Polycyclic Aromatic Hydrocarbons:
15 Listings
Reasonably anticipated to be human carcinogens
Also known as PAHs or polynuclear aromatic hydrocarbons

The term “polycyclic aromatic hydrocarbon” (PAH) commonly refers to a large class of organic compounds that contain carbon and hydrogen and consist of two or more fused aromatic rings. Fifteen individual PAHs (not the entire class) are listed separately in the Report on Carcinogens as reasonably anticipated to be a human carcinogen:

- Benz[a]anthracene, benzo[b]fluoranthene, benzo[j]-fluoranthene, benzo[a]pyrene, dibenz[a,h]acridine, dibenz[a,j]acridine, dibenz[a,h]anthracene, 7H-dibenzo[c,g]-carbazole, dibenzo[a,h]pyrene, dibenzo[a,i]pyrene, and indeno[1,2,3-cd]pyrene were first listed in the Second Annual Report on Carcinogens (1981).
- Benzo[k]fluoranthene, dibenzo[a,e]pyrene, dibenzo[a,l]pyrene, and 5-methylchrysene were first listed in the Fifth Annual Report on Carcinogens (1989).

The chemical structures of the 15 listed PAHs are shown below. Evidence for their carcinogenicity from studies in experimental animals is then discussed separately for each PAH. However, most of the information on mechanisms of carcinogenesis, cancer studies in humans, use, production, exposure, and regulations is common to all 15 listed PAHs and therefore is discussed for the overall class of PAHs, following the discussions of cancer studies in experimental animals.

Benz[a]anthracene
CAS No. 56-55-3
Also known as BA

Benzo[b]fluoranthene
CAS No. 205-99-2
Also known as B[b]F

Benzo[j]fluoranthene
CAS No. 205-82-3
Also known as B[j]F

Benzo[k]fluoranthene
CAS No. 207-08-9
Also known as B[k]F

Benzo[a]pyrene
CAS No. 50-32-8
Also known as B[a]P

Dibenz[a,h]acridine
CAS No. 226-36-8
Also known as DB[a,h]AC

Dibenz[a,j]acridine
CAS No. 224-42-0
Also known as DB[a,j]AC

Dibenz[a,h]anthracene
CAS No. 53-70-3
Also known as DB[a,h]A
7H-Dibenzo[c,g]carbazole  
CAS No. 194-59-2  
Also known as 7H-DB[c,g]C

Dibenzo[a,e]pyrene  
CAS No. 192-65-4  
Also known as DB[a,e]P

Dibenzo[a,h]pyrene  
CAS No. 189-64-0  
Also known as DB[a,h]P

Dibenzo[a,i]pyrene  
CAS No. 189-55-9  
Also known as DB[a,i]P

Dibenzo[a,l]pyrene  
CAS No. 191-30-0  
Also known as DB[a,l]P or dibenzo[def,p]chrysene

Indeno[1,2,3-cd]pyrene  
CAS No. 193-39-5  
Also known as IP

5-Methylchrysene  
CAS No. 3697-24-3  
Also known as 5-MC

Carcinogenicity  
The 15 individual PAHs are 
reasonably anticipated to be human carcinogens based on sufficient evidence of carcinogenicity from studies in experimental animals.

Cancer Studies in Experimental Animals

Benz[a]anthracene  
Benz[a]anthracene caused tumors in mice at several different tissue sites and by several different routes of exposure. Benz[a]anthracene administered by stomach tube to adult mice or by subcutaneous injection to newborn mice caused benign or malignant lung tumors (adenoma or adenocarcinoma). Administration by stomach tube also caused liver cancer (hepatocellular carcinoma) in adult mice. Benz[a]anthracene caused tumors in mice at the site of administration: skin tumors were observed after application to the skin, cancer at the injection site (sarcoma) after subcutaneous injection, and urinary-bladder cancer (carcinoma) after implantation in the bladder (IARC 1973).

Since benz[a]anthracene was listed in the Second Annual Report on Carcinogens, additional studies in mice have been identified. In newborn mice, intraperitoneal injection of benz[a]anthracene caused benign lung tumors (adenoma) in both sexes and benign or malignant liver tumors (adenoma or carcinoma) in males (Levin et al. 1984, Wislocki et al. 1986, Von Tungeln et al. 1999).

Benzo[b]fluoranthene  
Benzo[j]fluoranthene

Dermal exposure to benzo[j]fluoranthene caused benign or malignant skin tumors (papilloma or carcinoma) in female mice (IARC 1973). Since benzo[j]fluoranthene was listed in the Second Annual Report on Carcinogens, additional studies in rodents have been identified. Intraperitoneal injection of benzo[j]fluoranthene caused lung cancer (squamous-cell carcinoma) in female rats (IARC 1983). In newborn mice, intraperitoneal injection of benzo[j]fluoranthene caused benign and malignant lung tumors (alveolar/bronchiolar adenoma and carcinoma) in both sexes and benign or malignant liver tumors (hepatocellular adenoma or carcinoma) in males (Lavoie et al. 1987, 1994).

Benzo[k]fluoranthene

Benzo[k]fluoranthene caused tumors in two rodent species, at two different tissue sites, and by two different routes of exposure. Intraperitoneal injection of benzo[k]fluoranthene caused lung cancer (squamous-cell carcinoma) in female rats, and subcutaneous injection of benzo[k]fluoranthene caused cancer at the injection site (sarcoma) in mice of both sexes (IARC 1983).

Benzo[a]pyrene

Benzo[a]pyrene caused tumors in eight species, including nonhuman primates, at several different tissue sites, and by several different routes of exposure. Benzo[a]pyrene had both local and systemic carcinogenic effects and caused tumors after a single dose, after prenatal exposure, and in newborn mice. Benzo[a]pyrene caused lung tumors (1) in mice after dietary exposure, prenatal exposure, or subcutaneous or intravenous injection, (2) in rats after administration in the trachea or the bronchus, and (3) in hamsters and nonhuman primates after intratracheal instillation (Andervont and Shimkin 1940, IARC 1973). Oral administration (in the diet or drinking water or by stomach tube) also caused forestomach and esophageal tumors in mice and hamsters, intestinal tumors in hamsters, and mammary-gland tumors in female rats (Horie et al. 1965, IARC 1973). Mammary-gland tumors in rats were also observed after intravenous injection. Benzo[a]pyrene caused skin tumors in prenatally exposed mice and in dermally exposed mice, rats, and rabbits. Cancer at the injection site (sarcoma or fibrosarcoma) was observed in mice, rats, hamsters, guinea pigs, newts, monkeys, and nonhuman primates exposed by subcutaneous injection and in mice exposed by intraperitoneal injection (IARC 1973).

Since benzo[a]pyrene was listed in the Second Annual Report on Carcinogens, numerous additional studies in experimental animals have been identified. These studies reported that benzo[a]pyrene caused tumors (1) by additional routes of exposure (including inhalation and other types of injections), (2) in additional species of experimental animals (including fish), and (3) at several additional tissue sites. In studies published since the early 1980s, benzo[a]pyrene caused tumors at the following tissue sites:

- The upper respiratory system (mainly the nose and larynx) and upper digestive system (mainly the pharynx, but also the forestomach and esophagus) in male hamsters exposed by inhalation (Thyszen et al. 1981).
- The tongue and larynx (papilloma or carcinoma) in female mice following dietary exposure (Culp et al. 1998, Goldstein et al. 1998).
- The anus in mice of both sexes exposed by intracolonic injection (Toth 1980).
- The cervix in female mice exposed by intravaginal injection (Näslund et al. 1987).

Other studies (not described here) confirmed the earlier findings or found that benzo[a]pyrene caused tumors at similar tissue sites in additional species or by additional routes of exposure. Lung tumors were observed following exposure by (1) intratracheal or intrabronchial instillation in female mice (Kim and Lee 1996) and in rabbits of both sexes (Hirao et al. 1980), (2) intracolonic injection in female mice (Anderson et al. 1983), (3) intrafetal administration in mice of both sexes (Rossi et al. 1983), and (4) intraperitoneal injection in rats (Deutsch-Wenzel 1983, Wenzel-Hartung 1990). Intratracheal injection of benzo[a]pyrene in mice also caused tumors at tissue sites where it had previously been shown to cause tumors by other routes of exposure: the forestomach, esophagus, mammary gland, and skin (Toth 1980, Anderson et al. 1983). Benzo[a]pyrene caused forestomach tumors in mice exposed by intraperitoneal injection (Weyand et al. 1995), mammary-gland tumors in rats exposed by intramammary injection (Cavaliere et al. 1988, 1991), and sarcoma in mice exposed by intraperitoneal injection. Benzo[a]pyrene implanted in the buccal cavity caused intestinal tumors in rats (Solt et al. 1987), and a single intraperitoneal injection of benzo[a]pyrene caused abdominal tumors (mesothelioma and sarcoma) in rats (Roller et al. 1992).

Dibenzo[a,j]acridine

Dibenzo[a,j]acridine caused tumors in mice at several different tissue sites and by several different routes of exposure. Subcutaneous or intravenous injection of dibenzo[a,j]acridine caused lung tumors; subcutaneous injection also caused cancer at the injection site (sarcoma), and dermal exposure caused skin tumors (IARC 1973). Since dibenzo[a,j]acridine was listed in the Second Annual Report on Carcinogens, one study in rats has been identified. Intraperitoneal implantation of pellets containing dibenzo[a,j]acridine caused lung cancer (carcinoma) in female rats (Deutsch-Wenzel 1983).

Dibenzo[a]acridine

Dibenzo[a]acridine caused tumors in mice at several different tissue sites and by two different routes of exposure. Dermal exposure to dibenzo[a]acridine in mice caused benign or malignant skin tumors (papilloma, carcinoma, or epithelioma). Subcutaneous injection of dibenzo[a]acridine caused cancer at the injection site (sarcoma) in all mouse strains tested and lung tumors in strain A mice (a strain with a high spontaneous incidence of lung cancer) (IARC 1973).

Dibenzo[a,h]anthracene

Dibenzo[a,h]anthracene caused tumors in several species of experimental animals, at several different tissue sites, and by several different routes of administration. Dibenzo[a,h]anthracene caused lung tumors in mice after a single intravenous or subcutaneous injection (IARC 1973), in newborn mice after intraperitoneal injection (Buening et al. 1979), and in hamsters after intratracheal instillation (Pott et al. 1978, as cited in IARC 2010). In mice, oral exposure to dibenzo[a,h]anthracene caused cancer of the lung (adenomatosis or alveologenic carcinoma) and mammary gland (carcinoma), benign or malignant tumors of the forestomach (squamous-cell papilloma or carcinoma), and tumors of the blood vessels (hemangioendothe-
Additional studies in mice have been identified. Intraperitoneal injection of newborn mice with dibenzo[a,h]pyrene caused lung tumors in both sexes and liver tumors in males (Chang et al. 1982). In female rats, intramammary injection of dibenzo[a,h]pyrene caused cancer of the mammary gland (fibrosarcoma or adenocarcinoma) (Cavaliere et al. 1989), and subcutaneous injection caused cancer at the injection site (sarcoma) (Bahna et al. 1979).

**Dibenzo[a,l]pyrene**

Dibenzo[a,l]pyrene caused tumors in two rodent species, at two different tissue sites, and by several different routes of administration. Dermal exposure to dibenzo[a,h]pyrene caused benign or malignant skin tumors (papilloma or epithelioma) in mice, and subcutaneous injection caused cancer at the injection site (sarcoma) in mice and hamsters (IARC 1973).

Since dibenzo[a,l]pyrene was listed in the Second Annual Report on Carcinogens, additional studies in rodents have been identified. Intraperitoneal injection of newborn mice with dibenzo[a,l]pyrene caused lung tumors in both sexes and liver tumors in males (Chang et al. 1982), and intratracheal instillation caused respiratory-system cancer (mostly squamous-cell carcinoma, but also adenocarcinoma and anaplastic carcinoma) in hamsters of both sexes (Sellakumar and Shubik 1974, Stenbäck and Sellakumar 1974). Dibenzo[a,l]pyrene administered by intramammary injection caused cancer of the mammary gland (fibrosarcoma and adenocarcinoma) in female rats (Cavaliere et al. 1989).

**Dibenzo[a,e]pyrene**

Dibenzo[a,e]pyrene caused tumors in mice at two different tissue sites and by two different routes of exposure. Subcutaneous injection of dibenzo[a,e]pyrene caused cancer at the injection site (sarcoma) in mice of both sexes (IARC 1973), and dermal exposure caused skin tumors in female mice (IARC 1983).

Since dibenzo[a,e]pyrene was listed in the Fifth Annual Report on Carcinogens, additional studies in experimental animals have been identified, which reported that dibenzo[a,e]pyrene caused tumors (1) by additional routes of exposure (oral, prenatal, and intraperitoneal injection), (2) in additional species of experimental animals (rats, hamsters, and fish), and (3) at additional tissue sites, including sites distant from the route of administration. Administration of dibenzo[a,e]pyrene by stomach tube to female mice caused ovarian tumors (predominately granulosa) (Buters et al. 2002). Dietary administration of dibenzo[a,e]pyrene to fish caused benign or malignant liver tumors (hepatocellular adenoma or carcinoma or cholangiocellular adenoma) and benign tumors of the stomach (papillary adenoma) and swim bladder (papillary adenoma) (Reddy et al. 1999a,b). Intraperitoneal injection of dibenzo[a,e]pyrene caused lung tumors in strain A/J mice (Prahalad et al. 1997). Lung and liver tumors were observed in prenatally exposed mice (Yu et al. 2006) and in newborn mice exposed by intraperitoneal injection (Platt et al. 2004); lung tumors occurred in both sexes, and liver tumors in males. In addition, prenatal exposure to dibenzo[a,e]pyrene caused T-cell lymphoblastic lymphoma in mice of both sexes (Yu et al. 2006). Local tumors also were observed in rats and hamsters: intramammary injection of dibenzo[a,e]pyrene caused mammary-gland cancer (adenocarcinoma or fibrosarcoma) in female rats (Cavaliere et al. 1989, 1991), and application of dibenzo[a,e]pyrene directly to the tongue
caused cancer of the oral cavity (squamous-cell carcinoma) in female hamsters (Schwartz et al. 2004).

**Indeno[1,2,3-cd]pyrene**

Indeno[1,2,3-cd]pyrene caused tumors in mice at two different tissue sites and by two different routes of exposure. Dermal exposure to indeno[1,2,3-cd]pyrene caused benign and malignant skin tumors (papilloma and carcinoma) in females, and subcutaneous injection caused cancer at the injection site (sarcoma) in males (IARC 1973). Since indeno[1,2,3-cd]pyrene was listed in the Second Annual Report on Carcinogens, an additional study in rodents has been identified. Intraperitoneal administration of indeno[1,2,3-cd]pyrene caused lung cancer (carcinoma) in female rats (Deutsch-Wenzel 1983).

**5-Methylchrysene**

5-Methylchrysene caused tumors in mice at two different tissue sites and by two different routes of exposure. Dermal exposure to 5-methylchrysene caused skin cancer (carcinoma) in females, and subcutaneous injection caused cancer at the injection site (sarcoma) in males (IARC 1983). Since 5-methylchrysene was listed in the Fifth Annual Report on Carcinogens, additional studies in mice have been identified. Intraperitoneal injection of 5-methylchrysene caused lung tumors in male strain A mice (Ross et al. 1995, Nesnow et al. 1998) and lung and liver tumors in newborn mice of both sexes (Hecht et al. 1985, el-Bayomy et al. 1989).

**Studies on Mechanisms of Carcinogenesis**

Most PAHs with potential biological activity range in size from two to six fused aromatic rings (IARC 2010). Because of the vast range in molecular weight of PAHs, several of the physicochemical properties that are critical to their biological activity also vary greatly. Five properties in particular have a decisive influence on the biological activity of PAHs: their vapor pressure, their adsorption on surfaces of solid carrier particles, their absorption into liquid carriers, their lipoidal aqueous partition coefficient in tissues, and their limits of solubility in the lipid and aqueous phases of tissues. These properties are intimately linked with the metabolic activation of the most toxic PAHs, and an understanding of the nature of this interaction helps in the understanding of their deposition and disposition. It has been proposed that PAHs share a similar mechanism of carcinogenic action. In general, PAHs are converted to oxides and dihydrodiols, which in turn are oxidized to diol epoxides. Both oxides and diol epoxides are ultimate DNA-reactive metabolites. PAH oxides can form stable DNA adducts, and diol epoxides can form stable and depurinating adducts with DNA through formation of electrophilic carbonium ions. Most of the 15 listed PAHs have been shown to be initiators of skin cancer (IARC 1983, 2010). The International Agency for Research on Cancer concluded that benzo[a]pyrene was carcinogenic to humans based on data on the mechanism of carcinogenicity (IARC 2010).

**Cancer Studies in Humans**

No epidemiological studies on exposure to the individual PAHs were identified. Individual PAHs are found in the environment not in isolation but as components of highly complex mixtures of chemicals. PAHs are very widespread environmental contaminants, because they are formed during incomplete combustion of materials such as coal, oil, gas, wood, or garbage or during pyrolysis of other organic material, such as tobacco or charbroiled meat. Data on the carcinogenicity of PAHs in humans are available only for mixtures containing PAHs. It is difficult to ascertain the carcinogenicity of the component PAHs in these mixtures because of potential chemical interactions and the presence of other carcinogenic substances in the mixtures.

In 2005, IARC reevaluated PAHs. Although certain occupations with high PAH exposure (e.g., coal gasification and coke production) were classified as carcinogenic in humans, the roles of individual PAHs could not be defined (IARC 2010).

**Properties**

Three of the listed PAHs (dibenzo[a,h]acridine, dibenzo[a,j]acridine, and 7H-dibenzo[c,g]carbazole) contain a nitrogen atom as part of a ring and therefore are classified as heterocyclic PAHs. The PAHs can exist as leaflets, plates, needles, or at room temperature and range in color from colorless to yellow, green or blue. All PAHs are soluble in water and slightly soluble in ethanol, acetone or acid; most are soluble in benzene. Physical and chemical properties of the 15 PAHs are listed in the table below. In addition to the properties listed in the table, benzo[a]pyrene has a specific gravity of 1.351 and a vapor density relative to air of 8.7, and dibenzo[a,j]anthracene has a specific gravity of 1.282 (HSDB 2009).

**Use**

IARC (1983) reported that no commercial uses or applications were known for dibenzo[a,h]pyrene, dibenzo[a,j]pyrene, and 5-methylchrysene. The remaining twelve listed PAHs are used only in biochemical, biomedical, laboratory, or cancer research (HSDB 2009). At least five of the listed PAHs are present in coal tar, which is used as a fuel in the steel industry in open-hearth and blast furnaces (HSDB 2009). Coal tar is also used in the clinical treatment of skin disorders such as eczema, dermatitis, and psoriasis. Coal tar is distilled to produce a variety of products, including coal-tar pitch and creosote. At least two of the listed PAHs are present in coal-tar pitch, which is used primarily as a binder for aluminum smelting electrodes in the aluminum reduction process. Coal-tar pitch is also used in roofing, in surface coatings, for pitch-coke production, and for a variety of other applications (IARC 1985). At least two of the listed PAHs are found in creosote, which is used to preserve railroad ties, marine pilings, and telephone poles. Some creosote is used for fuel by steel producers (NIOSH 1977). At least three of the listed PAHs are present in bitumens and asphalt, which are used for paving roads, sound- and water-proofing, and coating pipes.

**Production**

PAHs are not produced for commercial use in the United States (IARC 1983, HSDB 2009). Production data for tar, pitch, creosote, mineral oils, and coke, which contain various PAHs, are included in their respective profiles in the Report on Carcinogens (see Coal Tars and Coal-Tar Pitches, Coke-Oven Emissions, and Mineral Oils: Untreated and Mildly Treated).

**Exposure**

PAHs are ubiquitous in the environment, and the general population is exposed to measurable background levels (IPCS 1998). Sources of PAHs in ambient air (both outdoors and indoors) include forest fires, volcanoes, industrial emissions, residential and commercial heating with wood, coal, or other biomass fuels (oil and gas heating produce much lower quantities of PAHs), motor-vehicle exhaust (especially diesel), and other indoor sources such as cooking and tobacco smoke (IARC 1983, IPCS 1998). Food is a major source of exposure to PAHs for the general population (IPCS 1998). Skin contact with PAH-contaminated soils and the use of dermatally applied pharmaceutical products based on coal tar also have been identified as sources of exposure for the general population (Jongeneelen et al. 1985, Viau and Vyskocil 1995, IPCS 1998, Jongeneelen 2001).
According to the U.S. Environmental Protection Agency’s Toxics Release Inventory, industrial releases of PAHs to the environment peaked in 2000, when over 4.9 million pounds was released, mostly to on-site and off-site landfills and to air. Releases have been relatively stable at a lower level since 2002. In 2008, 1,192 facilities released over 1.2 million pounds of PAHs to air, water, or on- or off-site landfills (mostly to air or landfills) (TRI 2010).

In the past, benzo[a]pyrene often was used as a marker for measuring exposure to PAHs. However, it is now possible or even common to measure many PAHs individually. Mean concentrations of individual PAHs in ambient urban air usually range from 1 to 30 ng/m³ (IPCS 1998). The concentrations of PAHs in the air during winter, when residential heating is a major source, generally are at least an order of magnitude higher than those in summer (IPCS 1998). Areas near sources such as motor-vehicle traffic also have higher air concentrations of PAHs. For individuals who smoke, mainstream tobacco smoke is a major source of exposure to PAHs. Concentrations of total PAHs in mainstream smoke ranged from 1 to 1.6 μg/cigarette. Sidestream smoke is a major source of PAHs in indoor air. Concentrations of benzo[a]pyrene in sidestream smoke ranged from 52 to 95 ng/cigarette—more than three times the concentration in mainstream smoke.

PAHs in water may originate from surface runoff (e.g., from the erosion of asphalt pavement or from air deposition of smaller particles) (IPCS 1998). Industrial effluents also can contribute to PAH concentrations in surface waters. However, concentrations of PAHs in water usually are very low, because of their low solubility. Surface-water concentrations typically do not exceed 50 ng/L; higher concentrations are found in more contaminated areas. PAH concentrations are higher in rainwater than in surface waters (100 to 200 ng/L, with some samples exceeding 1,000 ng/L). Because PAHs have very high octanol-water partition coefficients (log \( K_{ow} \)), they bind tightly to soil particles and are relatively immobile in soil; therefore, concentrations in groundwater and drinking water typically are very low (0.02 to 1.8 ng/L), and concentrations of PAHs in sediments may be very high, ranging up to several thousand micrograms per kilogram.

Estimates of daily PAH intake from food vary widely, ranging from a few nanograms to a few micrograms per person. Sources of PAHs in the diet include barbecued, grilled, broiled, and smoke-cured meats; roasted, baked, and fried foods (prepared by high-temperature processing); breads, cereals, and grains (at least in part from gas or flame drying of grains); and vegetables grown in contaminated soil or with surface contamination from atmospheric deposition of PAHs (IARC 1983, IPCS 1998, JECFA 2005). The Joint United Nations Food and Agriculture Organization—World Health Organization Expert Committee on Food Additives and Contaminants determined a representative mean daily human intake of benzo[a]pyrene to be 4 ng/kg of body weight and a high-end daily human intake of total PAHs to be 10 ng/kg (JECFA 2005). Among common foods, the highest PAH levels were found in grilled or barbecued steak, chicken with skin and bones, and hamburgers, especially when “well done” or “very well done” (Larsson et al. 1983, Lijinsky 1991, Lodovici et al. 1995, Kazerouni et al. 2001). Because PAHs form on or near the surface of meat, rather than in the interior, foods that are cooked to the same degree without being exposed to flames do not show significant levels of PAHs. However, a study of PAHs in the Italian diet indicated a total PAH concentration of about 4 ng/g in fried beef (Lodovici et al. 1995) and benzo[a]pyrene concentrations of up to about 4 ng/g in well-done grilled meat. PAHs are also introduced by certain methods of preserving meat and other food products (Lijinsky 1991). In foods smoked in traditional smoking kilns, the average concentration was 1.2 μg/kg for benzo[a]pyrene and 9 μg/kg for total PAHs, compared with 0.1 μg/kg for benzo[a]pyrene and 4.5 μg/kg for total PAHs in foods treated in a modern kiln (Guillen 1994).

Accumulation of PAHs in foods of animal origin, especially livestock, is due mainly to the consumption of contaminated feed (Ramesh et al. 2004). Unprocessed foods such as vegetables, fruits, vegetable oils, dairy products, and seafood can be contaminated with PAHs by deposition of particles and vapors from the atmosphere and uptake from soil, water, and sediment (Roth et al. 1998, Ramesh et al. 2004). PAH levels are low in cereals and beans, but drying techniques used for preservation, such as combustion gas heating and smoking, increase concentrations of PAHs in these foods. Eggs and dairy products such as cheese, milk, and butter contain low levels of PAHs. Consumption of seafood, especially bottom-feeding shellfish and finfish, may contribute considerably to the amount of PAHs in the diet. Species near the top of the food chain, such as humans, do not bioaccumulate PAHs, because of their higher capacity to metabolize PAHs (Ramesh et al. 2004).

A specific urinary metabolite of pyrene, 1-hydroxypyrene, has been suggested as a biomarker of human exposure to PAHs (Jongeneelen et al. 1985, Jongeneelen 2001). In representative samples from the general population, 1-hydroxypyrene has been detected in the urine of nearly all individuals, at median concentrations typically less than 0.1 μmol/mol of creatinine (Huang et al. 2004). The National Health and Nutrition Examination Survey analysis of 2,312 urine samples collected from the U.S. general population in 1999 to 2000 found a geometric mean concentration of 1-hydroxypyrene of 0.039 μmol/mol of creatinine (95% CI = 0.034 to 0.046 μmol/mol). The level for adult smokers was three times that for nonsmokers (geometric mean = 0.080 vs. 0.025 μmol/mol). These data are comparable with other recent data on non-occupationally exposed populations in Europe and Canada.

Occupational exposure to PAHs is primarily through inhalation and dermal contact. Industrial processes that involve the pyrolysis or combustion of coal and the production and use of coal-derived products, including coal tar and coal-tar-derived products, are major sources of occupational exposure to PAHs. Workers in coal-tar production plants, coking plants, bitumen and asphalt production plants, gasification sites, smoke houses, aluminum-production plants, coal-tarring facilities, and municipal trash incinerators are exposed to PAHs. Exposure may also result from inhaling engine exhaust and using products that contain PAHs in a variety of other industries, such as mining, oil refining, metalworking, chemical production, transportation, and the electrical industry (Vanrooij et al. 1992). Studies in Germany measured concentrations of PAHs in the breathing zone of chimney sweeps during so-called “black work”; the PAHs in the air samples varied depending on the type of fuel burned (oil, oil/solid, or solid) (Knecht et al. 1989). Specific occupational exposure to coal tar, coal-tar pitch, creosote, mineral oils, and coke that contain various PAHs is described in the profiles for these substances (see Coal Tar and Coal-Tar Pitches, Coke Oven Emissions, and Mineral Oils). Concentrations of PAHs in coal-tar products may range from less than 1% to 70% or more (ATSDR 2002). Occupational exposure can lead to PAH body burdens among exposed workers that are considerably higher than those in the general population. There is growing awareness that uptake of PAHs through the skin is substantial (Jongeneelen 2001). Dermal uptake has been shown to contribute to the internal exposure of workers to PAHs; a study in the creosote industry found that the total internal dose of PAHs did not necessarily correlate with inhalation-exposure levels alone, and that dermal exposure contributed significantly (Vanrooij et al. 1992).
Regulations
Environmental Protection Agency (EPA)
Clean Air Act
Mobile Source Air Toxics: Polycyclic organic matter is listed as a mobile source air toxic for which regulations are to be developed.
National Emissions Standards for Hazardous Air Pollutants: Polycyclic organic matter is listed as a hazardous air pollutant.
Urban Air Toxics Strategy: Polycyclic organic matter is identified as one of 33 hazardous air pollutants that present the greatest threat to public health in urban areas.

Clean Water Act
Effluent Guidelines: Polynuclear aromatic hydrocarbons are listed as toxic pollutants.
Water Quality Criteria: Based on fish or shellfish and water consumption = 0.0038 μg/L for benz[a]-anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenzo[a,h]-anthracene, and indeno[1,2,3-cd]-pyrene; based on fish or shellfish consumption only = 0.018 μg/L for benzo[a]anthracene, benzo[a]pyrene, benzo[k]fluoranthene, benzo[a]-anthracene, dibenzo[a,h]-fluoranthene, dibenzo[a]anthracene, and indeno[1,2,3-cd]-pyrene.
Comprehensive Environmental Response, Compensation, and Liability Act
Maximum contaminant level = 0.0002 mg/L for benzo[a]pyrene.

Food and Drug Administration (FDA)
Maximum permissible level in bottled water = 0.0002 mg/L for benzo[a]pyrene. Limit on PAH levels in various color additives are prescribed in 21 CFR 74 and 178.

Guidelines
American Conference of Governmental Industrial Hygienists (ACGIH)
Threshold limit value – time-weighted average (TLV-TWA) = exposure by all routes should be as low as possible for benzo[a]anthracene, benzo[a]pyrene, and benzo[b]fluoranthene.

References

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