

HEALTH EFFECTS: GENOTOXICITY AND CARCINOGENICITY

Polycyclic aromatic hydrocarbons (PAHs) in food deserve attention because some members of the family can damage genetic material and contribute to cancer development. However, not all PAHs have the same biological activity, and finding a substance in food does not, by itself, demonstrate harm to consumers. Understanding risk requires distinguishing a substance's properties from the amount actually absorbed and the duration of exposure.

ONE CHEMICAL FAMILY, DIFFERENT HAZARDS

PAHs are numerous and differ in structure, absorption, metabolism and toxicological potency. Some have been extensively studied, while information on others remains insufficient. The International Agency for Research on Cancer (IARC) assigns carcinogenicity classifications to individual substances or particular mixtures and exposure circumstances: the same classification cannot automatically be assigned to the entire family.

Benzo[a]pyrene is especially significant: IARC classifies it in Group 1, carcinogenic to humans. This classification describes the strength of the evidence for a carcinogenic hazard, not the probability that a particular ingested amount will cause cancer. Evidence is less complete, or classifications differ, for other PAHs.

FROM INGESTION TO ABSORPTION

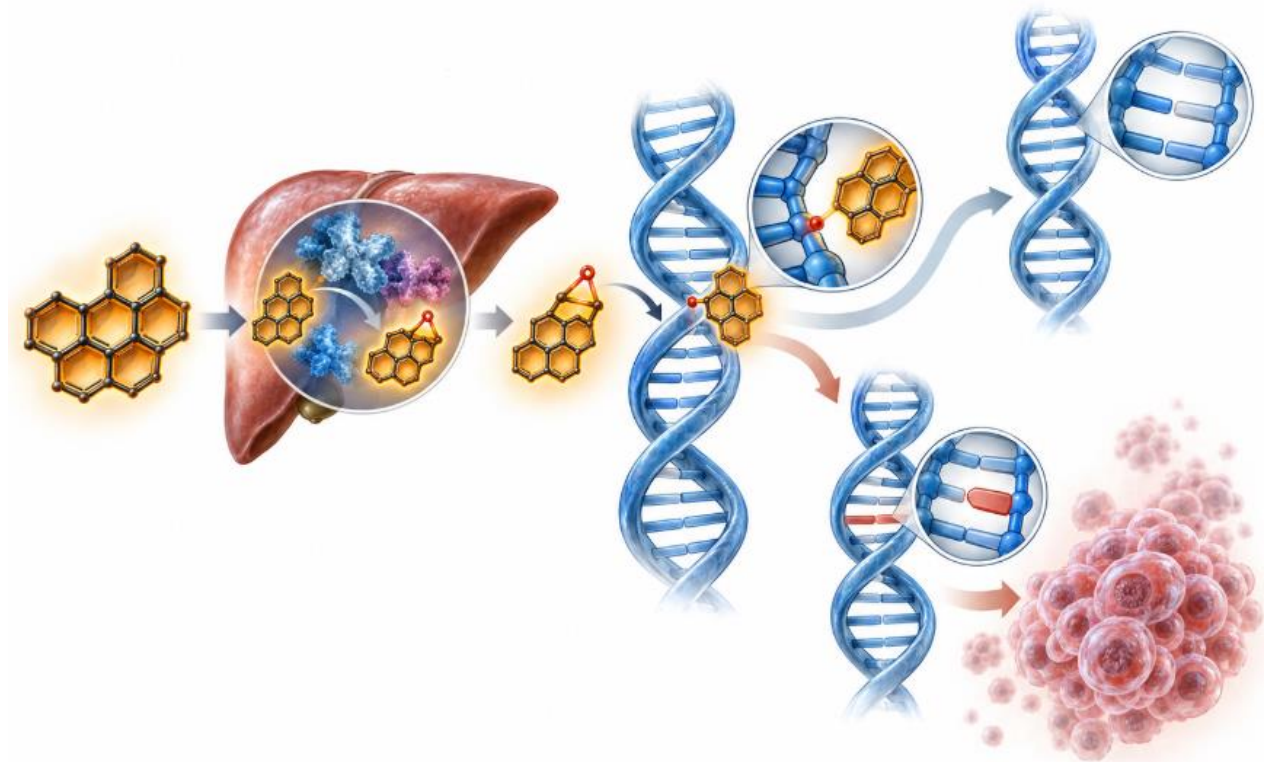
When PAHs are ingested in food, only a fraction may become available for absorption. The absorbed fraction depends on the molecule, food matrix and digestive conditions. Because many PAHs are lipophilic, the fat content of food can influence their availability; nevertheless, lipophilicity alone cannot predict the absorbed dose.

After absorption, PAHs are transformed by enzyme systems primarily in the liver, but also in other tissues. Metabolic transformations often facilitate elimination, yet in some cases they produce intermediate compounds more reactive than the original molecule. This is why certain PAHs are described as undergoing metabolic activation.

HOW DNA DAMAGE MAY OCCUR

A well-documented activation pathway for benzo[a]pyrene produces metabolites capable of binding to DNA and forming so-called adducts. If damage is not properly repaired

before cell replication, mutations may occur. The accumulation of genetic changes, together with other biological events, can contribute to cancer development. Formation of an adduct does not mean that cancer will necessarily arise: cells have repair and defence mechanisms, and outcomes depend on many factors.



Genotoxicity and carcinogenicity are not synonyms. The former is the ability to damage genetic material; the latter is the ability to cause cancer. Genetic damage can be an important mechanism in carcinogenesis, but overall assessment requires experimental evidence, information about mechanisms of action and, where relevant, epidemiological data.

WHAT SCIENTIFIC STUDIES SHOW

Animal studies provide essential evidence on the carcinogenicity of various PAHs. Research in cells and biological systems helps explain the underlying mechanisms. In humans, it is harder to isolate the contribution of dietary PAHs because exposure

usually involves mixtures and overlaps with other factors, including smoking, air pollution and dietary habits.

Epidemiological findings from occupational or inhalation exposure cannot simply be transferred to quantities ingested with food without further consideration. Food-specific risk assessment therefore requires concentration data, food-consumption information and appropriate dose–response relationships.

FROM HAZARD TO DIETARY RISK

A contaminant may be hazardous because of its intrinsic properties, but health risk depends on exposure. Two foods containing the same concentration of a PAH can make very different contributions to total exposure if portion sizes or consumption frequencies differ. Similarly, a measurable concentration does not automatically imply high risk.

For genotoxic and carcinogenic substances, EFSA uses the Margin of Exposure (MOE), comparing a reference dose derived from toxicological studies with estimated population exposure. In this context, the MOE is neither a threshold automatically separating ‘safe’ from ‘dangerous’ nor an individual’s probability of developing disease. In general, a lower MOE indicates a greater priority for attention and risk management.

In its 2008 scientific opinion on PAHs in food, EFSA examined indicators based on groups of compounds as well as benzo[a]pyrene individually. The choice of indicators and how contamination should be interpreted are examined further in Chapter 7.

OTHER POSSIBLE EFFECTS AND KNOWLEDGE GAPS

Toxicological research also investigates developmental, reproductive and immune-system effects for some PAHs or mixtures. Findings cannot be applied uniformly to every compound and do not always allow reliable quantification of risk from usual dietary exposures. Associations or experimental findings must not be presented as proof of a certain clinical effect in consumers.

WHAT THIS MEANS FOR CONSUMERS

The scientifically appropriate conclusion is neither that every trace of PAHs causes cancer nor that a compliant food results in zero exposure. Where possible, avoidable sources of contamination, especially during heat processing, should be reduced, while assessments should rely on representative analytical data, food-consumption patterns and toxicology. Official surveillance and regulatory limits are essential prevention tools, although they cannot eliminate every scientific uncertainty.

SCIENTIFIC REFERENCES

[EFSA CONTAM Panel \(2008\). Polycyclic Aromatic Hydrocarbons in Food. EFSA Journal, 6\(8\), 724. https://doi.org/10.2903/j.efsa.2008.724](https://doi.org/10.2903/j.efsa.2008.724)

[IARC \(2012\). Chemical Agents and Related Occupations, Monographs Volume 100F, section on Benzo\[a\]pyrene.](#)

[IARC. Agents Classified by the IARC Monographs \(current list and classifications\).](#)

[EFSA \(2026\). Margin of Exposure — explanation of the MOE approach.](#)