Polycyclic Aromatic Hydrocarbons

TXP-2

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Note: We also have an abridged version of this article!

Overview

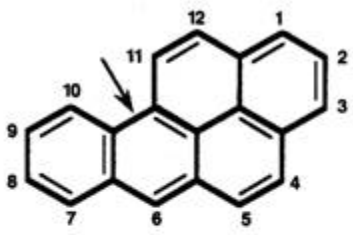
The term polycyclic aromatic hydrocarbons (PAHs) refers to a ubiquitous group of several hundred chemically-related, environmentally persistent organic compounds of various structures and varied toxicity. Most of them are formed by a process of thermal decomposition (pyrolysis) and subsequent recombination (pyrosynthesis) of organic molecules. PAHs enter the environment through various routes and are usually found as a mixture containing two or more of these compounds, e.g. soot. However, some PAHs are manufactured and these pure PAHs usually exist as colorless, white, or pale yellow solids. Polycyclic aromatic hydrocarbons affect organisms through various toxic actions. The mechanism of toxicity is considered to be interference with function of cellular membranes as well as with enzyme systems which are associated with the membrane. They have been shown to cause carcinogenic and mutagenic effects and are potent immunosuppressants. Effects have been documented on immune system development, humoral immunity and on host resistence. The most extensively studied PAHs are 7,12-dimethylbenzo anthracene (DMBA) and benzo(a)pyrene (BaP).

Chemical Characteristics

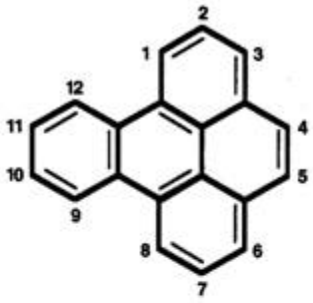
Polycyclic aromatic hydrocarbons have two or more single or fused aromatic rings with a pair of carbon atoms shared between rings in their molecules. The term "PAH" refers to compounds consisting of only carbon and hydrogen atoms. PAHs containing up to six fused aromatic rings are often known as "small" PAHs, and those containing more than six aromatic rings are called "large" PAHs. The majority of research on PAHs has been conducted on small PAHs due to the availability of samples of various small PAHs.

The general characteristics of PAHs are high melting and boiling points (therefore they are solid), low vapor pressure, and very low aqueous solubility, which both tend to decrease with increasing molecular weight, whereas resistance to oxidation and reduction increases. PAHs are highly lipophilic and therefore very soluble in organic solvents. PAHs also manifest various functions such as light sensitivity, heat resistance, conductivity, emittability, corrosion resistance, and physiological action.

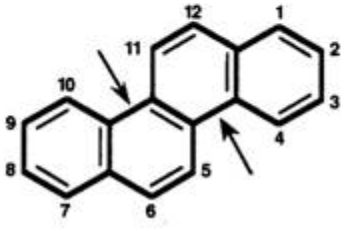
PAHs possess very characteristic UV absorbance spectra. Each ring structure has a unique UV spectrum, thus each isomer has a different UV absorbance spectrum. This is especially useful in the identification of PAHs. Most PAHs are also fluorescent, emitting characteristic wavelengths of light when they are excited (when the molecules absorb light). Aqueous solubility decreases for each additional ring.



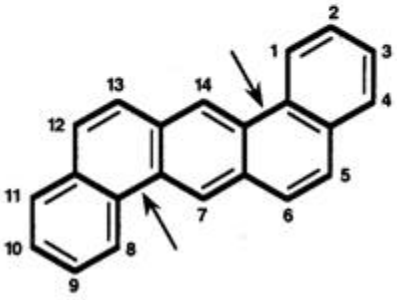
Benzo[*a*]pyrene



Benzo[*e*]pyrene



Chrysene



Dibenz[*a*,*h*]anthracene

The simplest PAHs, as defined by the International Union of Pure and Applied Chemistry (IUPAC), are phenanthrene and anthracene, which both contain three fused aromatic rings. Smaller molecules, such as benzene, are not PAHs. Naphthalene, which consists of two coplanar six-membered rings sharing an edge, is another aromatic hydrocarbon. By formal convention, it is not a true PAH, though is referred to as a bicyclic aromatic hydrocarbon.

Although the health effects of individual PAHs are not exactly alike, these 17 PAHs have been identified as being of greatest concern with regard to potential exposure and adverse health effects on humans and are thus considered as a group (profile issued by the Agency for Toxic Substances and Disease Registry):

acenaphthene
acenaphthylene
anthracene
benz(a)anthracene
benzo(a)pyrene
benzo(e)pyrene
benzo(b)fluoranthene
benzo(ghi)perylene
benzo(j)fluoranthene
benzo(k)fluoranthene
chrysene
dibenz(ah)anthracene
fluoranthene
fluorene
indeno(1,2,3-cd)pyrene
phenanthrene
pyrene

(Image of selected PAHs from ATSDR. The arrows indicate bay regions.)

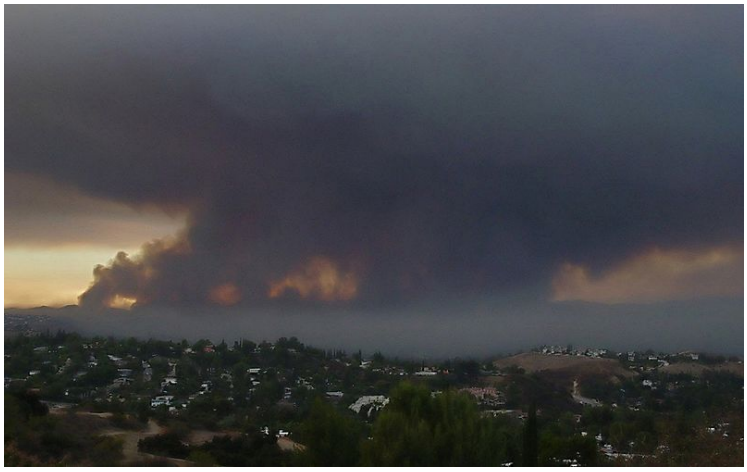
Sources

Sources of PAHs can be both natural and anthropogenic.
Natural sources include:

- forest and grass fires
- oil seeps
- volcanoes
- chlorophyllous plants, fungi, and bacteria

Anthropogenic sources of PAHs include:

- petroleum
- electric power generation
- refuse incineration
- home heating
- production of coke, carbon black, coal tar, and asphalt
- internal combustion engines



Uses

PAHs are not synthesized chemically for industrial purposes. Rather than industrial sources, the major source of PAHs is the incomplete combustion of organic material such as coal, oil and wood. However, there are a few commercial uses for many PAHs. They are mostly used as intermediaries in pharmaceuticals, agricultural products, photographic products, thermosetting plastics, lubricating materials, and other chemical industries. General uses are:

- Acenaphthene: manufacture of dyes, plastics, pigments, pharmaceuticals and pesticides
- Anthracene: manufacture of dyes and pigments;diluent for wood preservatives;
- Fluoranthene: manufacture of dyes, pharmaceuticals and agrochemicals.
- Fluorene: manufacture of dyes, pigments, pesticides, thermoset plastic and pharmaceuticals;
- Phenanthrene: manufacture of pesticides and resins
- Pyrene: manufacture of pigments

Other PAHs may be contained in asphalt used for the construction of roads, as well as roofing tar.
Precise PAHs, specific refined products, are used also in the field of electronics, functional plastics, and liquid crystals.

Routes of Exposure

The major route of exposure to PAHs in the general population is from breathing ambient and indoor air, eating food containing PAHs, smoking cigarettes, or breathing smoke from open fireplaces. Tobacco smoke contains a variety of PAHs, such as benzo(a)pyrene, and more than 40 known or suspected human carcinogens. For non-smokers the main route of exposure is through food. PAH concentrations in foodstuffs vary. Charring meat or barbecuing food over a charcoal, wood, or other type of fire greatly increases the concentration of PAHs. Some crops, such as

wheat, rye, and lentils, may synthesize PAHs or absorb them via water, air, or soil. Water can also contain substantional amounts of PAHs since those chemicals can leach from soil into water or thex can enter water from industrial effluents and accidental spills during oil shipment at sea. Soil also contains PAHs, primarily from airborne fallout.

Therefore, PAH exposure occurs on a regular basis for most people. Occupational exposure may also occur in workers breathing exhaust fumes, such as mechanics, street vendors, motor vehicle drivers, as well as those involved in mining, metal working, or oil refining. Routes of exposure include ingestion, inhalation, and dermal contact in both occupational and non-occupational settings. Some exposures may involve more than one route simultaneously, affecting the total absorbed dose (such as dermal and inhalation exposures from contaminated air).

Metabolism

Since exposure to PAHs is never to single PAHs, understanding what differences may occur in mixtures of PAHs gives an accurate assessment of the dangers of PAHs. Understanding the dynamics of complex metabolism vis-a-vis single metabolism of PAHs and possible effects on the toxicity expression of PAHs is a necessary advancement to accurately impact and guide remediation strategies.

Studies were carried out comparing the metabolism of the PAHs Phenanthrene (PHE), Flouranthene (FLA) and Benzo(a)pyrene (BAP) in single, binary, and ternary mixtures by monitoring the disappearance of the parent compound. It was observed that PAH metabolism in the single PAH experiment differed from metabolism in both binary and ternary mixtures. Enzyme competition was evident in the metabolism of mixtures, changing significantly the metabolism patterns of individual PAHs. PAH structure was also seen to affect metabolism in mixtures and the possible creation of toxicity effects during mixture metabolism. PAH concentration changed over time with faster change during single PAH metabolism followed by ternary mixture metabolism and finally binary metabolism. These results affirm that substrate interactions must be considered in the risk assessment approaches to the dangers posed by exposure to PAHs.

Due to the high lipophilicity of this class of compounds, their bioavailability after ingestion and inhalation is significant. Scientific investigations have shown that detectable levels of PAH occur in almost all internal organs, particularly in organs that are rich in adipose tissue. These organs can serve as storage depots from which the hydrocarbons can be gradually released. Once they enter the organism polycyclic aromatic hydrocarbons require a multistep metabolic activation by specific enzymes. The enzyme system primarily responsible for PAH metabolism is the mixed-function oxidase system. The first reaction is an epoxidation. PAH epoxides can then be conjugated with glutathione and this is regarded as a true detoxification reaction. The epoxides that are not conjugated with glutathione are converted into phenols and diols. These PAH metabolites, however, are sometimes not sufficiently polar to be excreted and are therefore have to be conjugated with glucuronic or sulfuric acids to enable excretion. Most metabolites of PAH are excreted in feces and urine.

Human Health Effects

Acute or Short-term Health Effects

The effects on human health will depend mainly on the length and route of exposure, the amount or concentration of PAHs one is exposed to, and of course the innate toxicity of the PAHs. A variety of other factors can also affect health impacts including subjective factors such as pre-existing health status and age. The ability of PAHs to induce short-term health effects in humans is not clear. Occupational exposures to high levels of pollutant mixtures containing PAHs has resulted in symptoms such as eye irritation, nausea, vomiting, diarrhoea and confusion. However, it is not known which components of the mixture were responsible for these effects and other compounds commonly found with PAHs may be the cause of these symptoms. Mixtures of PAHs are also known to cause skin irritation and inflammation. Anthracene, benzo(a)pyrene and naphthalene are direct skin irritants while anthracene and benzo(a)pyrene are reported to be skin sensitizers, i.e. cause an allergic skin response in animals and humans (IPCS, 1998).

Chronic or Long-term Health Effects

Health effects from chronic or long-term exposure to PAHs may include decreased immune function, cataracts, kidney and liver damage (e.g. jaundice), breathing problems, asthma-like symptoms, and lung function abnormalities, and repeated contact with skin may induce redness and skin inflammation. Naphthalene, a specific PAH, can cause the breakdown of red blood cells if inhaled or ingested in large amounts. If exposed to PAHs, the harmful effects that may occur largely depend on the way people are exposed.

Carcinogenicity

Although unmetabolized PAHs can have toxic effects, a major concern is the ability of the reactive metabolites, such as epoxides and dihydrodiols, of some PAHs to bind to cellular proteins and DNA. The resulting biochemical disruptions and cell damage lead to mutations, developmental malformations, tumors, and cancer. Evidence indicates that mixtures of PAHs are carcinogenic to humans. The evidence comes primarily from occupational studies of workers exposed to mixtures containing PAHs and these long-term studies have shown an increased risk of predominantly skin and lung, but as well as bladder and gastrointestinal cancers. However, it is not clear from these studies whether exposure to PAHs was the main cause as workers were simulataneoulsly exposed to other cancer-causing agents (e.g. aromatic amines).

Animals exposed to levels of some PAHs over long periods in laboratory studies have developed lung cancer from inhalation, stomach cancer from ingesting PAHs in food, and skin cancer from skin contact. Benzo(a)pyrene is the most common PAH to cause cancer in animals and this compound is notable for being the first chemical carcinogen to be discovered. Based on the available evidence, both the International Agency for Research on Cancer (IARC, 1987) and US EPA (1994) classified a number of PAHs as carcinogenic to animals and some PAH-rich mixtures as

carcinogenic to humans. The EPA has classified seven PAH compounds as probable human carcinogens: benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(ah)anthracene, and indeno(1,2,3-cd)pyrene.

Teratogenicity

Embryotoxic effects of PAHs have been described in experimental animals exposed to PAH such as benzo(a)anthracene, benzo(a)pyrene, and naphthalene. Laboratory studies conducted on mice have demonstrated that ingestion of high levels of benzo(a)pyrene during pregnancy resulted in birth defects and decreased body weight in the offspring. It is not known whether these effects can occur in humans. However, the Center for Children's Environmental Health reports studies that demonstrate that exposure to PAH pollution during pregnancy is related to adverse birth outcomes including low birth weight, premature delivery, and heart malformations. High prenatal exposure to PAH is also associated with lower IQ at age three, increased behavorial problems at ages six and eight, and childhood asthma. Cord blood of exposed babies shows DNA damage that has been linked to cancer.

Genotoxicity

Genotoxic effects for some PAH have been demonstrated both in rodents and in vitro tests using mammalian (including human) cell lines. Most of the PAHs are not genotoxic by themselves and they need to be metabolised to the diol epoxides which react with DNA, thus inducing genotoxic damage. Genotoxicity plays important role in the carcinogenicity process and maybe in some forms of developmental toxicity as well.

Immunotoxicity

PAHs have also been reported to suppress immune reaction in rodents. The precise mechanisms of PAH-induced immunotoxicity are still not clear; however, it appears that immunosuppression may be involved in the mechanisms by which PAH induce cancer.

(Photo by Zakysant from de.wikipedia.org)

Environmental Fate and Ecotoxic Effects

PAHs are usually released into the air, or they evaporate into the air when they are released to soil or water. PAHs often adsorb to dust particles in the atmosphere, where they undergo photo oxidation in the presence of sunlight, especially when they are adsorbed to particles. This oxidation process can break down the chemicals over a period of days to weeks.

Since PAHs are generally insoluble in water, they are generally found adsorbed on particulates and precipitated in the bottom of lakes and rivers, or solubilized in any oily matter which may contaminate water, sediments, and soil. Mixed microbial populations in sediment/water systems may degrade some PAHs over a period of weeks to months.

The toxicity of PAHs to aquatic organisms is affected by metabolism and photo-oxidation, and they are generally more toxic in the presence of ultraviolet light. PAHs have moderate to high acute toxicity to aquatic life and birds. PAHs in soil are unlikely to exert toxic effects on terrestrial invertebrates, except when the soil is highly contaminated. Adverse effects on these organisms include tumors, adverse effects on reproduction, development, and immunity. Mammals can absorb PAHs by various routes e.g. inhalation, dermal contact, and ingestion.

Plants can absorb PAHs from soils through their roots and translocate them to other plant parts. Uptake rates are generally governed by concentration, water solubility, and their physicochemical state as well as soil type. PAH-induced phytotoxic effects are rare, howerver the database on this is still limited. Certain plants contain substances that can protect against PAH effects, whereas others can synthesize PAHs that act as growth hormones.

PAHs are moderately persistent in the environment, and can bioaccumulate. The concentrations of PAHs found in fish and shellfish are expected to be much higher than in the environment from which they were taken. Bioaccumulation has been also shown in terrestrial invertebrates, however PAH metabolism is sufficient to prevent biomagnification.

Regulation

U.S. government agencies have established standards that are relevant to PAHs exposures in the workplace and the environment. There is a standard relating to PAH in the workplace, and a standard for PAH in drinking water.

The Occupational Safety and Health Administration (OSHA) regulated exposures to PAHs under OSHA's Air Contaminants Standard for substances termed coal tar pitch volatiles (CTPVs) and coke oven emissions. Employees exposed to CTPVs in the coke oven industry are covered by the coke oven emissions standard. The OSHA coke oven emissions standard requires employers to control employee exposure to

coke oven emissions by the use of engineering controls and work practices. Wherever the engineering and work practice controls which can be instituted are not sufficient to reduce employee exposures to or below the permissible exposure limit, the employer shall nonetheless use them to reduce exposures to the lowest level achievable by these controls and shall supplement them by the use of respiratory protection. The OSHA standard also includes elements of medical surveillance for workers exposed to coke oven emissions.

The OSHA PEL (permisable exposure levels) for PAHs in the workplace is 0.2 mg/m3 for 8-hour TWA (time-weighted average).

The National Institute for Occupational Safety and Health (NIOSH) has recommended that the workplace exposure limit for PAHs be set at the lowest detectable concentration, which was 0.1 mg/m3 (REL=recommended exposure limit) for coal tar pitch volatile agents for a 10-hour workday, 40-hour workweek.

In 1980, EPA developed ambient water quality criteria to protect human health from the carcinogenic effects of PAH exposure. The recommendation was a goal of zero (nondetectable level for carcinogenic PAHs in ambient water). EPA, as a regulatory agency, sets a maximum contaminant level (MCL) for benzo(a)pyrene, the most carcinogenic PAH, at 0.2 ppb.

Recommendations for the Protection of Human Health and the Environment

The International Programme on Chemical Safety offers these general guidelines for protecting human health.

Owing to their proven immunotoxic effects, coal-tar shampoos should be used for anti-dandruff therapy only if no other treatment is available.
In view of the proven immunotoxic and carcinogenic effects of PAH in coke-oven workers, exposure to PAH in occupational settings should be eliminated or minimized by reducing emissions to the extent possible or, when they cannot be sufficiently reduced, by providing effective personal protection.
Public education about the sources and health effects of exposure to PAH should be improved. Use of unvented indoor fires, as in many developing countries, should be discouraged, and they should be replaced by more efficient, well-vented combustion devices. The risk of exposure to PAH from passive smoking should be stressed and measures taken to avoid it. Urban air pollution should be monitored all year round and not only seasonally.

This programme also provides suggestions on ways to reduce PAH emissions:

- filtration and scrubbing of industrial emissions,
- treatment of effluents,
- use of catalytic converters and particle traps on motor vehicles.

Relevant Topics on Toxipedia

- Persistent Environmental Contaminants
- Oil Dispersant
- What Causes Cancer?
- Bioaccumalation

Breaking News

PAHs News from * Environmental Health News *

Breaking Stories
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Go Green, Jennifer Parrish: Is burning right for you?
BP Oil Spill Taking Toll on Louisiana Indian Tribe
FDA seafood testing procedures called inadequate for Gulf residents
We can't afford to get it wrong at Barangaroo
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Oil spill's toxic trade-off
Pollution worsens asthma symptoms in kids

Scientific studies
Relationships among Polycyclic Aromatic Hydrocarbon-DNA Adducts, Proximity to the World Trade Center, and Effects on Fetal Growth
A summary of recent findings on birth outcomes and developmental effects of prenatal ETS, PAH, and pesticide exposures.
PAH-DNA Adducts in Cord Blood and Fetal and Child Development in a Chinese Cohort
International Studies of Prenatal Exposure to Polycyclic Aromatic Hydrocarbons and Fetal Growth
Increased health risk in Bangkok children exposed to polycyclic aromatic hydrocarbons from traffic-related sources.
Prenatal PAHs linked to lower cognition at age five.
Plants take up drugs, antibacterials from biosolids used as fertilizers.
Prenatal exposure to air pollutants lowers children's IQ.
Local children perform better on tests after coal-burning power plant closes.
Environmental pollutants and breast cancer

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