

POLYCYCLIC AROMATIC HYDROCARBONS - INFORMATION SHEET

THE COMPOUNDS

Polycyclic aromatic hydrocarbons (PAH) are a large group of compounds made up of two or more fused benzene rings. They are primarily formed by incomplete combustion or pyrolysis of organic matter and during various industrial processes. Consequently, they are found in automobile exhaust, smoke from fires or cigarettes and as a component of air pollution. PAHs generally occur in complex mixtures which may consist of hundreds of compounds.

SOURCES

PAH are formed whenever organic matter is burned. Emissions from vehicles, domestic heating, agricultural fires and industrial processes are likely to be important sources of atmospheric PAH in New Zealand. PAH are ubiquitous in the environment which means that food, air, water and soil are all potential routes of exposure. For non-smokers, food is regarded as the primary route of exposure.

Foods which have been shown to have the highest levels of PAH include charcoal broiled or smoked meats, leafy vegetables, grains and fats and oils. The presence of PAH in leafy vegetables is believed to be due to atmospheric deposition (Benford *et al.*, 2006).

There is little information on PAH levels in New Zealand foods. In 1976 levels in a variety of plant oils, fats and smoked foods were similar or lower than overseas values. A more recent study of margarines available in New Zealand showed total levels for the 16 priority pollutants of less than 20 µg/kg although this was occasionally exceeded, with levels up to 140 µg/kg (Thomson *et al.*, 1993).

POTENTIAL HEALTH EFFECTS

Not all PAH show biological activity and there is often significant variation between isomers. There is evidence from cell culture and animal testing that certain PAH are mutagenic, genotoxic and carcinogenic.

Epidemiological evidence has correlated PAH exposure from cigarettes and urban air pollution with cancer incidence. Little information is available on health effects of PAH in humans due to dietary exposure. Two studies have linked PAH exposure to gastric cancer (Dungal and Sigurjonsson, 1967; López-Abente *et al.*, 2001). However, these studies represent quite unusual dietary circumstances (high consumption of smoking fish and meat and consumption of wine from leather bottles lined with a tar-like substance, respectively) and lack statistical power. The limited number of animal studies that involved oral administration of PAHs also resulted in tumours of the gastrointestinal tract, as well as the liver, lungs and mammary glands (Panel on Contaminants in the Food Chain, 2008).

ESTIMATES OF DIETARY EXPOSURE

An estimate for New Zealand made in 1994 gave an intake of 11 carcinogenic PAH from food of 4.8 µg/day (benzo(a)pyrene intake 0.23 µg/day) (Thomson and Lake, 1994). This was considered to be slightly high as the food consumption data used for the estimate were too high. A more recent estimate of dietary benzo(a)pyrene exposure for New Zealanders gave mean exposures in the range 0.04-0.16 µg/day and high (95th percentile) exposures in the range 0.1-0.27 µg/day (Benford *et al.*, 2006). It should be noted that these estimates were calculated using UK data for the concentrations of PAH in foods.

A US study in 1992 estimated that the median daily dietary exposure to the carcinogenic PAH was 3 µg/day (c.f. air 0.05 µg/day, water 0.006 mg/day and soil 0.096 µg/day) (Menzie *et al.*, 1992). The exposure from 1 pack of cigarettes was put at 2-5 µg/day.

The FAO/WHO Joint Expert Committee on Food Additives (JECFA) summarised dietary exposure estimates from a number of countries finding estimates of benzo(a)pyrene exposure in the range <1-2.0 µg/day and for a further nine PAHs in the range <1-12 µg/day (Benford *et al.*, 2006).

The European Food Safety Authority (EFSA) Panel on Contaminants in the Food Chain estimates dietary PAH exposure for a range of European countries (Panel on Contaminants in the Food Chain, 2008). For benzo(a)pyrene alone estimates were in the range 0.24-0.39 µg/day, while for the sum of eight PAH median estimates were in the range 1.7-3.1 µg/day.

FACTORS INFLUENCING RISK

PAH are effective inducers of cancer when applied to the skin of animals but their ability to do the same when ingested is much less. There is little epidemiological evidence for PAH risk from ingestion. Although the exposure from foods is comparable with cigarette smoking, the difference in route of exposure makes comparisons uncertain.

While current estimates of dietary exposure are considered to be of low concern for human health, certain food processing measures (avoidance of direct contact of food with flames or cooking by heating from above, replacement of direct smoking of food with indirect smoking, peeling of fruits and vegetables) would help to reduce exposure.

SAFETY ASSESSMENTS

The first PAH to be identified as carcinogenic was benzo(a)pyrene. Many early reports focus on this compound as an indicator of overall PAH contamination although it probably only contributes a small percentage of the total carcinogenicity of the PAH present in a mixture.

The US Environmental Protection Agency has identified sixteen PAH as "priority pollutants" for attention and eight of these (generally the larger PAH with four or more fused rings) are considered to be potential human carcinogens.

A similar approach was taken by JECFA, which concluded that 13 PAHs were probably genotoxic carcinogens (Benford *et al.*, 2006). Using benzo(a)pyrene as a surrogate for PAH exposure, JECFA concluded that current estimates of dietary exposure were of low concern for human health, but that certain food processing measures (avoidance of direct contact of food with flames or cooking by heating from above, replacement of direct smoking of food with indirect smoking, peeling of fruits and vegetables) would help to reduce exposure.

EFSA's Panel on Contaminants in the Food Chain have also assessed PAHs and concluded that average estimated levels of exposure were of low concern for human health, but that exposure to PAHs should be kept as low as reasonably achievable (ALARA) (Panel on Contaminants in the Food Chain, 2008). The panel noted that estimates of exposure for high level consumers indicated a potential cause for concern and a possible need for risk management action. The Panel also concluded that benzo(a)pyrene was not a suitable marker for the presence of PAHs in food and instead suggested use of a group of four or eight PAHs.

The carcinogenicity of individual PAHs has recently been re-evaluated, with benzo(a)pyrene being upgraded to group 1 (carcinogenic to humans), while several other PAHs (cyclopenta(cd)pyrene, dibenz(a,h)anthracene, dibenzo(a,l)pyrene) have been upgraded from group 2B (possibly carcinogenic to humans) to 2A (probably carcinogenic to humans (International Agency for Research on Cancer, In preparation).

SAFETY AND REGULATORY LIMITS

Safety limits are levels of dietary exposure that are without appreciable risk for a lifetime of exposure. Regulatory limits define the maximum amount of a substance that is permitted in a particular food.

Source	Limit Type	Limit
<i>Safety Limits</i>		
Due to their suspected human carcinogenicity, no safety limits have been set for PAHs. It is generally considered that their presence in the diet should be as low as reasonably achievable (ALARA)		
<i>Regulatory Limits</i>		
No regulatory limits for PAHs in food have been set for New Zealand or Australia		

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