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# POLYCYCLIC AROMATIC HYDROCARBONS (PAHs) AND OCCUPATIONAL HEALTH ISSUES

## Position Paper





**PREPARED BY**
**AIOH Exposure Standards Committee**
**June 2016**

**AUTHORISATION**
**This Position Paper has been prepared by the AIOH Exposure Standards Committee and authorised by AIOH Council.**
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## AUSTRALIAN INSTITUTE OF OCCUPATIONAL HYGIENISTS INC (AIOH)

The Australian Institute of Occupational Hygienists Inc. (AIOH) is the association that represents professional occupational hygienists in Australia. Occupational hygiene is the science and art of anticipation, recognition, evaluation and control of hazards in the workplace and the environment. Occupational hygienists specialise in the assessment and control of:

- Chemical hazards (including dusts such as silica, carcinogens such as arsenic, fibrous dusts such as asbestos, gases such as chlorine, irritants such as ammonia and organic vapours such as petroleum hydrocarbons);
- Physical hazards (heat and cold, noise, vibration, ionising radiation, lasers, microwave radiation, radiofrequency radiation, ultra-violet light, visible light); and
- Biological hazards (bacteria, endotoxins, fungi, viruses, zoonoses).

Therefore, the AIOH has a keen interest in the potential for workplace exposures to polycyclic aromatic hydrocarbons (PAHs), as its members are the professionals most likely to be asked to identify associated hazards and assess any exposure risks.

The Institute was formed in 1979 and incorporated in 1988. An elected governing Council, comprising the President, President Elect, Secretary, Treasurer and three Councillors, manages the affairs of the Institute. The AIOH is a member of the International Occupational Hygiene Association (IOHA).

The overall objective of the Institute is to help ensure that workplace health hazards are eliminated or controlled. It seeks to achieve this by:

- Promoting the profession of occupational hygiene in industry, government and the general community.
- Improving the practice of occupational hygiene and the knowledge, competence and standing of its practitioners.
- Providing a forum for the exchange of occupational hygiene information and ideas.
- Promoting the application of occupational hygiene principles to improve and maintain a safe and healthy working environment for all.
- Representing the profession nationally and internationally.

More information is available at our website – http://www.aioh.org.au.

## EXPOSURE STANDARDS COMMITTEE MISSION STATEMENT

The AIOH established the Exposure Standards Committee to provide expert guidance and comment to the exposure standards setting process at a State and National level and internationally, where appropriate, through development of AIOH Position Papers, AIOH guidance publications or comment on relevant Standards, Regulations and Codes of Practice. The Committee's remit is to confirm that the exposure standards numbers, and Standards and Codes of Practice, are changed for valid occupational hygiene and scientific reasons.

## STATEMENT OF POSITION REGARDING AIOH POSITION PAPERS

The AIOH is not a standards setting body. Through its Position Papers, the AIOH seeks to provide relevant information on substances of interest where there is uncertainty about existing Australian exposure standards. This is done primarily through a review of the existing published, peer-reviewed scientific literature but may include anecdotal evidence based on the practical experience of certified AIOH members. The Position Papers attempt to recommend a health-based exposure value that can be measured; that is, it is technically feasible to assess workplace exposures against the derived OEL. It does not consider economic or engineering feasibility. As far as reasonably possible, the AIOH formulates a recommendation on the level of exposure that the typical worker can experience without adverse health effects.

Any recommended exposure value should **not** be viewed as a fine line between safe and unsafe exposures. They also do not represent quantitative estimates of risk at different exposure levels or by different routes of exposure. Any recommended exposure value should be used as a guideline by professionals trained in the practice of occupational hygiene to assist in the control of health hazards.

## CONSULTATION WITH AIOH MEMBERS

AIOH activities are managed through committees drawn from hygienists nationally. This Position Paper has been prepared by the Exposure Standards Committee, with comments sought from AIOH members generally and active consultation with particular members selected for their known interest and/or expertise in this area. Various AIOH members were contributors in the development of this Position Paper. Key contributors included: Ross Di Corleto.

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## LIST OF ABBREVIATIONS AND ACRONYMS

| | |
|---|---|
| 8 h | Eight hour |
| ACGIH | American Conference of Governmental Industrial Hygienists |
| AIOH | Australian Institute of Occupational Hygienists |
| AS/NZS | Australian New Zealand Standard |
| AWES | Australian Work Exposures Study |
| B[a]P | Benzo(a)pyrene |
| BMGV | Biological Monitoring Guidance Value |
| BSF | Benzene soluble fraction |
| BSM | Benzene-soluble material |
| cr | Creatinine |
| CSF | Cyclohexane-soluble fraction |
| CTPV | Coal tar pitch volatiles |
| GM | Geometric mean |
| HPLC | High Pressure Liquid Chromatography |
| HSE | Health and Safety Executive (United Kingdom) |
| HSIS | Hazardous Substance Information System |
| IARC | International Agency for Research on Cancer |
| $J/cm^2$ | Joules per centimetre squared |
| IOHA | International Occupational Hygiene Association |
| $mg/m^3$ | milligrams ($10^{-3}$ gram) per cubic metre |
| $\mu g/m^3$ | microgram ($10^{-6}$ gram) per cubic metre |
| µmol/mol cr | micromoles ($10^{-6}$ mole) per mole of creatinine |
| NIOSH | National Institute for Occupational Safety and Health |
| nmol/mol cr | nanomoles ($10^{-9}$ mole) per mole of creatinine |
| OEL | Occupational exposure limit |
| OSHA | Occupational Safety and Health Administration |
| PAH | Polycyclic aromatic hydrocarbons |
| PAPR | Powered air purifying respirator |
| PPE | Personal protective equipment |
| PTFE | Polytetrafluoroethylene |
| SWA | Safe Work Australia |
| TEF | Toxic equivalence factors |
| TLV | Threshold limit value |
| TWA | Time weighted average |
| UK | United Kingdom |
| WES | Workplace Exposure Standard |
| WHO | World Health Organization |

## AIOH POSITION ON POLYCYCLIC AROMATIC HYDROCARBONS (PAHS) AND THEIR POTENTIAL FOR OCCUPATIONAL HEALTH ISSUES

### Key messages

- PAHs enter the body via breathing in of dust and fume or via skin contact, and can potentially cause bladder and lung cancer. They are also known to cause an abnormally high sensitivity of the skin to natural sunlight resulting in sunburn.
- While there may be a number of adverse effects as a result of over-exposure to PAHs individually and combined, of primary concern is the potential for cancer. Hence the focus of this document is directed at measurement of the key cancer-causing PAH compounds, the 4-6 ring structures.
- The AIOH believes that exposure may be adequately controlled by conventional means such as local exhaust ventilation and segregation of workers from areas of high concentration and/or potential skin contact. Respiratory protection may be required where conventional engineering controls are impractical.
- A standard to limit exposure to no more than 0.2 µg of benzo(a)pyrene (B[a]P) in each cubic metre of air, measured as an 8-hour time weighted average, is recommended for PAHs where exposure to the particulate fraction presents an exposure risk. Where the fraction of PAH exposure is unknown, exposure to the 16 priority PAHs should be assessed in the first instance.
- A guidance value of 4.0 µmol/mol cr 1-hydroxypyrene in urine should be considered as a way to demonstrate that controls are adequate for all routes of exposure across the broad group of PAHs for the 90$^{th}$ percentile of workers.

### Summary

This paper was compiled to give guidance on the assessment of occupational exposure to polycyclic aromatic hydrocarbons (PAHs) as a group. The current Safe Work Australia (SWA) workplace exposure standard (WES) and current international occupational exposure limits (OEL) are discussed and the possible health effects examined.

The occupational health risk associated with exposure of chimney sweeps to soot now known to contain PAHs is one of the earliest known cancers and was famously documented in 1775 by Sir Percival Pott. Since then the association between occupational exposure to PAHs and adverse health effects has been the subject of many studies.

PAHs are formed when natural or synthetic organic materials are burnt in the presence of less than ideal oxygen levels. They are derived from the elements of carbon and hydrogen. The major building block of their structure is the benzene ring, resulting in molecules containing fused-ring systems. Some well-known PAHs are naphthalene, benzo(a)pyrene (B[a]P), benzo(a)anthracene and dibenzo(a,h)anthracene. Human exposure occurs through a number of sources, including diet, tobacco smoking, pollution and occupational exposure.

They are generally measured via personal exposure monitoring by way of air. Biological monitoring can also be used in which the level of a metabolite of a specific PAH such as 1-hydroxypyrene from pyrene or 3-hydroxybenzo(a)pyrene from B[a]P is measured in urine.

Dermal effects of PAHs are enhanced by exposure to ultraviolet light. Due to exposure to pitch and tars, the skin can be prone to redness and inflammation, and photosensitivity (an abnormally high reactivity in the skin to ultraviolet radiation or natural sunlight). Skin lesions may develop on sun exposed areas and in some cases with progression to skin cancer.

B[a]P is classified by the *International Agency for Research on Cancer* (IARC) as a confirmed human carcinogen Category 1, and a number of other PAHs have been classified as Category 2A, i.e. as probably carcinogenic to humans. Studies have shown a statistically significant excess of bladder and lung cancer incidence for men. B[a]P is also classified as a mutagen and teratogen.

SWA has a WES for one specific PAH, naphthalene, but this position paper makes no recommendations in relation to any change in its current level. Discussion is principally directed at the exposure limit for coal tar pitch volatiles (CTPV) (as benzene solubles) of 0.2 mg/m$^3$. There are a number of issues associated with this exposure limit particularly in relation to its non-specific nature. A more appropriate approach would be to place the emphasis of exposure on the measurement of the levels of the 16 priority EPA PAHs and specifically B[a]P.

There is no formal biological limit for exposure to PAHs. However, the UK HSE has introduced a Biological Monitoring Guidance Value (BMGV) for biological monitoring for PAHs based on measurement of end-of-shift urinary 1-hydroxypyrene concentrations, while in France a guidance value based on the end of the last workday shift urinary 3-hydroxybenzo(a)pyrene concentration is recommended. The level of 4 µmol/mol creatinine (cr) and 0.4 nmol/mol cr has been recommended, respectively.

The most effective means of restricting PAH absorption is by controlling exposure. The hierarchy of controls must be utilised when determining the appropriate controls to be utilised; for example:

- provision of improved enclosure and ventilation to capture vapour, fume and particulate,
- good housekeeping,
- provision and use of change room facilities for scrupulous personal hygiene,
- no eating or smoking in PAH-contaminated areas,
- administrative controls (e.g. limits on overtime),
- education and
- use of protective clothing and appropriate respiratory protection.

Engineering controls must always be accompanied by a preventative maintenance program to ensure that the equipment's performance is maintained at the design specification and checked on a regular basis.

The AIOH suggests that the use of CTPVs as a WES for PAHs may not be a reliable measure of exposure to the carcinogenic PAH compounds due to the non-specific nature of the analysis. Also there are analytical issues that arise with measurements below 0.04 mg/m$^3$. Whilst the CTPV WES could still be used as an initial screening or action level, the AIOH recommends that it should be replaced by a B[a]P 8-hour TWA WES of 0.2 μg/m$^3$.

The information gaps for health aspects of exposure via skin and oral absorption would indicate that additional research is needed to confirm the long-term health impact via this route of entry. However, it is believed that biological monitoring is a viable and informative method of measuring total body burden associated with exposure to PAHs. As such the AIOH recommends that biological monitoring of 1-hydroxypyrene be used and exposures interpreted against a biological guidance value of 4.0 μmol/mol cr. Where this level is exceeded, control measures to reduce exposure should be reviewed.

## 1. What are Polycyclic Aromatic Hydrocarbons (PAHs)?

In 1775, Sir Percival Pott, an English surgeon, published the first detailed description of the adverse effects of exposure to smoke and resulting soot cancer in chimney-sweeps. This was attributed to soot penetrating the clothing of chimney sweeps and poor hygiene practices (Pott, 1775). Chimney soot is now known to contain high levels of polycyclic aromatic hydrocarbons (PAHs) (Doll, 1975). Exposure to PAHs is still associated with soot and fire related occupations (Baxter *et al*, 2014; Kirk & Logan, 2015) but there are now many other exposure scenarios identified.

PAHs are formed as a result of incomplete combustion or pyrolysis of natural or synthetic organic substances. They are derived from the elements of carbon and hydrogen.

The major building block of their structure is the benzene ring, resulting in molecules containing fused-ring systems. This structure includes the most basic two-ring naphthalene or four-ring pyrene and higher five-ring benzo(a)pyrene (B[a]P) and six-ring dibenzo(a,e)pyrene molecular compounds (See Attachment 1). PAHs with three or fewer aromatic ring structures exist predominately in the vapour phase with boiling points between 217 and 295°C. Those with four rings can exist in both the vapour and particulate phases. Where the compound comprises five or more rings with boiling points greater than 375°C, they mainly exist in the particulate phase (Cirla *et al*, 2007). The key carcinogenic PAH compounds of interest tend to be in the 4-6 ring structures (i.e. B[a]P). PAHs do not generally exist in the environment as discrete compounds, but are found as complex mixtures of many different concentrations and configurations. Whilst there have been more than 100 different PAHs identified (ATSDR, 1995) they are often considered in groups of 12 to 24 compounds in terms of convenience in assessment and analysis. The US EPA has listed 16 priority PAHs which are detailed in Attachment 1.

Due to their low vapour pressure, most PAHs entering the atmosphere as vapour will be adsorbed onto existing particles, condense on particles such as soot, or form very small particles themselves. Their presence in the environment is not restricted to the air, as they are often found in surface waters as a result of airborne fallout or industrial discharges and also in the soil. Human exposure occurs through a number of sources, including diet, tobacco smoking, pollution and occupational exposure. The route of entry to the body may be via inhalation, ingestion or through dermal absorption. Some well-known PAHs are naphthalene, benzo(a)pyrene, benzo(a)anthracene and dibenzo(a,h)anthracene.

## 2. How do we measure exposure to PAHs?

The concentration of PAH in the workplace air should be measured at regular intervals commensurate with the risk of exposure, and whenever there are changes in engineering controls, production methods or materials used. The nature and degree of exposure to PAHs and effectiveness of controls can be determined by monitoring either the workplace or the employee's personal exposure. Biological monitoring can also be used in which the level of a metabolite of a specific PAH such as 1-hydroxypyrene derived from pyrene or 3-hydroxybenzo(a)pyrene from B[a]P is measured in urine.

***In-air methods***

NIOSH methods 5506 and 5515 are the most often used air sampling methods for PAHs. Samples are collected by drawing a known amount of air through a cassette containing a PTFE filter in conjunction with a back-up sorbent tube. NIOSH method 5042 and OSHA 58 do not use the additional sorbent tube and are used more specifically for the benzene soluble fraction (BSF) and asphalt fume. In these two methods, the filter (glass fibre for OSHA 58) is analysed by extracting with benzene and gravimetrically determining the benzene-soluble material (BSM). Due to the carcinogenicity of benzene, toluene or cyclohexane may be used as the extractant (e.g. in UK, Europe and Australia). These methods assume that the PAHs in the particulate collected, measured as the BSF (or CSF now), are completely desorbed along with other hydrocarbons in the analysis process. This results in some shortcomings in that the true carcinogenic potential may be either over- or under-estimated, depending on the specific PAHs present in the mixture. There is also the additional complication that any other substances that are benzene soluble will also be measured.

For NIOSH method 5515 and 5506 the sample from both filter and sorbent tube are analysed by gas chromatography and high pressure liquid chromatography (HPLC) respectively. A mass spectrometer, fluorescence or ultraviolet detector is often used to determine the presence of selected PAHs.

Not all air sampling methods and sampling equipment are equivalent, hence allowance must be made when comparing results obtained using different sampling techniques. The collection of particulate (or vapour) alone can potentially provide a lower estimate of PAH exposure than

for collection of both particulate and vapour where both are present. A comprehensive review of the sampling and analysis methodologies was undertaken by Pandey *et al* in 2011.

There is a good relationship between B[a]P and total PAH concentrations in CTPVs, providing a useful tool for assessing exposure to a complex mixture such as PAHs (Farrant & Gariepy, 1998; Sanderson *et al*, 2005) in air. Note however that this can change as the pitch or combustible source varies. For example, asphalt fumes are generally lower in the higher 4–6 ring PAHs than CTPV.

Australian Standard AS/NZS 3580.16 "Determination of polycyclic aromatic hydrocarbons (PAH)", although for ambient air quality measurement, describes some of the issues associated with filter collection only, including the losses due to volatilisation of "particulate PAH".

Whilst the BSF (or CSF) analysis may be less specific and unable to give a true picture of the ratio of the different types of PAHs it could still be utilised as an initial screening method in preliminary analysis. If elevated CSF levels are encountered when dealing with new or unfamiliar potential PAH sources, some additional form of initial characterisation of the compounds present is also recommended. It is also important to note that whilst the focus of this position paper is on PAHs the presence of other substances should also be considered.

***Biological methods***

Biological monitoring is a more informative method of estimating an individual's internal exposure. This method usually involves the determination of a parent chemical, which may be representative of a mixture of chemicals (e.g. pyrene for PAHs), by assessing the level of a metabolite of that chemical in body fluids (blood or urine) or expired air.

The use of 1-hydroxypyrene as a biological marker was primarily developed by Jongeneelen through a number of studies and with some human validation carried out via therapeutically treated human subjects (Jongeneelen *et al*, 1985; Jongeneelen, Bos *et al*, 1988; Jongeneelen, Anzion *et al*, 1988; Jongeneelen, Scheepers *et al*, 1988; Tsai *et al*, 2002). Talaska *et al* (2014) showed that mean levels of 1-hydroxypyrene correlated well with carcinogen DNA adducts in coke oven workers showing an increased risk of bladder cancer, illustrating that 1-hydroxypyrene is a useful biomarker to show the effective dose to initiate the carcinogenic process. Ciarrocca *et al* (2014) concluded from a meta-analysis study that 1-hydroxypyrene appears to be a reliable biomarker for studying occupational exposure to PAHs from urban pollution, as long as environmental and behavioural factors are considered. Unwin *et al* (2006) also showed that 1-hydroxypyrene in urine correlated well ($r^2$ = 0.768) with airborne B[a]P.

The half-life for urinary excretion of 1-hydroxypyrene is biphasic at 6 to 35 hours (Jongeneelen *et al*, 1990) hence samples are best collected as per the American Conference of Governmental Industrial Hygienists guidelines (ACGIH, 2005), which recommend pre-shift and end of shift workweek post-shift urine samples. There may be some scenarios where pre-shift and post shift samples are collected each day to better characterise an exposure or task but this monitoring protocol can become costly when used for extended durations and is not always justified. The analytical method consists of analysis of urine samples via HPLC separation and detection with spectrofluorescence (Buckley & Lioy, 1992) with the results usually expressed as µmol/mol creatinine (cr).

More recently there have been studies where alternative biomarkers for exposure to PAHs have been utilised. Naphthalene has been proposed (Rappaport *et al*, 2004), utilising its biomarkers 1- and 2-hydroxynapthalene in urine as an alternative to 1-hydroxypyrene. Naphthalene (with two rings) is present almost entirely in the gaseous phase and would be a suitable marker for industries where the predominant exposure is airborne. However, where there is a mixture of dermal and airborne exposure, an alternative marker correlating better with the higher number ring compounds could be more suitable. It is important to note that the carcinogenic potency tends to be greatest among the 4 to 6- ring compounds.

Sobus *et al* (2009) concluded that levels of naphthalene and phenanthrene in urine reflect airborne exposures to these compounds and are promising surrogates for occupational exposures to PAH mixtures. Their study included PAH exposures to diesel exhausts (low PAH exposure), asphalt emissions (medium PAH exposure) and coke oven emissions (high PAH exposure) however, dermal exposure was not prevalent in this study. Seidel *et al* (2008) determined that metabolites of phenanthrene in urine were also reliable biomarkers for PAH exposure however, Rossbach *et al* (2007) found that these metabolites did not correlate well with B[a]P.

Another parent-metabolite pairing that has been under investigation is B[a]P and 3-hydroxybenzo(a)pyrene, which was the subject of a study carried out in a selection of industries in France (LaFontaine & Gendre, 2003). It was suggested that the monitoring of 3-hydroxybenzo(a)pyrene is a better biomarker to monitor exposure to PAHs as B[a]P is at present the only proclaimed IARC Category 1 carcinogen in PAH mixtures.

Further work by Förster *et al* (2008) demonstrated that 3-hydroxybenzo(a)pyrene was a specific and sensitive biomarker for determining the internal exposure of workers to B[a]P in different industries. They noted that the procedure for analysing 3-hydroxybenzo(a)pyrene is "complex" (Barbeau *et al*, 2014) and this was a potential limitation particularly in Australia where the method at this point in time is not available. While it appears logical to concentrate on B[a]P, the most carcinogenic component of the PAH mixture, the analytical capability needed to achieve such a low detection limit for 3-hydroxybenzo(*a*)pyrene is limiting. Given also the analytical precision needed to separate background levels from exposed workers, it would make the interpretation of the test results challenging. This is an evolving area and this approach may in the future be more widely adopted but at present it is being used by only a few laboratories with the capability to achieve the precision and detection limit required (Jongeneelen, 2014).

The UK HSE has introduced a Biological Monitoring Guidance Value (BMGV) for biological monitoring for PAHs based on measurement of end-of-shift urinary 1-hydroxypyrene concentrations. A level of 4 µmol/mol cr was recommended, as this value represents the 90th percentile of measurements taken from industries deemed to have good control (Pedersen, 2003; Armstrong *et al*, 2003).

Within Australia the Workcover NSW Biological Occupational Exposure Limit Committee has recommended a level of 1µg 1-hydroxypyrene/L urine (1.4 µmol/mol creatinine (cr)) which has not at this stage been formally adopted and is under review.

## 3. Hazards and risks associated with PAHs

***Single or short-term exposure***

*Effects on Human Skin / Eyes* - Dermal effects of PAHs are enhanced by exposure to ultraviolet light. Due to exposure to pitch and tars, the skin can be prone to redness and inflammation, photosensitivity (an abnormally high reactivity in the skin to ultraviolet radiation or natural sunlight) and skin lesions on sun exposed areas and in some cases with progression to skin cancer. Cases of skin inflammation have been identified in industry and are sometimes referred to as "pitch burn". It is a form of phototoxicity that results in delayed erythema and skin pain. The presence of pitch burn within an industry is often an indication that there are issues with exposure to PAHs, hence the reporting of such instances should be monitored and could possibly be used as an indication of control effectiveness.

PAHs are irritating to the eyes and can cause photosensitivity and volatile fumes can affect the eyes. Symptoms will vary with the amount of ultraviolet radiation, type and amount of photosensitiser, skin type and age and sex of the person exposed. Figure 1 illustrates the effect of pitch on the skin with ultra violet light exposure in Joules/cm$^2$ (J/cm$^2$).

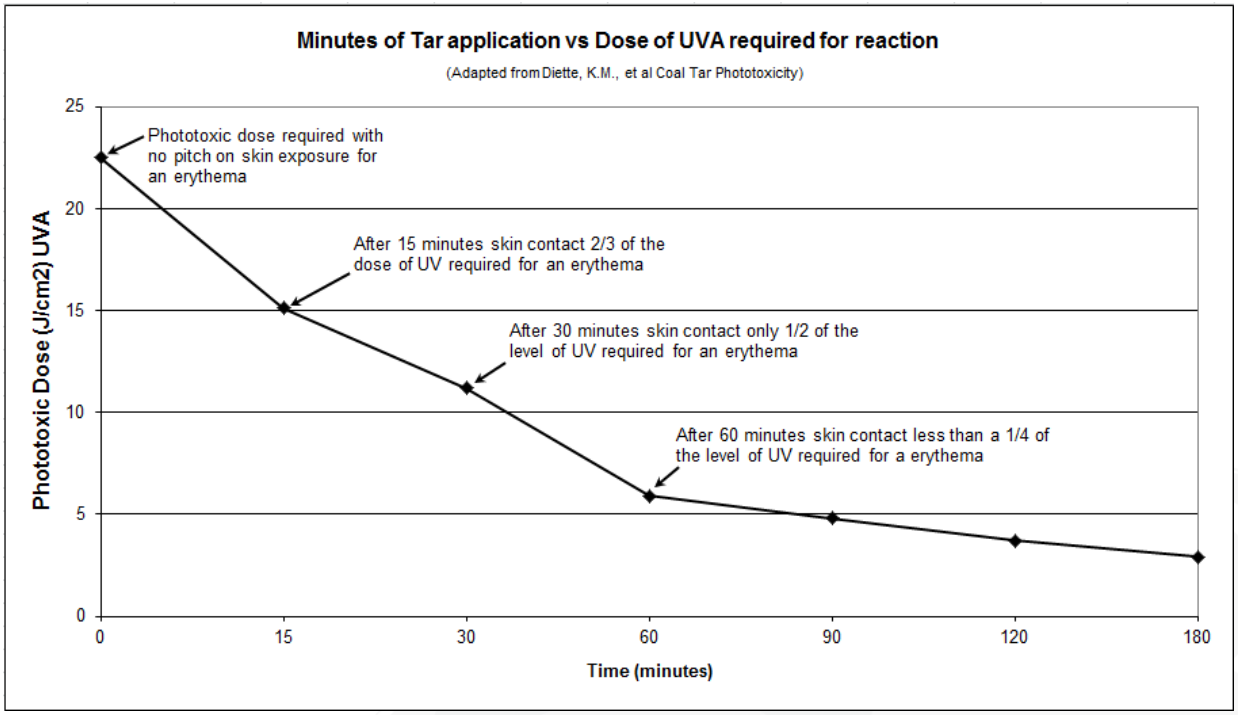


Figure 1: Effect of pitch containing PAHs contact time on skin. (Adapted from Diette *et al*, 1983)

***Repeated or long-term exposure***

*Effects on Inhalation / Ingestion* - Cough, chronic bronchitis and haematuria have been noted. There is sufficient evidence that PAHs are carcinogenic to experimental animals.

The carcinogenicity of 17 PAH compounds has been confirmed (IARC, 1983; IARC, 2012; Straif *et al*, 2005). Many other compounds are known to be mutagenic. Data has indicated a relationship between the site of tumour development and the route of administration. PAHs can induce tumours at other sites as well, since tissues such as the skin can metabolize PAHs to their ultimate metabolites, and metabolites formed in the liver can reach various sites via the blood stream. Theoretical explanations for the carcinogenic action of PAHs are considered in detail in the WHO (1998) Environmental Health Criteria Publication number 202.

Increased lung tumour rates linked to exposure were found in coke-oven workers, asphalt workers and workers in Söderberg potrooms of aluminium reduction plants (WHO, 1998).

Aluminium production and CTPVs have been classified by the IARC in 1984 as being Category 1 carcinogens; i.e. confirmed human carcinogens. B[a]P, benzo(a)anthracene and dibenzo(a,h)anthracene are listed in the Safe Work Australia (2015a) Hazardous Substance Information System (HSIS) and are, as with IARC, classified as carcinogens. B[a]P is also classified as a mutagen and teratogen.

A cancer incidence study undertaken in Quebec (Gibbs & Sevigny, 2007a; Gibbs & Lebrèche, 2014) concluded that a statistically significant excess of cancer incidence was found for men. Bladder cancer incidence was strongly linked to BSM and B[a]P exposure. An associated mortality study (Gibbs & Sevigny, 2007b) showed that it has diminished in post 1950 hired workers but there is still an elevated incidence of this cancer in post 1950 cohorts.

A study in Australian prebake aluminium smelters found no overall excess of mortality or cancer, but incident mesothelioma and kidney cancer risks were elevated (Sim *et al*, 2009). In another such study, an association between smelter exposures and respiratory cancer was found, but bladder cancer was not associated with B[a]P or BSM exposure (Friesen *et al*, 2009). The lack of excess risk for lung or bladder cancer or deaths from respiratory disease was thought to be related to the different level and pattern of exposure between Söderberg and prebake smelters.

A recent meta-analysis (Wagner *et al*, 2015) has also suggested that there is "a robust positive association between PAH and larynx cancer".

Occupational exposures to B[a]P-containing mixtures have been associated with a series of cancers:

- coke production: lung;
- coal gasification: lung, bladder;
- paving and roofing: lung;
- coal tar distillation: skin;
- soots: lung, oesophagus, haematolymphatic system, skin;
- aluminium smelting: lung, bladder;
- tobacco smoking: lung, lip, oral cavity, pharynx, oesophagus, larynx, bladder.

## 4. Sources of PAH and potential for exposure in Australia

PAHs are ubiquitous in the environment. The most important sources of occupational and non-occupational PAHs are as follows (WHO, 1998):

- production of aluminium (mainly from anodes)
- metal production (particularly iron, and steel)
- foundries in the binding agents used in moulding sand
- domestic and residential heating
- road paving and asphalt works
- motor vehicle traffic and diesel exhaust
- bushfires and structural fires
- coal-fired power plants
- incineration of refuse
- tobacco smoking
- medicinal use
- cooking processes (food)
- coke ovens
- spontaneous combustion of coal
- remediation of contaminated land, specifically former manufactured gas plants

PAH exposure may occur through the three main routes of uptake by the body: inhalation, ingestion and skin absorption (ATSDR, 1995), with air inhalation and skin usually being the two key routes. The main route of exposure will be dependent on the environment, process, work practices and, in some cases, the level and type of PPE worn (Di Corleto, 2010). The route of exposure to PAHs can play a major role in their fate within the body.

Unwin *et al* (2006), in a cross-industry occupational hygiene survey in the UK, found that 8-hour (8 h) TWA levels of total PAHs in air ranged from 0.4 to 1,913 µg/m$^3$ with a geometric mean (GM) of 15.8 µg/m$^3$. The profile of PAHs was dominated by naphthalene, the most volatile 2-ring PAH. Airborne B[a]P correlated well with levels of carcinogenic 4–6 ring PAHs and was an effective marker of exposure for all industries where significant particle bound PAH levels were found and, in particular, for CTPV exposure. The 8 h TWA levels of B[a]P ranged from <0.01 to 6.2 µg/m$^3$ with a GM of 0.036 µg/m$^3$; 90% were <0.75 µg/m$^3$ and 95% were <2.0 µg/m$^3$.

A study of anode plant workers in an aluminium reduction plant in the Netherlands (van Rooij *et al*, 1992) found the total skin contamination in exposed workers was estimated to be more than three times higher than the intake via the respiratory tract. In a study of PAH exposure among asphalt paving workers, McClean *et al* (2004) estimated that dermal exposure was eight times the impact of inhalation exposure. Similar results were reported by Borak *et al* (2002) in their study of creosote facility workers.

In 2014 the Australian Work Exposures Study (AWES) was conducted which investigated work-related exposures among Australian workers and included PAH exposures as part of the study. It did not specifically focus on industries suspected of high exposures to PAHs; however, it revealed that approximately 6% of workers who participated in the study were probably exposed to PAHs when performing common tasks. These included tasks such as burning waste, repairing equipment powered by combustion engines like mowers or similar equipment, cooking, fighting fires, and fire overhaul and clean-up (Driscoll, 2014). Most with 'probable exposure' were male (81%) and 40% of the total worked in the agriculture industry. The main control measure was respiratory protection equipment and only about 40% of AWES respondents performing firefighting, back burning and welding appeared to be using appropriate respiratory protection. As a result, about two thirds of exposed workers were assessed as having high or medium task-based exposures to PAHs.

The AWES project provided only qualitative information on current exposures to PAHs based on job tasks. It was recommended that quantitative measures of PAH exposure in the workplace may be of use to validate the data collected in AWES and to improve understanding of the absolute levels of exposure to PAHs.

## 5. Available controls

The control of PAHs aligns with those controls used in the management of general particulate and vapours and should follow the hierarchy of controls. There is also skin exposure that will need to be considered as this is a known route of exposure for PAHs.

- Efficient ventilation – Areas with activities likely to generate harmful vapour, particulate or fume concentrations should be enclosed if practicable, and well ventilated (exhaust ventilation). An alternative to source containment and ventilation is worker enclosure; e.g. provide cabs on vehicles or enclosed control rooms with filtered / pressurised / air-conditioned air supply. Where ventilation systems are installed, these systems should be maintained in good working order and should be operated in the correct manner to provide optimum protection from airborne contaminant exposure. Fume scrubbing systems are sometimes employed to capture PAHs in fumes for removal.
- Good housekeeping - The maintenance of a high standard of housekeeping will minimise exposure to particulate and potential contact with the skin. Methods of wet cleaning can be used in some circumstances where downstream contamination is not an issue. Surface or equipment contamination may be checked by using a wipe test.
- The provision of regular education and training is particularly important as employees need to understand the different routes of exposure and the impact on how they approach their work. The conduct of workplace and employee in-air monitoring, and health monitoring of employees may also be considered a part of the controls for mitigating exposures.
- Administrative controls - A reduction of level and duration of exposure of employees to exposure may be achieved by work organisation and limits on overtime. The principle of periodic rotation of employees both through and in areas with potentially harmful dust/fume/vapours exposures is an acceptable practice; this should be restricted however, only to plant trained employees.
- Where practicable a clean/dirty change-room process similar to that employed in the lead industry should be utilised. Employees should be encouraged to shower or wash any skin contaminated with PAHs as soon as possible to reduce photo-sensitization and potential skin inflammation from UV exposure. Work clothing should not be taken home where there exists a risk of cross contamination with domestic laundry.
- Good personal hygiene must be practiced in areas where potential exposure may occur and no eating, drinking and smoking allowed in these areas.
- Inspection and maintenance routines for engineering controls, and periodic formal review of the practicality of engineering controls, are essential administrative controls. The periodicity of these activities will vary according to the health risk associated with the hazard being controlled and the functionality of the control being used.
- Provision and sensible use of personal protective equipment (PPE) – Half-face respirators and/or powered air purifying respirator (PAPR) helmets fitted with P1 particulate filters, when worn correctly, will normally provide adequate protection against dust exposures. Full-face respirators fitted with a P2 particulate filter can also provide additional protection of the eyes and facial skin and would be preferred in areas of elevated fume or particulate. The use of organic vapour cartridge respirators should be utilised in situations where elevated levels of PAH vapour may be encountered. An air-line respirator may be required for some scenarios of high level exposure. In all cases the respiratory protection program should follow the requirements of AS/NZS 1715, "*Selection, use and maintenance of respiratory protection devices*".
- Minimisation of skin contact time or elimination should be strongly promoted via the prompt washing of contact areas and personal protective equipment. This may require the use of disposable coveralls and gloves. PPE should be used as a last resort, where other control measures have been unsuccessful.
- The use of water based barrier creams prior to exposure can help to reduce skin absorption (Prior, 1996).

It is important to emphasize that there is a need to establish, embed and continue to maintain control systems and to put in place methodology for ongoing auditing and measurement of their effectiveness. These systems require the input of suitably qualified and appropriately experienced occupational hygienists.

## 6. Current applicable legislation and standards

SWA has a specific WES for only one of the lower numbered ring PAHs (naphthalene) as well as a TWA exposure limit for CTPVs (as BSF) of 0.2 $mg/m^3$.

Use of this TWA to discern PAH is useful as a screening exposure standard but has shortcomings as a specific TWA because of limitations in the analytical method as outlined in the 'In-air methods' section above. It should also be again noted at this point that the use of methods involving benzene as a solvent are no longer recommended due to the health implications associated with its use. In more recent times, cyclohexane has been used as an alternative in this method, and while it is a suitable substitute there are concerns in relation to accuracy and precision (Unwin *et al*, 2006). With the exposure standards being driven towards the 0.05 $mg/m^3$ level, the limit of detection for this method is also now under question, particularly as sites utilise action levels which are often 20-50% of the OEL.

It is known that B[a]P is a hazardous component of most PAH mixtures and has been classified as a Category 1 carcinogen, around which studies and measures (i.e. toxic equivalence factors - TEF) have been developed (Willes *et al*, 1992; Petry *et al*, 1996). Accordingly, the emphasis of exposure on the measurement of the levels of B[a]P is seen to be important and of priority. There are other scenarios where elevated levels of lower ring number PAHs may be present. In such circumstances the monitoring, assessment and control in line with specific WESs such as naphthalene may be more appropriate.

SWA have not assigned an exposure standard for B[a]P, with the statement that "For a few substances, usually the more potent probable and established human carcinogens, it is not currently possible to assign an appropriate exposure standard. For these substances, exposure should be controlled to the lowest practicable level" (Safe Work Australia, 2015b). It has been noted that occupational studies have found no effects for average PAH workplace exposures below 0.25 to 2.5 μg/m$^3$ of B[a]P (Tremblay *et al*, 1995) and this would be a potential level at which to target an exposure limit.

There is also a requirement under Safe Work Australia that baseline health monitoring be undertaken for those workers starting work in a process associated with potential PAH exposure. Further detail may be found at http://www.safeworkaustralia.gov.au/sites/SWA/about/Publications/Documents/803/Polycyclic-Aromatic-Hydrocarbons-(PAH).pdf.

## 7. OELs from other jurisdictions, regulators and organisations

UK HSE - There is no Health and Safety Commission-approved OEL for PAHs. In June 2003 the reason cited was: "no maximum exposure limit (MEL) is likely to be appropriate for the whole of industry exposed to PAHs… because of the low numbers of workers exposed at relatively high levels and because the benefits are minimal, the MEL appears to have little benefit" (Pederson, 2003).

The current ACGIH recommended occupational exposure limit for CTPVs is listed as 0.2 mg/m$^3$ as a threshold limit value/time-weighted average (TLV©-TWA) for CTPVs as benzene soluble fraction. This "is defined operationally in terms of the benzene (or cyclohexane) extractable fraction of total airborne particulate as collected by a personal sampler. If the extractable material contains detectable quantities of benz[a]anthracene, benzo[b]fluoranthene, chrysene, anthracene, benzo[a]pyrene, phenanthrene, acridine, or pyrene, then the TLV-TWA for that material is 0.2 mg/m$^3$ total aerosol" (ACGIH, 2013).

## 8. AIOH RECOMMENDATION

The AIOH suggests that CTPV is not a reliable measure of exposure to the carcinogenic PAH compounds due to the non-specific nature of the analysis. Also there are analytical issues that arise with measurements below 0.04 mg/m$^3$. The AIOH recommends that it should be replaced by measuring B[a]P in comparison to a WES of 0.2 μg/m$^3$, measured as an 8-hour time weighted average, where exposure to the PAH particulate fraction presents an exposure risk. Where the fraction of PAH exposure is unknown, exposure to the 16 priority PAHs should be assessed in the first instance.

The information gaps for health aspects of exposure via skin absorption indicate that additional research that is undertaken to confirm the long-term health impact via this route of entry. However, it is believed that biological monitoring is a viable and informative method of measuring total body burden associated with exposure to PAHs. As such the AIOH recommends that biological monitoring of 1-hydroxypyrene be used and exposures interpreted against a biological guidance value of 4.0 μmol/mol cr, as a way to demonstrate that controls are adequate for all routes of exposure across the broad group of PAHs for the 90th percentile of workers.

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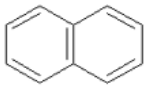
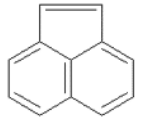
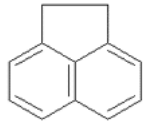
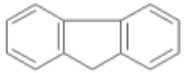
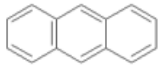
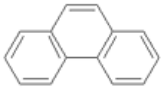
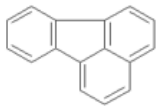
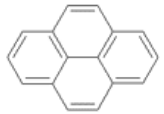
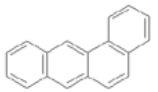
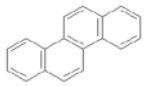
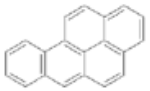
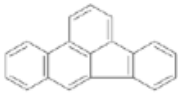
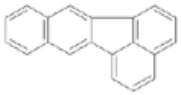
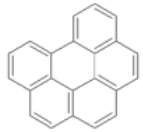
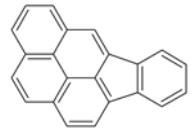
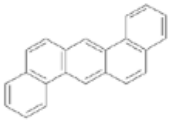
**Attachment 1**

| | Compound | CAS Number | Molar Mass (g/mol) | Number of Fused Aromatic Rings | Primary Exposure Phase | IARC Group | Structure |
|---|---|---|---|---|---|---|---|
| 1 | Naphthalene | 91-20-3 | 128 | 2 | Volatile | 2B (2002) |  |
| 2 | Acenaphthylene | 208-96-8 | 152 | 3 | Volatile | - |  |
| 3 | Acenaphthene | 83-32-9 | 154 | 3 | Volatile | 3 (2010) |  |
| 4 | Fluorene | 86-73-7 | 166 | 3 | Volatile | 3 (2010) |  |
| 5 | Anthracene | 120-12-7 | 178 | 3 | Volatile | - |  |
| 6 | Phenanthrene | 85-01-8 | 178 | 3 | Volatile | 3 (2010) |  |
| 7 | Fluoranthene | 206-44-0 | 202 | 4 | Volatile | 3 (2010) |  |

| | Compound | CAS Number | Molar Mass (g/mol) | Number of Fused Aromatic Rings | Primary Exposure Phase | IARC Group | Structure |
|---|---|---|---|---|---|---|---|
| 8 | Pyrene | 129-00-0 | 202 | 4 | Semi-volatile | 3 (2010) |  |
| 9 | Benz[*a*]anthracene | 56-55-3 | 228 | 4 | Semi-volatile | 2B (2010) |  |
| 10 | Chrysene | 218-01-9 | 228 | 4 | Semi-volatile | 2B (2010) |  |
| 11 | Benzo[*a*]pyrene | 50-32-8 | 252 | 5 | Particulate | 1 (2012) |  |
| 12 | Benzo[*b*]fluoranthene | 205-99-2 | 252 | 5 | Particulate | 2B (2010) |  |
| 13 | Benzo[*k*]fluoranthene | 207-08-9 | 252 | 5 | Particulate | 2B (2010) |  |
| 14 | Benzo[*ghi*]perylene | 191-24-2 | 276 | 6 | Particulate | 3 (2010) |  |

| | Compound | CAS Number | Molar Mass (g/mol) | Number of Fused Aromatic Rings | Primary Exposure Phase | IARC Group | Structure |
|---|---|---|---|---|---|---|---|
| 15 | Indeno[123-cd]pyrene | 193-39-5 | 276 | 6 | Particulate | 2B (2010) |  |
| 16 | Dibenz[*ah*]anthracene | 53-70-3 | 278 | 5 | Particulate | 2A (2010) |  |

IARC Group Classification: Group 1: Carcinogenic to humans; Group 2A: Probably carcinogenic to humans; Group 2B: Possibly carcinogenic to humans; Group 3: Not classifiable as to carcinogenicity to humans