### Key Points

**Fire**
- Combustible
- Reacts violently with oxidising agents
- Emits toxic and irritating fumes on oxidation

**Health**
- Toxic by inhalation, ingestion or dermal absorption
- Carcinogen, mutagen and reproductive toxin
- Inhalation may cause respiratory tract irritation, damage the reproductive system and cause cancer
- Ingestion may cause gastrointestinal irritation
- Dermal exposure may cause skin irritation

**Environment**
- Dangerous for the environment
- Inform Environment Agency of substantial incidents
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Polycyclic aromatic hydrocarbons
(Benzo[a]pyrene)

General information

Key Points

Identity
- The term polycyclic aromatic hydrocarbons (PAHs) refers to a group of several hundred chemically-related environmentally persistent organic compounds of various structures and varied toxicity
- Benzo[a]pyrene (BaP) is commonly used as an indicator species for PAH contamination and most of the available data refer to this compound

Fire
- May react violently with oxidising agents
- Emits toxic and irritating fumes on decomposition
- In the event of fire involving PAHs, use fine water spray and liquid tight chemical protective equipment

Health
- Toxic by inhalation, ingestion or skin absorption
- Carcinogen, mutagen and reproductive toxin
- Long-term inhalation can cause a decrease in lung function, chest pain and irritation
- Long-term skin contact can cause dermatitis and warts
- BaP is thought to probably cause lung and skin cancer in humans

Environment
- Environmentally hazardous substance
Inform Environment Agency of substantial release incidents
Polycyclic aromatic hydrocarbons (PAHs) are a diverse class of organic compounds. There are several hundred PAHs, which usually occur as complex mixtures rather than as individual compounds. The most well known PAH is benzo[a]pyrene (BaP), on which this report focuses. PAH are flammable, colourless solids or crystals at room temperature with no perceptible odour.

PAHs may be formed during natural processes such as incomplete combustion of organic materials such as coal and wood, or during forest fires. PAHs are released during industrial activities such as aluminium, iron and steel production in plants and foundries, waste incineration, mining or oil refining. PAHs have also been detected at low levels in cigarette smoke and motor vehicle emissions. They are persistent organic pollutants and are slow to degrade in the environment.

Overall, 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.
Production and Uses

Key Points

- PAHs are produced due to the incomplete combustion of organic materials
- PAHs are components of asphalt and roofing tar

The major source of PAHs is the incomplete combustion of organic material such as coal, oil and wood rather than industrial sources.

There are few commercial uses for many PAHs, including BaP. Anthracene is used in the production of dyes, synthetic fibres, and acenaphthene is used in the manufacture of dyes, plastics, pigments, pharmaceuticals and pesticides. Other PAHs may be contained in asphalt used for the construction of roads, as well as roofing tar.
Frequently Asked Questions

What are PAHs?

PAHs are a group of chemicals that are produced during the incomplete combustion of organic substances such as coal, oil, gas and wood.

How do PAHs get into the environment?

The main way PAHs can enter the environment is due to the incomplete combustion of organic materials such as coal and wood, from forest fires and from vehicle exhausts.

How will I be exposed to PAHs?

The major sources of PAHs to the general public include inhalation of tobacco smoke, wood smoke and ambient air, and consumption of PAHs in food such as cereals, bread or processed food, as well as fruit and vegetables grown in contaminated soil. Charbroiling and grilling food at high temperatures also increases the amount of PAHs in the food. Other sources of PAHs include vehicle exhausts, asphalt roads and waste incineration.

Occupational exposure may occur in workers inhaling engine exhaust, such as mechanics, street vendors and drivers, as well as those working in industries such as mining, oil refining and metal working.

If there are PAHs in the environment will I have any adverse health effects?

The presence of PAHs in the environment does not always lead to exposure as you must come into contact with the chemical. You may be exposed by breathing, eating, or drinking the substance or by skin contact. Following exposure to any hazardous chemical, the adverse health effects you may encounter depend on several factors, including the amount to which you are exposed (dose), the duration of exposure, the way you are exposed and if you were exposed to any other chemicals.

Some PAHs, including BaP caused tumours in animals when they breathed, ate or had skin contact for long periods. Studies in humans have shown that breathing or skin contact for long periods may cause lung or skin cancer.

Can PAHs cause cancer?

Several PAHs, including BaP, have been classified by the International Agency for Research on Cancer (IARC) as being carcinogenic in humans.

Do PAHs affect children or damage the unborn child?

Animals fed high concentrations of BaP during pregnancy had future fertility problems, as did the offspring, which also showed some birth defects in some breeds of mice. There are few studies in humans so it is unclear whether these effects also occur in people.

What should I do if I am exposed to PAHs?

It is very unlikely that the general population will be exposed to a level of PAHs high enough to cause adverse health effects.
# Polycyclic aromatic hydrocarbons (Benzo[a]pyrene)

## Incident management

<table>
<thead>
<tr>
<th>Key Points</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fire</strong></td>
</tr>
<tr>
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<td>- Dermal exposure may cause skin irritation</td>
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<tr>
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<tr>
<td>- Inform Environment Agency of substantial incidents</td>
</tr>
</tbody>
</table>

Prepared by S Bull
CHAPD HQ, HPA
2008
Version 1
## Hazard Identification

*Standard (UK) Dangerous Goods Emergency Action Codes*

<table>
<thead>
<tr>
<th>UN</th>
<th>EAC</th>
<th>APP</th>
<th>Data not available</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hazards</td>
<td>Class</td>
<td>Sub risks</td>
<td></td>
</tr>
<tr>
<td>HIN</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

UN – United Nations number; EAC – Emergency Action Code; APP – Additional Personal Protection; HIN - Hazard Identification Number
### Chemical Hazard Information and Packaging for Supply Classification

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
<th>Symbol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carc. Cat 2</td>
<td>Category 2 carcinogen</td>
<td><img src="image" alt="Skull and Crossbones" /></td>
</tr>
<tr>
<td>Muta. Cat 2</td>
<td>Category 2 mutagen</td>
<td><img src="image" alt="Skull and Crossbones" /></td>
</tr>
<tr>
<td>Repr. Cat 2</td>
<td>Category 2 reproductive toxin</td>
<td><img src="image" alt="Skull and Crossbones" /></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk phrases</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>R43</td>
<td>May cause sensitisation by skin contact</td>
</tr>
<tr>
<td>R45</td>
<td>May cause cancer</td>
</tr>
<tr>
<td>R46</td>
<td>May cause heritable genetic damage</td>
</tr>
<tr>
<td>R60</td>
<td>May impair fertility</td>
</tr>
<tr>
<td>R61</td>
<td>May cause harm to the unborn child</td>
</tr>
<tr>
<td>R43</td>
<td>May cause sensitisation by skin contact</td>
</tr>
<tr>
<td>R50/53</td>
<td>Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Safety phrases</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>S53</td>
<td>Avoid exposure - obtain special instructions before use</td>
</tr>
<tr>
<td>S45</td>
<td>In case of accident or if you feel unwell seek medical advice immediately (show the label where possible)</td>
</tr>
<tr>
<td>S60</td>
<td>This material and its container must be disposed of as hazardous waste</td>
</tr>
<tr>
<td>S61</td>
<td>Avoid release to the environment. Refer to special instructions/safety data sheet</td>
</tr>
</tbody>
</table>

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### Specific concentration limits$^a$

<table>
<thead>
<tr>
<th>Concentration</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>$C \geq 25%$</td>
<td>$T, N; R45-46-60-61-43-50/53$</td>
</tr>
<tr>
<td>$2.5% \leq C &lt; 25%$</td>
<td>$T, N; R45-46-60-61-43-51/53$</td>
</tr>
<tr>
<td>$1% \leq C &lt; 2.5%$</td>
<td>$T; R45-46-60-61-43-52/53$</td>
</tr>
<tr>
<td>$0.5% \leq C &lt; 1%$</td>
<td>$T; R45-46-60-61-52/53$</td>
</tr>
<tr>
<td>$0.25% \leq C &lt; 0.5%$</td>
<td>$T; R45-46-52/53$</td>
</tr>
<tr>
<td>$0.1% \leq C &lt; 0.25%$</td>
<td>$T; R45-46$</td>
</tr>
<tr>
<td>$0.01% \leq C &lt; 0.1%$</td>
<td>$T; R45$</td>
</tr>
</tbody>
</table>

## Physicochemical Properties

<table>
<thead>
<tr>
<th>Property</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CAS number</strong></td>
<td>50-32-8</td>
</tr>
<tr>
<td><strong>Molecular weight</strong></td>
<td>252</td>
</tr>
<tr>
<td><strong>Empirical formula</strong></td>
<td>C_{20}H_{12}</td>
</tr>
<tr>
<td><strong>Common synonyms</strong></td>
<td>3,4-Benzo[pyrene; Benzo(d,e,f)chrysene Note; polycyclic hydrocarbons are now also commonly referred to as polycyclic hydrocarbons</td>
</tr>
<tr>
<td><strong>State at room temperature</strong></td>
<td>Solid, crystals</td>
</tr>
<tr>
<td><strong>Volutility</strong></td>
<td>Vapour pressure negligible at 25°C</td>
</tr>
<tr>
<td><strong>Specific gravity</strong></td>
<td>1.4 at 25°C (water = 1)</td>
</tr>
<tr>
<td><strong>Flammability</strong></td>
<td>Combustible</td>
</tr>
<tr>
<td><strong>Lower explosive limit</strong></td>
<td>Data not available</td>
</tr>
<tr>
<td><strong>Upper explosive limit</strong></td>
<td>Data not available</td>
</tr>
<tr>
<td><strong>Water solubility</strong></td>
<td>Practically insoluble in water at 25°C. Soluble in benzene, toluene and xylene. Sparingly soluble in ethanol and methanol</td>
</tr>
<tr>
<td><strong>Reactivity</strong></td>
<td>Benzo[a]pyrene decomposes in light and air. Reacts violently with oxidising agents and may be decomposed by strong acids</td>
</tr>
<tr>
<td><strong>Reaction or degradation products</strong></td>
<td>Releases toxic and irritating fumes on decomposition</td>
</tr>
<tr>
<td><strong>Odour</strong></td>
<td>Odourless</td>
</tr>
<tr>
<td><strong>Structure</strong></td>
<td>![Structure Image]</td>
</tr>
</tbody>
</table>

### References

Threshold Toxicity Values

<table>
<thead>
<tr>
<th>EXPOSURE VIA INGESTION</th>
<th>ppm</th>
<th>mg m⁻³</th>
<th>SIGNS AND SYMPTOMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Data not available</td>
</tr>
</tbody>
</table>
Published Emergency Response Guidelines

Emergency Response Planning Guideline (ERPG) Values\(^{(a)}\)

<table>
<thead>
<tr>
<th>Listed value (ppm)</th>
<th>Calculated value (mg m(^{-3}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>ERPG-1*</td>
<td></td>
</tr>
<tr>
<td>ERPG-2**</td>
<td>Data not available</td>
</tr>
<tr>
<td>ERPG-3***</td>
<td></td>
</tr>
</tbody>
</table>

* Maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hr without experiencing other than mild transient adverse health effects or perceiving a clearly defined, objectionable odour.

** Maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hr without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action.

*** Maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hr without experiencing or developing life-threatening health effects.

Interim Acute Exposure Guideline Levels (AEGLs)\(^{(b)}\)

<table>
<thead>
<tr>
<th>ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 min</td>
</tr>
<tr>
<td>AEGL-1†</td>
</tr>
<tr>
<td>AEGL-2‡‡</td>
</tr>
<tr>
<td>AEGL-3‡‡‡</td>
</tr>
</tbody>
</table>

Data not available

† The level of the chemical in air at or above which the general population could experience notable discomfort.

‡‡ The level of the chemical in air at or above which there may be irreversible or other serious long-lasting effects or impaired ability to escape.

‡‡‡ The level of the chemical in air at or above which the general population could experience life-threatening health effects or death.


Exposure Standards, Guidelines or Regulations

**Occupational standards**

<table>
<thead>
<tr>
<th>WEL</th>
<th>LTEL (8 hour reference period): No guideline value specified</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>STEL (15 min reference period): No guideline value specified</td>
</tr>
</tbody>
</table>

**Public health guidelines**

<table>
<thead>
<tr>
<th>DRINKING WATER QUALITY GUIDELINE(^{(a)})</th>
<th>Benzo[(a)]pyrene; 0.01 (\mu g) L(^{-1}) ()PAH; 0.1 (\mu g) L(^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIR QUALITY GUIDELINE(^{(b)})</td>
<td>1.2, 0.12 and 0.012 ng m(^{-3}) for a excess lifetime cancer risk of 1:10000, 1:100000 and 1:1000000 respectively</td>
</tr>
<tr>
<td>SOIL GUIDELINE VALUE AND HEALTH CRITERIA VALUES(^{(c)})</td>
<td><strong>Index dose</strong> (_{\text{inhalation}})(^{\text{(0.07 \times 10^{-3} \mu g \text{ kg}^{-1} \text{ bw day}^{-1})}})</td>
</tr>
<tr>
<td></td>
<td><strong>Index dose</strong> (_{\text{oral}})(^{\text{(0.02 \mu g \text{ kg}^{-1} \text{ bw day}^{-1})}})</td>
</tr>
</tbody>
</table>

WEL – Workplace exposure limit; LTEL - Long-term exposure limit; STEL – Short-term exposure limit

---


Health Effects

Major route of exposure\(^{(a)}\)

- Toxic by inhalation, ingestion or dermal absorption.

Immediate signs or symptoms of acute exposure

- Inhalation may cause respiratory tract irritation.
- Ingestion may cause gastrointestinal irritation.
- Dermal exposure may cause skin irritation.

---

\(^{a}\) TOXBASE: Benzo[a]pyrene - SEPA, 2005.
Decontamination and First Aid

Important Notes

- Ambulance staff, paramedics and emergency department staff treating chemically-contaminated casualties should be equipped with Department of Health approved, gas-tight (Respirex) decontamination suits based on EN466:1995, EN12941:1998 and prEN943-1:2001, where appropriate.
- Decontamination should be performed using local protocols in designated areas such as a decontamination cubicle with adequate ventilation.

Dermal exposure\(^\text{(a)}\)

- Remove patient from exposure.
- The patient should remove all clothing and personal effects.
- Double-bag soiled clothing and place in a sealed container clearly labelled as a biohazard.
- Brush away any adherent solid particles from the patient.
- Wash hair and all contaminated skin with copious amounts of water (preferably warm) and soap for at least 10-15 minutes. Decontaminate open wounds first and avoid contamination of unexposed skin.
- Pay special attention to skin folds, axillae, ears, fingernails, genital areas and feet.

Ocular exposure\(^\text{(b)}\)

- Remove patient from exposure.
- Remove contact lenses if necessary and immediately irrigate the affected eye thoroughly with water or 0.9% saline for at least 10-15 minutes.
- Patients with corneal damage or those whose symptoms do not resolve rapidly should be referred for urgent ophthalmological assessment.

Inhalation\(^\text{(c)}\)

- Remove patient from exposure.
- Ensure a clear airway and adequate ventilation.
- Give oxygen to symptomatic patients.
- Apply other supportive measures as indicated by the patient’s clinical condition.

Ingestion\(^\text{(c)}\)

- Give oxygen to symptomatic patients.
- Apply other supportive measures as indicated by the patient’s clinical condition.

TOXBASE - http://www.toxbase.org
\(^\text{a}\) TOXBASE: Skin decontamination – irritants, 1996.
\(^\text{b}\) TOXBASE: Eye irritants, 2002.
\(^\text{c}\) TOXBASE: Benzo[a]pyrene - SEPA, 2005.
Polycyclic aromatic hydrocarbons
(Benzo[a]pyrene)

Toxicological overview

Key Points

Kinetics and metabolism

- Benzo[a]pyrene (BaP) is readily absorbed following inhalation, ingestion and skin exposure
- It is rapidly distributed to the kidney, small intestine, trachea, stomach, testes, liver and oesophagus
- BaP is metabolised by cytochrome P450 enzymes to form a reactive epoxide metabolite
- Metabolites are excreted in the urine or faeces

Health effects of acute exposure

- Few studies were identified that reported the effects of BaP in humans following acute inhalation, ingestion or dermal exposure

Health effects of chronic exposure

- Chronic inhalation of BaP may cause a decrease in respiratory function, chest pain and irritation and cough and chronic skin exposure may lead to dermatological effects such as warts
- BaP is considered to be carcinogenic to humans

Prepared by S Bull
CHAPD HQ, HPA
2008
Version 1
Toxicological Overview

Summary of Health Effects

Polycyclic aromatic hydrocarbons (PAHs) are a group of hydrocarbons that are mainly formed by the incomplete combustion of organic materials. There are several hundred PAHs, which usually occur as complex mixtures rather than as individual compounds. Benzo[a]pyrene (BaP) is the most widely studied, being one of the most potent, hence most of the data in this document refers to BaP, although it seldom occurs in the environment on its own.

For the general public, the main route of exposure to PAHs is from inhalation of ambient and indoor air or ingestion of food. Inhalation and skin absorption are the main routes of occupational exposure.

BaP is readily absorbed following inhalation, ingestion and skin exposure. Following inhalation and ingestion, BaP is rapidly distributed to several tissues in rats, including the kidney, small intestine, trachea, stomach, testes, liver and oesophagus. BaP is metabolised by cytochrome P450 enzymes resulting in a number of metabolites being formed, including the reactive epoxide metabolite, BaP 7,8 diol-9,10-epoxide, which is believed to be responsible for its carcinogenicity. Following metabolism the metabolites are excreted in the urine and faeces.

No data on the acute effects of BaP in humans were identified and few studies were reported in animals. Following acute exposure of rats to BaP, effects on the liver were observed.

Following chronic exposure in an occupational setting a decrease in lung function was reported, as well as chest pain, respiratory irritation, cough, dermatitis and depressed immune system, although in most cases it was not possible to evaluate the contribution of BaP to such effects. In animals, few adverse effects were observed in rats or hamsters exposed to BaP via inhalation. Following ingestion, myelotoxicity was observed in poor affinity Ah-receptor mice but not in high affinity mice. Hepatotoxicity was also reported.

BaP can cross the placenta and was found to cause adverse developmental and reproductive effects in mice. Dietary administration during gestation reduced fertility and fetal abnormalities whereas administration by gavage caused an increase in fetal death and decreased fertility.

Numerous epidemiologic studies have shown an association between exposure to various mixtures of PAHs containing BaP and increased risk of lung and skin cancer. However, it was not possible to evaluate the contribution of BaP to the carcinogenicity of these mixtures.

In animals, short-term dietary administration of BaP caused forestomach tumours in mice and hamsters. Chronic exposure of mice to BaP by gavage or in the diet resulted in forestomach and lung tumours and in rats an increase in tumours of the forestomach, oesophagus, liver, larynx and mammary gland was observed.

Chronic inhalation of BaP caused an increase in lung tumours in mice, and tumours of the nasal cavity, pharynx, trachea, oesophagus and forestomach in hamsters.

Many studies, in which BaP has been topically applied to various species, have shown that BaP can induce skin tumours, although mice appear to be the most sensitive.
**Kinetics and metabolism**

PAHs are lipophilic compounds that are readily absorbed from the lungs following inhalation, the gastrointestinal (GI) tract following ingestion and the skin following dermal exposure [1].

In humans, it was reported that BaP measured in the lungs following inhalation of soot particles was much lower than expected. This may be due to the ability of the pulmonary epithelial cells to metabolise BaP thereby facilitating its absorption and clearance from the lungs [2]. Occupational studies have inferred that inhaled PAHs are absorbed by humans, as urinary metabolites were present in workers exposed to PAHs [3]. The absorption of BaP following inhalation is highly dependent on the type of particles onto which it is adsorbed. Pulmonary absorption often occurs in parallel with mucociliary clearance, by which PAHs that are absorbed onto inhaled particulates are cleared out of the pulmonary tree and subsequently swallowed [2, 4].

Few data were available regarding the absorption in humans following ingestion, but in general it is thought to be low [3]. However, one study reported that most of a low oral dose of BaP was systemically absorbed as no BaP was detected in faeces, although the number of volunteers in the study was limited [4].

In animals, approximately 30% of absorption occurred through the GI tract following administration of a low dose of BaP directly into the duodenum, whereas slightly higher absorption occurred following administration of a high dose of BaP given by gavage or in the diet [2, 4, 5].

Percutaneous absorption of PAHs appears to be quite rapid in both animals and humans [3]. Extensive skin absorption has been demonstrated in mice as almost all of the applied dose of BaP appeared in the faeces following application to the skin [4]. Similarly, rapid absorption was demonstrated in rats, monkeys and guinea pigs [3].

No data were available regarding the distribution of PAHs in humans. *In-vivo*, PAHs appear to be widely distributed following both inhalation and ingestion, as levels have been detected in several organs [2, 3]. Following oral exposure in rats BaP was measured in the kidney, caecum, small intestine, trachea, stomach and testes, whereas following inhalation, levels were measured in the liver, oesophagus, stomach and small intestine, and later in the large intestine and caecum [3-5].

BaP can readily cross the placenta following oral, inhalation or dermal administration. One study reported that when pregnant rats were exposed to BaP via inhalation, an increase in BaP and metabolites was measured in both maternal and fetal blood and tissues. Similarly, BaP was measured in the fetus when rats were given oral BaP on day 21 of pregnancy [3, 5].

Many studies have investigated the metabolism of PAHs in tissues and cells following ingestion of food containing PAHs, or inhalation or ingestion of environmental PAHs. Consequently studies have been carried out in the bronchus, colon, keratinocytes, monocytes, macrophages and lymphocytes [2]. BaP is metabolised by microsomal cytochrome P-450 enzymes to a range of epoxides, the metabolites then undergoing phase II conjugation to form phenols, quinones and dihydriodiols. Dihydropolodiols undergo further oxidative metabolism to the carcinogenic metabolite BaP 7,8 diol-9,10-epoxide, which is believed to be the reactive metabolite responsible for the carcinogenicity of BaP [2, 5].

There are few data available regarding the excretion of PAHs in humans. In general, they are metabolised and the metabolites are excreted in the faeces and urine [2-4].
**Sources and route of human exposure**

PAHs are a large group of hydrocarbons containing two or more benzene rings fused together or to other hydrocarbon rings. They are mainly formed as pyrolysis by-products, especially during the incomplete combustion of organic materials during industrial and other human activities [4, 6]. There are several hundred PAHs, which usually exist as mixtures rather than as individual chemicals. BaP is the most well known and will be the focus of this compendium.

For the general population, the major sources of exposure to PAHs are from ambient and indoor air due to residential heating, cigarette smoke, coal and wood fires and vehicle exhaust, as well as from food. Various foods such as vegetables, meat and fish have been shown to contain PAHs, but they are largely formed due to the cooking at high temperatures such as charbroiling, grilling and frying. Smoked and barbequed food are particularly important sources of exposure [2], although the largest contribution to the daily PAH intake comes from oils and fats [2, 4, 6, 7].

PAHs are commonly detected in surface waters, due to urban runoff and industrial activities [2]. They are regularly monitored in UK drinking water for regulatory purposes. The main source of drinking water contamination with trace amounts of PAH is usually associated with coal-tar linings of the distribution pipes. However, drinking water contributes only a minor amount to the total intake of PAHs [4, 8].

PAHs are found in the majority of surface soils due to atmospheric deposition or urban runoff. Soils near industrial sources such as coal coking also often contain high concentrations of PAHs [2, 6].

Overall, the major route of exposure of the general public is through inhalation of ambient and indoor air and ingestion of food.

Occupational exposure is largely through inhalation and skin absorption. Workers employed in occupations such as road paving, asphalt roofing, aluminium plants, iron and steel foundries, as well as street vendors, firemen, mechanics and chimney sweeps may be occupationally exposed to PAHs [2].
Health Effects of Acute / Single Exposure

**Human Data**

**Inhalation**

No studies were identified that reported the effects of BaP in humans following acute inhalation exposure.

**Ingestion**

Data on acute oral toxicity of BaP in humans are not available.

**Dermal exposure**

No studies were identified that reported effects of BaP in humans following acute dermal exposure.

**Delayed effects following an acute exposure**

No data on delayed effects of BaP following an acute exposure were identified.

**Animal and In-Vitro Data**

**Inhalation**

No studies were identified that reported effects of BaP in animals following acute inhalation exposure.

**Ingestion**

Exposure of rats to 100 mg kg\(^{-1}\) bw day\(^{-1}\) BaP for four days increased relative liver weight by 27%, although administration of 51.4 mg kg\(^{-1}\) bw day\(^{-1}\) following partial hepatectomy did not cause an effect. Limited evidence suggested that acute ingestion of BaP (150 mg kg\(^{-1}\) bw day\(^{-1}\) for 4 days) does not cause adverse gastrointestinal effects in rats, although enzyme activity was altered. It was suggested that more serious effects may occur at higher concentrations [3]. Other studies have suggested that BaP has a fairly low toxicity in mice, it having a LD\(_{50}\) of more than 1600 mg kg\(^{-1}\) bw [4].

**Dermal exposure**

Acute topical application of BaP (concentration and duration of exposure not stated) to the backs of shaved mice suppressed sebaceous glands, although it was not possible to determine if such effects were due to the solvent or BaP as a control group was not used [3].
Health Effects of Chronic / Repeated Exposure

**Human Data**

**Inhalation**

One study investigated the respiratory effects of inhaled BaP in employees working in various areas of a rubber factory. The authors reported a decrease in ventilatory function following prolonged exposure, as assessed by duration of employment, the greatest effects being observed in workers that had the highest exposure to particulate matter and BaP. No attempt was made to identify other possible chemical exposures or to separate effects due to BaP or particulates [3].

Whilst many epidemiology studies have been carried out in various occupations, few have identified the role of individual compounds that contribute to symptoms including respiratory distress, chest pain, chest and throat irritation, cough, haematemesis, chronic dermatitis, depressed immune system and cancer or the skin and lung [3, 4].

**Ingestion**

Data on chronic oral toxicity of BaP in humans are not available.

**Dermal exposure**

Mixtures of PAHs have been reported to cause, and in some cases treat, skin disorders in humans, although few data are available about BaP alone.

Regressive verrucae (warts) were reported in humans following up to 120 applications of 1% BaP over a four month period [3].

**Genotoxicity**

Numerous studies on lymphocytes from workers exposed to PAHs (including BaP) have identified DNA adducts of BaP (mainly the diol epoxide). In one study on iron foundry workers, elevated levels of mutations at the hprt locus in lymphocytes were shown to correlate approximately with the levels of DNA adducts [2].

No studies were identified regarding genotoxic effects in humans following oral administration of BaP [3].

**Carcinogenicity**

No studies were reported regarding cancer in humans following inhalation of BaP alone [3].

There is extensive literature on the epidemiology of workforces exposed to complex mixtures of PAHs in, for example, asphalt works, coke production plants and aluminium smelters and in occupations where handling coal tar, coal tar pitches and soot occurs. Such studies clearly showed an elevated incidence of lung tumours following inhalation and skin tumours.
following chronic skin contact. However, it is not possible to assess with any confidence the contribution of BaP or any other individual PAH [2, 4, 9].

There were no studies available that investigated carcinogenicity in humans following oral exposure to BaP alone [3].

Overall, the International Agency for Research on Cancer (IARC) concluded that BaP was 'carcinogenic to humans' (Group 1), as there were limited human data but sufficient evidence of carcinogenicity in animals [10].

**Reproductive and developmental toxicity**

No studies could be identified in which reproductive or developmental effects in humans following exposure to BaP were reported.

**Animal and In-Vitro Data**

**Inhalation**

Rats exposed to BaP dust via inhalation (7.7 mg m\(^{-3}\), 2 hours per day, 5 days per week for 4 weeks) showed no treatment–related lesions in the lungs or nasal cavities. No dose-response relationship could be demonstrated as only one concentration of BaP was tested [2]. In the same study, kidney sections were also examined and no adverse effects were noted [3, 4]. Similarly, male hamsters did not show any adverse effects following exposure via inhalation to 9.8 mg m\(^{-3}\) or 44.8 mg m\(^{-3}\) BaP for 4.5 hours per day, five days per week for 16 weeks [2].

**Ingestion**

Few data on chronic oral toxicity of BaP in animals are available [5]. Daily oral administration of 120 mg kg\(^{-1}\) bw BaP to poor affinity Ah-receptor mice (DBA/2N) for one to four weeks caused deaths due to myelotoxicity, whereas high affinity mice (C57B1/6N) remained unaffected during the 6 month treatment. Hepatotoxicity, as well as effects on liver and kidney enzymes have also been reported at this concentration [2, 4].

Rats fed 1100 mg kg\(^{-1}\) day\(^{-1}\) BaP in the diet for more than 100 days showed a decreased growth rate [2].

**Dermal exposure**

BaP (16, 32 or 64 µg per application) was applied once a week for 29 weeks onto the skin of female mice. Dose-related epidermal thickening and a pronounced inflammatory response of the dermis, amongst other effects were reported in the first weeks of exposure in those administered the high dose, and subsequently in the lower dose groups [3].
BaP has consistently been shown to be positive in *in-vitro* assays for point mutations in *Salmonella* and for chromosome damage in mammalian cells, in the presence of an exogenous source of metabolic activation. Indeed it is often used as a positive control in such assays. Positive results have also been reported in a wide range of *in-vivo* studies in both somatic cells (e.g. bone marrow micronucleus test) and germ cells (dominant lethal assay and cytogenetics in spermatogonial cells) using both the inhalation and oral route [2].

In addition, several studies have reported genotoxicity of BaP following dermal exposure. A single topical application of BaP (0.5-500 μg per mouse) to hairless mice resulted in a significant increase in micronucleated keratinocytes. In addition, male mice treated with 20 μg topical BaP at 72 hour intervals exhibited increased DNA adduct formation in epidermis and lungs [3].

**Carcinogenicity**

Following a short-term exposure of two or more days, mice given BaP in the diet (33.3 mg kg⁻¹ bw day⁻¹) developed forestomach tumours. However, those given a lower dose of 13.3 mg kg⁻¹ bw day⁻¹ for up to seven days did not show any effects. Hamsters also had an increased incidence of tumours compared to control animals following a single dose of 100 mg kg⁻¹ BaP [3, 7].

Several studies have reported the increased incidence of tumours following a longer exposure to various doses of BaP by either inhalation or ingestion. Overall, inhalation of BaP caused lung tumours, whereas ingestion caused an increased incidence of tumours in various organs, including lung, forestomach, liver, oesophagus and tongue [10].

Following inhalation, a significant increase in lung tumours was reported in mice exposed to 0.05 or 0.09 μg m⁻³ BaP. Similarly, respiratory tract tumours were induced in a dose-dependent manner in the nasal cavity, pharynx, trachea, oesophagus and forestomach in hamsters exposed to 9.5 μg m⁻³ or 46.5 μg m⁻³ BaP for 109 weeks [3].

Forestomach and pulmonary tumours were reported in rats following administration of 67-100 mg kg⁻¹ bw day⁻¹ BaP by gavage or 33 mg kg⁻¹ bw day⁻¹ BaP given in the diet for 23-238 days. Forestomach tumours were also seen in mice given 33.3 mg kg⁻¹ bw day⁻¹ BaP in the diet for 30–197 days or 3 mg kg⁻¹ bw day⁻¹ by gavage for 98–197 days. In a two year study in mice, even the lowest dietary concentration of 0.75 mg kg⁻¹ bw day⁻¹ induced tumours in the forestomach [3, 4, 7, 10].

BaP (annual dose of 6-39 mg kg⁻¹ bw) was administered in the diet or by gavage to rats over their lifetime. Overall, there was a significant increase in the proportion of animals with tumours of the forestomach, oesophagus and larynx. In the dietary study, overall tumour incidence was increased only in the higher dose group, whereas in the gavage study, all test groups had a higher tumour incidence compared with controls [2, 4].

Many studies have been carried out in which BaP was applied to skin of mice. In such studies BaP has been demonstrated to be a potent local carcinogen. Topical application of BaP (up to 64 μg) for 29 weeks resulted in skin tumours which were initially benign but progressed to malignant carcinomas. No tumours were observed in mice lacking the Ah receptor. Administration of doses as low as 0.001% BaP to the skin throughout the lifetime; 12.5 μg BaP applied for 99 weeks; or 2 μg per mouse given two or three times per week for life caused malignant skin tumours [3, 4, 7, 10]. In contrast, when 0.05 mg BaP dissolved in 50 ml toluene was applied to shaved skin of mice twice a week for 6 months no tumours were observed [3, 10]. Skin tumours have also been reported in rats, rabbits and guinea pigs following dermal application [4].
IARC concluded that there is sufficient evidence that BaP is carcinogenic to experimental animals (Group 1) [9, 10].

**Reproductive and developmental toxicity**

Several studies have investigated the embryotoxicity of BaP after oral administration to pregnant mice. It has been shown to be embryotoxic in certain strains but not others, largely dependent on their Ah receptor status and the inducibility of cytochrome P450 enzymes [2].

Various strains of mice were given 120 mg kg⁻¹ bw day⁻¹ BaP in the diet on day 2-10 of gestation. Fetal malformations were seen in some strains but not others. This dose of BaP was reported to cause maternal toxicity [2-5].

Another study reported no adverse developmental effects in mice fed BaP in the diet (33.3-133.3 mg kg⁻¹ bw day⁻¹) during mating, gestation and parturition [3, 4]. Similarly, no reproductive or developmental toxicity was observed in male or female mice fed diets containing 0-1000 mg kg⁻¹ bw BaP over various time periods during mating, gestation, and lactation [5].

In a developmental toxicity/fertility study, CD1 mice were given 10, 40 or 160 mg kg⁻¹ bw day⁻¹ BaP by gavage on day 7-16 of pregnancy. Reduced survival of the pups was observed at the 2 highest dose levels, with reduced body weight reported at all doses. A marked effect on the fertility of the male offspring was seen, as pups exposed to the two highest doses were sterile, and a 20% decrease in fertility was seen a 10 mg kg⁻¹ [2-5].
References


This document will be reviewed not later than 3 years or sooner if substantive evidence becomes available.