalation, although there is also potential for significant dermal exposure (IARC 1989). Workers in petroleum refineries are exposed to PAHs from a variety of sources, including atmospheric distillation, catalytic cracking, residual fuel oil, lubricant oil processing, bitumen processing and loading, coking, and waste-water treatment (IARC 1989). In a study of nine US refineries, total PAH concentrations of 10 $\mu g/m^3$ were reported in personal samples taken in the fluid catalytic cracking and delayed coker units (Futagaki 1983). Total PAH concentrations ranging from approximately 1 to 40 $\mu g/m^3$ were observed in area samples from bitumen processing units. Total PAH concentrations in personal samples from the de-asphalting unit in one refinery were found to range from 2.5 to 49.8 $\mu g/m^3$. At least 85% of the total PAH in these samples was comprised of two-ring compounds (i.e., naphthalene and its derivatives) and 94% of two- or three-ring compounds. PAHs with five or more rings were found to contribute from <0.1% at the catalytic cracker unit to 2.5% at the delayed coker unit. The highest combined concentration of benzo[a]pyrene and benzo[e]pyrene was 9.3 $\mu g/g^3$ in a personal sample from a coker cutter; however, these two PAHs were not detected in most samples (detection limit, 0.01 μg/m3). Exposures to four- to six-ring PAHs

of <0.1 μg/m$^3$ have been associated with loading road tankers with bitumen in refineries (Brandt and Molyneux 1985). In an evaluation of turn-around operations on reaction and fractionator towers, concentrations of anthracene, benzo[a]pyrene, chrysene, and pyrene in personal samples were either too low to be detected or ≤1 μg/m$^3$; naphthalene and its methyl derivatives accounted for >99% of the total PAH measured in personal samples (Dynamac Corp. 1985). Area samples taken at various sites during shut-down, leak testing and start-up operations after turn-arounds showed the same distribution pattern of individual PAHs with total PAH concentrations generally ≤100 μg/m$^3$ (maximum 400 μg/m$^3$).

Metalworkers may also be exposed to PAHs from refined mineral oils used in machining operations, with the level of exposure depending on the type of oil refinement procedure used (IARC 1984). Acid refined mineral oils have a significant PAH content and have been shown to cause skin cancer in workers exposed to them (Jarvholm and Easton, 1990). Solvent refining procedures almost completely remove PAHs from mineral oils and, therefore, should almost completely eliminate the risks of exposure to carcinogenic PAHs (Bingham et al. 1965; Doak et al. 1983; IARC 1984). There is evidence, however, that the concentration of carcinogenic PAHs in solvent-refined cutting oils may increase during use, particularly during operations such as quenching where the oil is severely heated (Agarwal et al. 1986; Apostoli et al. 1993; IARC 1984; La Fontaine 1978; Thony et al. 1976). Total PAH concentrations in air samples from work areas related to the use of cutting, hardening, and extruding oilshave been reported to be 66, 90, and 106 ng/m$^3$, respectively (Apostoli et al. 1993).

In a summary of data on industrial exposures in IARC (1984), concentrations of airborne benzo[a]pyrene in aluminum production facilities from 1959 to 1982 in several countries ranged from not detected to 975 μg/m$^3$; concentrations in a U.S. aluminum reduction plant ranged from 0.03 to 53.0 μg/m$^3$, depending on the work site. Concentrations of airborne benzo[a]pyrene in coke oven operations ranged from 0 to 383 μg/m$^3$ depending on the work area; average concentrations in a U.S. plant were reported to range from 0.15 to 6.72 μg/m$^3$. More recently, inhalation exposures to phenanthrene, pyrene, and benzo[a]pyrene of 485, 108, and 48 μg/8 hours, respectively, have been reported for workers in a German coke plant (Grimmer et al. 1994). However, Van Rooij et al. (1993b) has recently concluded that among coke oven workers dermal absorption is a major route of exposure to PAHs, accounting for an average of 75% and 51% of total absorbed pyrene and benzo[a]pyrene, respectively; the mean dermal and respiratory uptakes of pyrene in 12 workers were reported to range from 4 to 34 and 0.5 to 32 μg/day, respectively.

The use of coal tar, pitch, asphalt, creosote, soot, and anthracene oil is widespread in the manufacture of fuel, dyes, plastics, paints, insulating materials, impregnating materials, building materials, road-building materials, embedding material, rubber, inks, and brushes (Hueper 1949). Faulds et al. (1981) faund that PAHs in diesel engine exhaust attach to respirable dust particles and travel long distances in underground mines, resulting in exposure of mine workers far removed from engine sites. Mechanics may also be exposed to PAHs resulting from the pyrolytic decomposition of the organic fractions of abraded particles from clutch and brake linings (Knecht et al. 1987). Potential exposure to PAHs in road sealing work involving coal tar and bitumen was discussed by Darby et al. (1986). In a study to evaluate inhalation and dermal exposures of 10 roofers removing an old coal tar pitch roof and applying a new asphalt roof, the PAH content of forehead skin wipes taken at the end of the workshift (0.097 μg/cm$^2$; equivalent to an estimated daily skin exposure of 19.4 μg/day) was found to correlate with the PAH concentrations in personal air samples (10.2 μg/m$^3$) (Wolff et al. 1989c). Relative concentrations of PAHs in air and wipe samples were: fluoranthene > pyrene > benz[a]anthracene > benzo[a]pyrene > benzo[b]fluoranthene > benzo[g,h,i]perylene > benzo[k]fluoranthene. Anthracene was found in the air samples but was not detected in the wipe samples.

Data on PAH exposures in the United States for many other occupations are limited. Most of the recent studies have been conducted in other countries. PAH concentrations (combined particle- and vapor-phase) in two work areas in a silicon carbide plant ranged from not detected (detection limit, 0.01 mg/m$^3$) for benzo[a]pyrene and benzo[e]pyrene, to 0.99 mg/m$^3$ for fluoranthene and 3.46 mg/m$^3$ for naphthalene (Dufresne et al. 1987). Higher ambient concentrations were observed in the furnace area, ranging from 0.04 mg/m$^3$ for benzo[a]pyrene and benzo[e]pyrene to 3.85 mg/m$^3$ for fluoranthene and 58.0 mg/m$^3$ for naphthalene. Total PAH concentrations in areas near cooking fume sources in the food and catering industries in Finland have been reported to range from 0.2 to 31.8 μg/m$^3$ (Vainiotalo and Matveinen 1993). Benzo[a]pyrene concentrations of <l-44 ng/m$^3$ in the breathing zone of urban bus drivers in France have been reported (Limasset et al. 1993). The mean particulate total PAH exposure level in a Swedish electrode paste plant was found to vary from 4.3 to 84.6 μg/m$^3$ over various work operations, with an overall mean particulate PAH exposure level in the plant of 14.4 μg/m$^3$ (Ovrebo et al. 1994). The mean PAH exposure level in a Belgian graphite electrode plant was reported to be 19.7 μg/m$^3$ (Van Hummelen et al. 1993). Drivers of large diesel-powered trucks in Switzerland were not found to have exposures to total PAHs or benzo[a]pyrene that were significantly different from controls (Guilleman et al. 1992).

Preliminary data from the National Occupational Exposure Survey (NOES), conducted by NIOSH from 1980 to 1983, estimated the number of workers potentially exposed to various chemicals in the workplace from 1981 to 1983 (NIOSH 1990). Data for the seven PAHs included in the survey are summarized below:

| Chemical | Number of industry/ occupation categories | Number of workers potentially exposed |
|---|---|---|
| Anthracene | 5 | 2,303 |
| Benz[a]anthracene | 4 | 2,310 |
| Benzo[a]pyrene | 1 | 896 |
| Chrysene | 2 | 9,358 |
| Fluoranthene | 36 | 21,339 |
| Fluorene | 6 | 2,912 |
| Pyrene | 3 | 9,368 |

The NOES database does not contain information on the frequency, level, or duration of exposure of workers to any of the chemicals listed. It provides only estimates of workers potentially exposed to the chemicals.

PAHs have generally not been detected in surveys of human tissue, presumably because the compounds are fairly rapidly metabolized. Phenanthrene was the only PAH detected in the 1982 National Human Adipose Tissue Survey; it was found in trace concentrations in 13% of the samples (EPA 1986). Acenaphthylene, acenaphthene, fluorene, and chrysene were not found at levels below the detection limit (0.010 µg/g; 10 ppt). However, autopsies performed on cancer-free corpses found PAH levels of 11–2,700 ppt (ng/g) in fat samples (Obana et al. 1981). Several PAHs were detected, including anthracene, pyrene, benzo[e]pyrene, benzo[k]fluoranthene, benzo[a]pyrene, and benzo[g,h,i]perylene, with pyrene being detected in the highest concentrations. A similar study done on livers from autopsied cancer-free corpses found levels of 6–500 ppt (ng/g) of all of the same PAHs except benzo[e]pyrene, which was not detected (Obana et al. 1981). As in the fat sample studies, pyrene appeared in the highest concentrations in the liver, but the overall levels were less than in fat.

Human exposure to PAHs can be monitored through analytical determination of PAHs and metabolites (e.g., 1-hydroxypyrene) in the urine of exposed individuals (Jongeneelen et al. 1985, 1987; Tolos et al. 1991; Weston et al. 1994). For example, Clonfero et al. (1990) detected increased levels of PAH metabolites in the urine of individuals occupationally (i.e., aluminum plant workers) and

therapeutically (i.e., psoriatic patients) exposed to coal tar, as compared to unexposed subjects. Weston et al. (1994) reported similar results for coal-tar treated psoriasis patients. Tolos et al. (1991) and Van Rooij et al. (1994) reported significant increases in the average 1-hydroxypyrene concentrations in the urine of smokers over nonsmokers. Significant increases in urinary 1-hydroxypyrene levels have also been observed in children living in areas of high density automobile traffic over those of children in suburban areas (Kanoh et al. 1993). Several researchers have reported substantial increases in the urinary concentrations of 1-Hydroxypyrene and other PAH metabolites among workers exposed to PAHs in a variety of occupational settings, including coke plants (Grimmer et al. 1994; Jongeneelen et al. 1990; Van Hummelen et al. 1993), graphite electrode plants (Van Hummelen et al. 1993), foundries (Santella et al. 1993), and creosote wood treatment plants (Viau et al. 1993) and during clean-up of dump sites contaminated with coal tars (Viau et al. 1993), handling of petroleum coke (Jongeneelen et al. 1989), and road surfacing operations (Jongeneelen et al. 1988). Most of these increases were statistically significant over controls. There is conflicting evidence regarding an exposure-response relationship between PAH exposures levels and urinary PAH metabolite concentrations. For example, Grimmer et al. (1994) reported a good correlation between PAH inhalation exposures and levels of urinary metabolites of benzo[a]pyrene, phenanthrene, and pyrene, whereas Jongeneelen et al. (1990) did not find a strong relationship between air monitoring data and urinary levels of 1-hydroxypyrene. In a study of PAH inhalation exposures of aluminum plant workers, Becher and Bjorseth (1983) found that the high concentrations in the occupational setting did not correspond to the measured concentrations of urinary PAH metabolites. The authors suggested that PAHs adsorbed to airborne particulate matter may not be bioavailable and that the exposure-uptake relationship may not be linear over the entire PAH concentration range. When urinary 1-hydroxypyrene excretion is used in the assessment of PAH exposure, the contributions of alternative routes of exposure (i.e., inhalation and dermal) and the variability in the baseline excretion among individual PAH metabolites due to tobacco smoking and dietary PAH intake should be taken into account (Van Rooij et al. 1993b, 1994). Assays for other biomarkers of PAH exposure are currently being developed in animal models (Singh and Weyard 1994), but have not been evaluated in humans.

The detection of PAH-DNA adducts in urine, blood and other tissues by immunoassay and $^{32}$P-postlabelling has also been used as an indicator of exposure (Harris et al. 1985; Herikstad et al. 1993; Ovrebo et al. 1994; Perera et al. 1993; Santella et al. 1993).

## 5.6 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Human exposure to PAHs is expected to be highest among certain occupational groups (e.g., individuals working with coal tar and its products, foundry workers, miners, chimney sweeps), smokers and nonsmokers living or working in close proximity to smokers, members of the general population who heat their homes with wood-burning stoves, individuals living in the vicinity of emission sources or using products containing PAHs, and people living in the vicinity of NPL sites where PAHs have been detected above background levels. People who consume grilled or smoked food may ingest high levels of these compounds. Anyone who works extensively with products such as roofing materials, asphalt, and other PAH-containing substances may be exposed through inhalation or skin contact.

Recreational and subsistence fishers that consume appreciably higher amounts of locally caught fish from contaminated waterbodies may be exposed to higher levels of PAHs associated with dietary intake (EPA 1993b). PAH contamination has triggered the issuance of several human health advisories. As of September 30, 1993, PAHs were identified as the causative pollutants in five fish consumption advisories in three different states. This information is summarized in Table 5-7 (RTI 1993). EPA is considering including PAHs as target analytes and has recommended that these chemicals be monitored in fish and shellfish tissue samples collected as part of state toxics monitoring programs. EPA recommends that residue data obtained from these monitoring programs be used by states to conduct risk assessments to determine the need for issuing fish and shellfish consumption advisories for the protection of the general public as well as recreational and subsistence fishers (EPA 1993b).

## 5.7 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of PAHs is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of PAHs.

## Table 5-7. Fish Consumption Advisories

| State | Waterbody | Extent |
|---|---|---|
| Massachusetts | Hocomoco Pond | Entire pond |
| Michigan | Hersey River | Downstream from Reed City |
| Ohio | Black River | 6.2 miles from the 31st Street Bridge (Loraine) to the harbor (includes confined disposed facility) |
| Ohio | Little Scioto River | 3.9 miles from Holland Road (Marion) south to St. Rt. 739 |
| Ohio | Mahoning River | 29.24 miles from Northwest Bridge Street (Warren) to Pennsylvania border |

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

### 5.7.1 Identification of Data Needs

**Physical and Chemical Properties.** The physical and chemical properties ($K_{ow}$, $K_{oc}$, vapor pressure, Henry's law constant, etc.) have been sufficiently characterized for most of the 17 PAHs and allow prediction of their environmental fate.

**Production, Import/Export, Use, Release, and Disposal.** According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI), which contains this information for 1992, .became available in May of 1994. This database will be updated yearly and should provide a list of industrial production facilities and emissions.

PAHs are produced primarily as a result of combustion processes both from anthropogenic and natural sources (HSDB 1994; IARC 1982). Of the 17 PAH compounds discussed in this profile, only acenaphthene, acenaphthylene, and anthracene are produced commercially. However, several other PAH compounds were imported into the United States in 1984 and 1985 (see Section 4.2).

There is no known commercial use for most of the 17 PAHs discussed in this profile. Anthracene, acenaphthene, fluorene, and phenanthrene are chemical intermediates used in the manufacture of dyes, plastics, pesticides, explosives, and chemotherapeutic agents (Hawley 1987; HSDB 1992; Windholz 1983). Fluoranthene is used as a lining material to protect the interior of steel and ductile iron drinking water pipes and storage tanks (NRC 1983).

PAHs are most likely to be released directly in the atmosphere. Other contaminated media of relevance to human exposure include foods and drinking water.

Rules governing the disposal of PAHs have been promulgated by EPA. Although information regarding recommended remedial techniques is available for most of the 17 PAHs discussed in this profile (EPA 1980, 1981a; HSDB 1992; IARC 1985), additional information about the amounts of these compounds disposed of by these remediation methods would be helpful in determining important routes of human exposure.

**Environmental Fate.** The environmental fate of PAHs is well characterized. No further studies are needed. PAHs are transported in and partitioned to the air, water, and soil. Transformation and degradation processes of PAHs in the air, water, and soil have been well studied. Atmospheric halflives of PAHs are generally less than 30 days. Photochemical oxidation of a number of PAHs has been reported (EPA 1988a). The National Research Council (NRC 1983) noted that compounds adsorbed to soot are more resistant to photochemical reactions than pure compounds. In surface water, PAHs can volatilize, photodegrade, oxidize, biodegrade, bind to particulates, or accumulate in aquatic organisms (with bioconcentration factors often in the 100-2,000 range). Half-lives for volatilization of benz[a]anthracene and benzo[a]pyrene (high molecular weight PAHs) from water have been estimated to be greater than 100 hours (Southworth 1979), and the half-life for volatilization of anthracene (a low molecular weight PAH) was estimated to be 18 hours (Southworth et al. 1978). Hydrolysis is not considered to be an important degradation process for PAHs (Radding et al. 1976). The rate and extent of photodegradation varies widely among the PAHs (Neff 1979). PAHs in soil can biodegrade or accumulate in plants. Microbial metabolism is the major process for degradation of PAHs in soil environments. Photolysis, hydrolysis, and chemical oxidation are not considered important processes for the degradation of PAHs in soils (Sims and Overcash 1983).

**Bioavailability from Environmental Media.** Limited information is available regarding the bioavailability of PAHs from plants grown in contaminated soils. PAHs can be absorbed following inhalation, oral, or dermal exposure. All of these routes are of concern to humans because PAHs have been shown to contaminate the air, drinking water, soil, and food. There is a need to conduct additional studies on the bioavailability of PAHs from plants grown in contaminated soils and from contaminated soils. However, bioavailability of PAHs from contaminated air, water, and food is of primary concern, and some information is available concerning bioavailability following exposure by these routes. Indirect evidence indicates that PAHs are absorbed by humans following inhalation exposure (Becher and Bjorseth 1983). Furthermore, indirect evidence suggests that benzo[a]pyrene may be absorbed following oral exposure in humans. The concentration of benzo[a]pyrene in human

feces was studied after eight volunteers ingested broiled meat that contained approximately 9 μg of benzo[a]pyrene (Hecht et al. 1979). Less than 0.1 μg/person of benzo[a]pyrene was measured in the feces of these individuals, suggesting absorption and perhaps metabolism of the compound. More direct data are needed on the extent of bioavailability of PAHs, particularly those that are particlebound, following the three major routes of exposure.

**Food Chain Bioaccumulation.** PAHs can bioaccumulate in plants, aquatic organisms, and animals from intake of contaminated water, soil, and food. Extensive metabolism of the compounds by high-trophic-level consumers, including humans, has been demonstrated; therefore, food chain biomagnification of the compounds does not appear to be significant (Edwards 1983; Eisler 1983; Gile et al. 1982; Wild et al. 1992). However, in some areas of the United States, fish consumption advisories have been issued based on elevated concentrations of PAHs found in locally caught fish or shellfish (see Section 5.6) (RTI 1993). Additional information is needed on levels of PAHs in aquatic organisms that are of concern for human health.

**Exposure Levels in Environmental Media.** PAHs have been produced and used in large volumes in the environment, home, and industry and. are widely distributed in the environment. They have been detected in air, water, sediment, soil, and food. Although some studies of background levels in different media have been conducted, additional site-specific concentration data in the vicinity of hazardous waste sites are needed. Studies should focus particularly on ambient air, in order to estimate exposure of the general population through inhalation of contaminated air as well as ingestion of or dermal contact with contaminated water or soil. Levels of PAHs tend to be higher in urban air than in rural air (Greenberg et al. 1985; Pucknat 1981). One study reported benzo[a]pyrene air levels of 0.2-19.3 ng/$m^3$ for urban air and 0.1-0.2 ng/$m^3$ in rural air (Pucknat 1981). Higher levels of other PAHs have been measured in urban areas. Basu and Saxena (1978a) reported concentrations of selected PAHs in surface waters used as drinking water sources in four U.S. cities (Huntington, West Virginia; Buffalo, New York; and Pittsburgh and Philadelphia, Pennsylvania) as ranging from 4.7 ng/L in Buffalo to 600 ng/L in Pittsburgh. Data collected as part of the Nationwide Urban Runoff Program indicate concentrations of individual PAHs in the range of 300-10,000 ng/L, with the concentrations of most PAHs above 1,000 ng/L (Cole et al. 1984). Few data are available on the concentrations of PAHs in U.S. groundwater. Basu and Saxena (1978b) reported total PAH concentrations in groundwater from three sites in Illinois, Indiana, and Ohio to be in the range of 3-20 ng/L. Data summarized by Sorrel et al. (1980) indicate low levels of PAHs in finished drinking waters of the

United States. Reported maximum concentrations for total PAHs (based on measurement of 1.5 PAHs) in the drinking water of 10 cities ranged from 4 to 24 ng/L. PAHs have been detected in unprocessed cereal, potatoes , grain, flour, bread, vegetables, fruits, and refined fats and oils. The concentrations in uncooked foods largely depend on the source of the food. The amount of PAHs found in food products depends as much on the method of preparation (especially grilling, smoking, or pickling) as on the origin of the food.

Reliable monitoring data for the levels of PAHs in contaminated media at hazardous waste sites and associated background sites are needed so that the information obtained on levels of PAHs in the environment can be used in combination with the known body burden of PAHs to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

**Exposure Levels in Humans.** No data are available regarding the levels of PAHs in body tissues or fluids for populations living near hazardous waste sites. PAHs and their metabolites can be measured in the urine of exposed individuals. In workers exposed to PAHs, the PAH metabolite 1-hydroxypyrene has been detected in the urine at concentrations of 0-40 µg/g creatinine (Jongeneelen et al. 1985). No correlation was found between occupational exposure levels and urine levels, so it is not known whether-urine metabolites could be detected following exposure to low levels of PAHs (as might be expected to occur in individuals living in the vicinity of hazardous waste sites).

PAHs have generally not been detected in surveys of human tissue, presumably because the compounds are fairly rapidly metabolized. Phenanthrene was the only PAH detected in the 1982 National Human Adipose Tissue Survey; it was found in trace concentrations in 13% of the samples (EPA 1986). Acenaphthylene, acenaphthene, fluorene, and chrysene were not found at levels below the detection limit (0.010 µg/g; 10 ppt). However, autopsies performed on cancer-free corpses found PAH levels of 11-2,700 ppt (ng/g) in fat samples (Obana et al. 1981). Several PAHs were detected, including anthracene, pyrene, benzo[e]pyrene, benzo[k]fluoranthene, benzo[a]pyrene, and benzo[g,h,i]perylene, with pyrene being detected in the highest concentrations. A similar study done on livers from cancer-free patients found levels of 6-500 ppt of all of the same PAHs except benzo[e]pyrene, which was not detected (Obana et al. 1981). As in the fat sample studies, pyrene appeared in the highest concentrations in the liver, but the overall levels were less than in fat.

A few exposure estimates for the general population have been made from inhalation of ambient air and ingestion of contaminated drinking water and food, but sparse monitoring data limit the reliability of these estimates. Relatively recent estimates of the size of the workforce exposed to a few of the PAHs (such as benzo[a]pyrene) are available from NIOSH. However, monitoring data on workplace exposure levels are generally inadequate, partially because of the complexity of air emissions in terms of number of compounds detected. Information on exposure levels in humans is needed to better define exposure estimates in the general population and workforce, and to examine the relationship between levels of PAHs in the environment, human tissue levels, and the subsequent development of health effects. These data should be collected simultaneously with data on levels of PAHs air, water, and soil. For a sound database to serve as a foundation for higher level environmental or toxicological research, it should contain information on human exposure levels to PAHs, particularly for individuals living near hazardous waste sites.

This information is necessary for assessing the need to conduct health studies on these populations.

**Exposure Registries.** No exposure registries for PAHs were located. These substances are not currently compounds for which a subregistry has been established in the National Exposure Registry. The substances will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to this substance.

### 5.7.2 Ongoing Studies

The National Institute of Environmental Health Sciences is funding research at Miami University, Oxford, Ohio, to evaluate the transfer of benzo[a]pyrene from sediments directly to a sediment-feeding fish. Specific objectives are to (1) to examine the effects of seasonal parameters (i.e., temperature, body lipids, gonadal development) on the rate and pattern of metabolism of benzo[a]pyrene in a range of size, age, and sexual maturity classes; and (2) evaluate the relative importance of sediment and water as vectors of uptake of benzo[a]pyrene.

The National Institute of Environmental Health Sciences is funding research at the State University of New York at Albany to determine the aquatic bioavailability of PAHs from sediments collected near

discharge points from two aluminum manufacturing plants in the Massena area of the St. Lawrence River. The results of this study should provide a better understanding of the relative contributions of individual industries to pollution of fish and wildlife consumed by area residents.

The National Institute of Environmental Health Sciences is funding research at the State University of New York at Albany to conduct an epidemiologic study of Mohawk women and infants to test the hypotheses that exposure to PAHs, polychlorinated biphenyls (PCBs), polychlorinated dibenzo-pdioxins and dibenzofurans (PCDD/Fs) from a nearby Superfund hazardous waste site elevates body burdens and affects the cytochrome P-450-dependent mixed function monooxygenase system. Determination of 15 PAHs in breast milk will be among the analyses included in this study. The results of the study should enhance our understanding of how these important classes of chemicals arising from hazardous waste bioaccumulate in human adults and infants and whether P-450IA2 induction is a sensitive biomarker of their early biologic effect.

The National Institute of Environmental Health Sciences is funding research at Johns Hopkins University to investigate the molecular dosimetry of ingested PAHs from cooked meats in humans, and identify susceptibility factors that modulate the formation of DNA and protein adducts with these dietary carcinogens. Ultimately, molecular biomonitoring may allow quantitation of biological dose from ingested PAHs, thus accounting for variation in exposure, cooking processes, and metabolism.

The National Institute of Environmental Health Sciences is funding the Massachusetts Institute of Technology to continue studies with fluoranthene by using a modification of a $^{32}$P-postlabelling assay to detect and quantify DNA adducts in mice and human lymphoblast cell lines from eventual application as a dosimeter to monitor DNA damage in tissues from occupationally or environmentally exposed human populations.