POLYCYCLIC AROMATIC HYDROCARBONS - III

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.

Source : http://www.toxipedia.org/display/toxipedia/Polycyclic+Aromatic+Hydrocarbons