Ann. Occup. Hyg., Vol. 50, No. 4, pp. 395–403, 2006
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doi:10.1093/annhyg/mel010

An Assessment of Occupational Exposure to Polycyclic Aromatic Hydrocarbons in the UK

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Received 28 November 2005; in final form 24 January 2006; published online 21 March 2006

A cross-industry occupational hygiene survey was commissioned by the Health and Safety Executive (HSE) to determine the levels of polycyclic aromatic hydrocarbon (PAH) exposure in UK industry and to determine if one or more target analytes were suitable as markers for assessing total exposure to PAHs. There were no broadly applicable UK exposure standards for assessing total exposure to PAHs. Until 1993 a guidance value for assessing exposure in coke ovens only, where PAH exposure is known to be the highest, was based on gravimetric analysis of cyclohexane-soluble material. Biological monitoring based on urinary 1-hydroxypyrene (1-OHP) is widely reported to be an effective indicator of exposure by both dermal and inhalation routes but there was no UK guidance value. The survey involved an occupational hygiene study of 25 sites using both airborne monitoring of a total of 17 individual PAHs and biological monitoring. The results showed 8 h TWA levels of total PAH in air ranged from 0.4 to 1912.6 μg m^{-3} with a GM of 15.8 μg m^{-3}. The profile of PAHs was dominated by naphthalene, the most volatile 2-ring PAH. Airborne benzo(a)pyrene (BaP) correlated well (r^2 = 0.971) 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 BaP ranged from <0.01 to 6.21 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}. Two hundred and eighteen urine samples collected from different workers at the end of shift and 213 samples collected pre-shift next day were analysed for 1-OHP. Levels of 1-OHP in end-of-shift samples were generally higher than those in pre-shift-next-day samples and showed a good correlation (r^2 = 0.768) to airborne BaP levels if samples from workers using respiratory protection or with significant dermal exposure were excluded. Urinary 1-OHP in end-of-shift samples ranged from the limit of detection (0.5 μmol mol^{-1} creatinine) to 60 μmol mol^{-1} creatinine with a mean of 2.49 μmol mol^{-1} and a 90th percentile value of 6.7 μmol mol^{-1} creatinine. The highest 1-OHP levels were found in samples from workers impregnating timber with creosote where exposure was dominated by naphthalene. If the 11 samples from these workers were excluded from the dataset, the 90% value for end-of-shift urine samples was 4 μmol mol^{-1} creatinine (n = 207) and this value has since been adopted by the HSE as a biological monitoring benchmark value.

Keywords: benzo(a)pyrene; biological monitoring; polycyclic aromatic hydrocarbons; urinary 1-hydroxypyrene

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a class of compounds found in crude oils, mineral oils, bitumens and tars (Gelboin and Ts'o, 1978; IARC 1985, 1987). They are also formed during the incomplete combustion of fossil fuels and oil products and as a result are widely distributed in the workplace and the environment. Industries where exposure to PAHs is likely to occur include coke ovens and coal tar use,

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iron and steel works, aluminium works, foundries, carbon electrode and carbon black manufacture, asphalt manufacture and use, and many others. In these industries inhalation of volatile compounds or of particles with adsorbed PAHs and dermal absorption of mixtures containing PAHs may be important routes of exposure. The number of workers potentially exposed may run into tens of thousands and there is concern about the potential for some PAHs to cause cancer. Occupational exposure to PAHs should be controlled to levels as low as reasonably practical; however, there are few occupational exposure limits for assessing exposure in the workplace.

In the case of the destructive distillation of coal in coke ovens where the potential for exposure is high owing to the release of coal tar pitch volatiles (CTPV), an industry-specific approach was adopted in the past to monitor exposure. A non-specific measurement method, based on the mass of benzene or cyclohexane soluble material extracted from total inhalable aerosol collected on a filter, was used to assess exposure. The American Conference of Governmental Industrial Hygienists (ACGIH®) has an 8 h threshold limit value of 0.2 mg/m^3 for CTPVs as benzene soluble aerosol (ACGIH®, 2005). Until 1993 the UK had a similar approach using cyclohexane instead of benzene. There were several major deficiencies with this measurement procedure; it exhibited poor accuracy and precision at the 8 h TWA guidance value of 1.4 mg m^{-3} (included in EH40 from 1989 to 1993, HSE, 1993); the measure was a surrogate, which was not linked to a specific toxicological hazard, e.g. carcinogenic PAHs; there was evidence that the relationship between cyclohexane soluble material and total PAH was not consistent for all CTPV sites or in other industries where coal tar pitch products were used; no assessment of exposure arising through skin absorption was made. Extensive work (Scobbie and Dabill, 1993) showed that the measurement of individual PAHs was the way forward to assess exposure to CTPV but there are few occupational exposure limits for individual PAHs. The Deutsche Forschungsgemeinschaft (German TRK) has 8 h TWA airborne limit values for BaP of 5 μg m^{-3} during production and loading of pencil pitch and near coke ovens, and 2 μg m^{-3} in other workplaces (Deutsche Forschungsgemeinschaft, 2004). A pragmatic approach such as this, or alternatively by quantifying a limited set of compounds, for example the US-EPA-16 (Table 1), is a way to simplify the measurement procedure. The IARC classification of the EPA-16 (IARC, 1987) raised the question as to whether this is the most appropriate set of compounds for a toxicologically based assessment of risk as not all compounds indicate clear carcinogenic or mutagenic activity. A group of nine potentially carcinogenic compounds was utilized by the Health and Safety Executive (HSE) (Table 1) to represent a measure of total exposure to carcinogenic PAHs (Department of Health, 1994). These were largely the 4–6 ring compounds present in the EPA-16 set. Groups of such compounds have commonly been adopted in the past as markers for total exposure to carcinogenic PAHs (Williams *et al*., 1986; Grimmer *et al*., 1987; Heinrich *et al*., 1994; Sivak *et al*.,1997; Binet *et al*., 2002). The most extensively studied single PAH as a risk indicator for total PAH exposure is BaP (Armstrong *et al*., 2004). It is released from a large number of industrial sources and has been known as a predominant carcinogenic compound in coal tar for some time (Kennaway, 1955; Phillips, 1983; Yamagiwa *et al*., 1977).

PAHs may be absorbed through the skin but some industries rely on the use of respiratory protective

Table 1. Industry types surveyed and airborne PAHs of interest

Compound	Industry type (site number in brackets)
Naphthalene[a]	High temperature coke oven (11 and 14)[d]
Acenaphthalene[a]	Low temperature coke oven[d] (2)
Acenaphthene[a]	High temperature tar distillation[d] (3)
Fluorene[a]	Low temperature tar distillation[d] (4)
Phenanthrene[a]	Aluminium smelting[d] (1)
Anthracene[a]	Clay Targets[d] (6)
Fluoranthene[a]	Pipeline coatings and wraps[d] (7)
Pyrene[a]	Coal fired power station[d] (15)
Benzo(a)anthracene[a–c]	Timber impregnation[d] (5)
Chrysene[a,b]	Electrical carbon[d] (24)
Benzo(b)fluoranthene[a–c]	Integrated oil refinery[e] (9)
Benzo(k)fluoranthene[a–c]	Petroleum tar distillation[e] (13)
Benzo(j)fluoranthene[a–#]	Petroleum coking[e] (10)
Benzo(a)pyrene[a–c]	Bitumen refinery[e] (8)
Indeno(1, 2, 3-c,d)pyrene[a–c]	Asphalt roofing[e] (22)
Benzo(g, h, i)perylene[a, b]	Asphalt road dressing (surfacing)[e] (25)
Dibenz(a, h)anthracene[a–c]	Road construction (base)[e] (23)
Anthanthrene[b]	Carbon black manufacture[f] (17)
Cyclopenta(c, d)pyrene[#]	Motor tyre manufacture[f] (12 and 16)
	Iron foundry[g] (18 and 19)
	Fish smokehouse[h] (20 and 21)

[a]US EPA-16 compounds.
[b]Compounds used in survey as surrogate for total 4–6 ring compound exposure, ([#] not measured).
[c]Classified by IARC (IARC, 1987) either Group 2A—probably carcinogenic to humans or Group 2B—possibly carcinogenic to humans.
[d]CTPV.
[e]Oil and Bitumen.
[f]Rubber.
[g]Foundry.
[h]Wood smoke. Site identification numbers are in brackets.

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equipment (RPE) to control exposure (Quinlan *et al*., 1995). Therefore, assessment of airborne exposure alone may not always give a complete picture of exposure. In such cases, biological monitoring has an important role to play but the complexity of the PAH mixtures, their low concentrations in biological media and their metabolism makes biological monitoring of the components of the mixture very difficult. The options for biological monitoring of PAH exposure include measurement of PAH adducts to DNA, haemoglobin, serum proteins and PAH metabolites in urine (Angerer *et al*., 1997). The HSE's preference for biological monitoring is that it should be based on non-invasive sample collection if possible (i.e. urine).

One of the major components of many PAH mixtures is pyrene, and although it is not one of the carcinogenic compounds, its metabolite 1-hydroxypyrene (1-OHP) is stable, relatively easy to detect in urine and has been proposed as a biomarker of exposure to PAHs (Jongeneelen *et al*., 1986). Subsequently, urinary 1-OHP has been extensively used as a biomarker of exposure (Jongeneelen, 1992; Wu *et al*., 1998; McClean *et al*., 2004; Ming-Tsang *et al*., 2004; Tsai *et al*., 2004).

A cross-industry occupational hygiene survey was commissioned by the HSE to determine the levels of PAH exposure in UK industry and to determine if one or more of the nine compounds were suitable as a marker for assessing total airborne exposure to PAHs. The survey included biological monitoring and a range of industries in order to give a broad consideration of occupational exposure to PAHs. For consistency and comparative purposes the full EPA-16 set was also measured.

MATERIALS AND METHODS

Study design

The industrial sites selected in this survey, listed in Table 1, involved PAHs originating from coal tar pitch, oil and bitumen, rubber fume, foundries and wood smoke. Based on historical data, the sites were expected to exhibit quantifiable levels of exposure to PAH and were typically visited on Wednesday or Thursday. The study was approved by the HSE's Research Ethics Committee.

Personal airborne sampling

Inhalable dust sampling was carried out using an IOM sampler fitted with a pre-extracted, pre-weighed 25 mm glass-fibre filter. An XAD-2 sorbent tube (SKC-26-30-04) was fitted in line directly after the sampling head to collect vapour phase components as well as those volatilized from the filter during sampling. Samples were taken at a flow rate of 2 l min^{-1} for a minimum period of 5 h. Up to 12 samples were collected at each site and included all the operators present or a full cross-section of the typical activities being carried out. The filter and sorbent tube were extracted separately by sonication for 30 min in 2 ml dichloromethane. The extracts were spiked with internal standards d_8-naphthalene, d_{10}-anthracene, d_{12}-chrysene and d_{12}-perylene prior to analysis.

Analysis of air samples

Analysis of the sample extracts was carried out using an Agilent Technologies 5890 gas chromatograph fitted with a 5971 mass selective detector. A HP5 column (30 m × 0.25 mm, film thickness 0.25 μm) operated under the temperature programme: 50°C for 1 min; 5°C min^{-1} to 280°C; hold at 280°C for 10 min. was used to separate the individual components. The helium carrier gas linear velocity was 40 cm s^{-1}. The detector was operated in the selected-ion monitoring mode for nominal molecular ions (m/z) 128,136, 152,153, 166, 178, 188, 202, 228, 240, 252, 264, 276 and 278. The results were compared with a 7-point calibration curve and fitted by linear regression analysis. Internally prepared quality assurance samples were run after every 10 unknown samples. The limit of detection for personal air samples (S:N 3:1), based on a 700 l air sample, ranged from 6 to 100 ng per compound.

Biological monitoring

Each worker participating in the survey provided urine samples in clean 30 ml plastic bottles at end of shift and pre-shift next day. The end-of-shift samples were brought back to the laboratory and pre-shift-next-day samples were returned by post. The samples were stored below −18°C prior to analysis.

Urine sample analysis was based on the method of Jongeneelen *et al*. (1986). Briefly, duplicate urine samples (200 μl) were incubated with glucurase (300 μl in 0.1 M acetate buffer pH 5) at 37°C for 4 h to hydrolyse the glucuronide and sulphate conjugates and 100 μl injected onto a 5 μm ODS column (100 mm × 4.6 mm) with a mobile phase of methanol:water (75:25) at a flow rate of 1 ml min^{-1}. 1-OHP was separated from other urine constituents and detected by fluorescence with an excitation wavelength of 244 nm and an emission wavelength of 388 nm. The results were compared with a 7-point calibration curve over the range 0–100 nmol l^{-1} and fitted by linear regression analysis. Internally prepared quality assurance samples were run after every five unknown samples and the laboratory participated in an external quality assurance scheme (Schaller *et al*., 2001). The analytical method has a detection limit, based on 3× background noise, of 5 nmol l^{-1} and an intra-assay and inter-assay coefficient of variation of 5 and 12%, respectively, at a concentration of 40 nmol l^{-1}. For statistical analysis 1-OHP results below the detection limit were treated

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as 0.25 μmol 1-OHP mol^{-1} creatinine (based on half the detection limit for 1-OHP and a nominal creatinine concentration of 10 mmol l^{-1}).

Creatinine

Creatinine was determined in all urine samples using an automated alkaline picrate method (Jaffe, 1886; Bonsnes and Taussky, 1945). Internal quality assurance samples were run after every 10 unknown samples; external proficiency samples for creatinine from the Finnish Institute of Occupational Health were run at regular intervals.

RESULTS AND DISCUSSION

Airborne PAH measurements

A summary of 219 personal exposures for the 25 sites in Table 2 demonstrates that total 8 h TWA levels (sum of all 17 components) ranged from 0.08 to 1912.6 μg m^3 (mean 93.62 μg m^{-3}; median 15.24 μg m^{-3}). The 8 h TWA levels of the carcinogenic 4–6 ring PAHs ranged from 0.01 to 35.93 μg m^{-3} (mean 2.2 μg m^{-3}; median 0.12 μg m^{-3}) and the highest values were predominantly found in CTPV industries. The highest concentrations as expected were found in the high-temperature and low-temperature coke ovens. However, some elevated levels were observed for specific activities in petroleum tar distillation, petroleum coke, asphalt roofing, road construction, carbon black and foundries. A weak correlation was observed between total PAH and total carcinogenic (4–6 ring) PAHs (r^2 = <<0.1).

Naphthalene is the most volatile PAH and dominates the PAH profile on all sites. It typically represented 50–90% of the total PAHs and consequently there was a strong correlation (r^2 = 0.97) between airborne naphthalene and total PAH (Fig. 1). This correlation would be insensitive to small but potentially toxicologically significant changes in concentration of the carcinogenic 4–6 ring compounds and as a consequence total PAH is an inappropriate measure for industries such

Table 2. Summary of personal exposure [8 h TWA (μg m^{-3})]

Site	Industry		Total PAH		Total 4–6 ring PAH		BaP		1-OHP μmol mol^{-1}	
		n	Mean	Range	Mean	Range	Mean	Range	Mean	Range
11	Coke oven	11	79.17	8.80–184.55	16.36	1.17–35.93	2.14	0.13–6.21	1.85	0.25–5.4
14	Coke oven	13	70.66	9.93–294.63	5.69	0.21–29.09	0.79	0.02–4.08	2.1	0.25–7.1
2	Coke oven (low T)	13	49.87	5.88–131.64	6.87	0.01–19.44	1.13	0.01–2.91	2.63	0.41–6.9
3	Tar distillation (high T)	12	278.82	51.9–1130.21	0.725	0.11–4.54	0.06	0.01–0.32	2.6	0.78–5.7
4	Tar distillation (low T)	8	12.17	5.20–38.59	0.01	0.01	0.01	0.01	0.36	0.25–1.1
1	Aluminium (green carbon)	9	60.88	10.45–138.38	0.31	0.01–0.85	0.03	0.03–0.10	0.72	0.25–2.6
6	Clay targets	8	24.67	13.39–38.69	5.44	1.91–12.84	0.82	0.26–2.02	11.3	3.9–11.3
7	Pipeline coating and wrap	11	263.64	73.34–758.22	3.78	0.4–18.30	0.32	0.04–1.65	10.6	1.7–21.3
15	Coal fired power station	12	1.37	0.40–2.11	0.20	0.04–0.79	0.02	<0.01–0.10	0.25	0.25
5	Timber impregnation	11	835.06	29.93–1912.6	0.05	0.01–0.11	0.01	0.01–0.011	16.0	1.4–60
24	Electrical carbon	1	12.79	—	1.72	—	0.24	—	0.25	0.25
9	Integrated oil refinery	12	5.24	0.77–20.34	0.01	0.01–0.03	0.01	0.01	0.25	0.25
13	Petroleum tar distillation	8	68.94	15.21–280.03	0.13	0.04–0.35	0.02	0.01–0.04	0.88	0.25–3.8
10	Petroleum coke	9	5.83	0.74–16.80	2.58	0.10–12.26	0.374	0.01–1.78	0.48	0.25–1.2
8	Bitumen refinery	12	2.38	0.08–7.01	0.02	0.01–0.06	0.01	0.01	0.25	0.25
22	Asphalt roofing	3	1.51	1.23–1.94	0.34	0.23–0.47	0.02	0.01–0.02	0.25	0.25
25	Asphalt road surfacing	7	3.21	1.83–4.63	0.01	0.01	0.01	0.01	0.25	0.25
23	Road construction (base)	5	9.56	3.83–17.60	0.38	0.07–0.72	0.01	0.01	0.25	0.25
17	Carbon black	11	10.70	1.98–69.09	0.52	0.03–4.49	0.05	0.01–0.41	0.63	0.25–0.31
16	Motor tyre	12	3.22	1.82–6.55	0.06	0.02–0.11	0.01	0.01–0.02	0.32	0.25–0.8
12	Motor tyre	11	3.22	1.63–4.54	0.03	0.01–0.11	0.01	0.01	0.25	0.25
18	Foundry	11	65.75	26.97–120.13	0.211	0.03–0.33	0.02	0.01–0.05	0.29	0.25–0.66
19	Foundry	7	21.12	8.88–47.38	0.09	0.05–0.13	0.01	0.01–0.02	0.25	0.25
20	Fish smokehouse	2	2.31	2.06–2.55	0.07	0.06–0.07	0.01	0.01	0.25	0.25
21	Fish smokehouse	1	—	17.24	—	1.55	—	0.22	0.25	0.25

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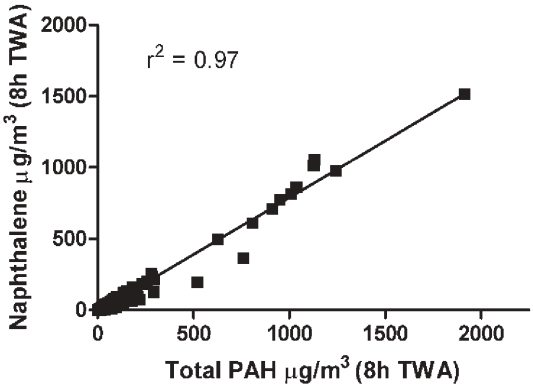

Fig. 1. Relationship between total PAH and naphthalene ($y = 0.797x - 7.49$).

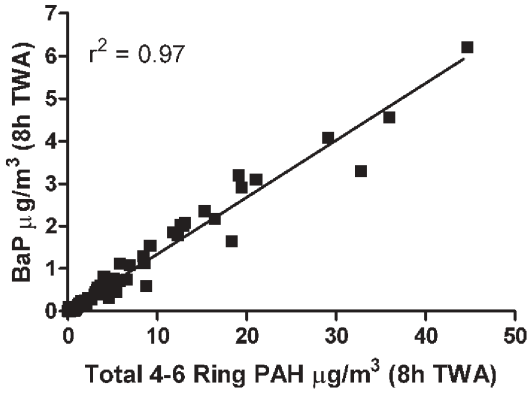

Fig. 2. Relationship between BaP and total 4–6 ring PAH ($y = 0.134x + 0.00429$).

as coke ovens where exposure to the carcinogenic compounds could be significant. Fig. 2 shows the strong correlation between BaP and 4–6 ring PAHs ($r^2 = 0.97$), which is not observed between BaP and total PAH ($r^2 = <<0.1$). The close similarity in volatility and physical and chemical properties between the 4–6 ring compounds means that any fluctuations due to condensation, absorption and evaporation will affect BaP and the 4–6 ring compounds similarly. This supports BaP as a candidate marker PAH and occupational hygiene tool where exposures to carcinogenic PAHs are significant.

The Deutsche Forschungsgemeinschaft (German TRK) has an airborne occupational exposure limit for BaP of 5 μg m^{-3} during production and loading of pencil pitch and near coke ovens, and 2 μg m^{-3} in other workplaces. The data from the study show that for BaP, 90% of all values would fall below 0.75 μg m^{-3} and 95% below 2.0 μg m^{-3}. Therefore utilization of this marker may be effective as a tool to ensure exposures are reduced as low as reasonably practicable based on BaP measurements.

Table 3. Summary of biological monitoring data (1-OHP μmol mol^{-1} creatinine)

Parameter	All sites	All sites except timber impregnation
n	218	207
Mean	2.50	1.78
SD	6.0	3.8
GM	0.70	0.61
GSD	1.67	1.29
90th Percentile	6.7	4.0

Urinary 1-OHP measurements

Two hundred and eighteen urine samples were collected from workers at the end of their shift and 213 samples were collected pre-shift next day and analysed for 1-OHP. The strategy behind the two sampling times was to investigate the best collection time either in terms of highest concentration of metabolite found in urine or correlation with air monitoring or occupational hygiene data. The biological monitoring data was not normally distributed and a Wilcoxan matched pairs test showed a statistically significant ($P < 0.0001$) difference between end-of-shift and pre-shift-next-day samples with end-of-shift samples generally with higher values. Because of this, a preliminary look at relationships between air and biological monitoring data showed no better correlation with pre-shift-next-day samples, the rest of the analysis of biological monitoring data concentrates on end-of-shift samples. A Wilcoxan signed rank test shows a statistically significant difference ($P < 0.0001$) between the 1-OHP results in end-of-shift urine samples from smokers (median 0.825; $n = 74$) and non-smokers (median 0.25; $n = 143$) and is in line with the contribution to urinary 1-OHP expected from smoking (Jongeneelen, 1994, 2001; Gundell and Angerer *et al*., 1999). The biological monitoring data is summarized in Table 3 and Fig. 3. Additional data are in Table 4 in Unwin *et al*. (2006). Three sites, timber impregnation (5), clay target manufacture (6) and pipeline coatings and wraps (7), have the highest exposures. The highest urinary 1-OHP value (60 μmol mol^{-1}) was found at the timber impregnation site where, in addition to significant potential for dermal exposure, the airborne PAH profile was dominated by naphthalene with relatively little BaP (mean 0.01 μg m^{-3}). Biological monitoring would clearly be useful at this site but would be better based on urinary naphthol rather than 1-OHP. Urinary 1-OHP levels found in coke oven workers in this study are in the range 0.25–7 μmol mol^{-1} and are in close agreement with previously published studies (Jongeneelen *et al*., 1990; Buchet *et al*., 1992; Van Rooij *et al*., 1993; Gundel and Angerer, 1999). Some of the workers with urinary 1-OHP levels below the detection limit were road construction workers exposed to

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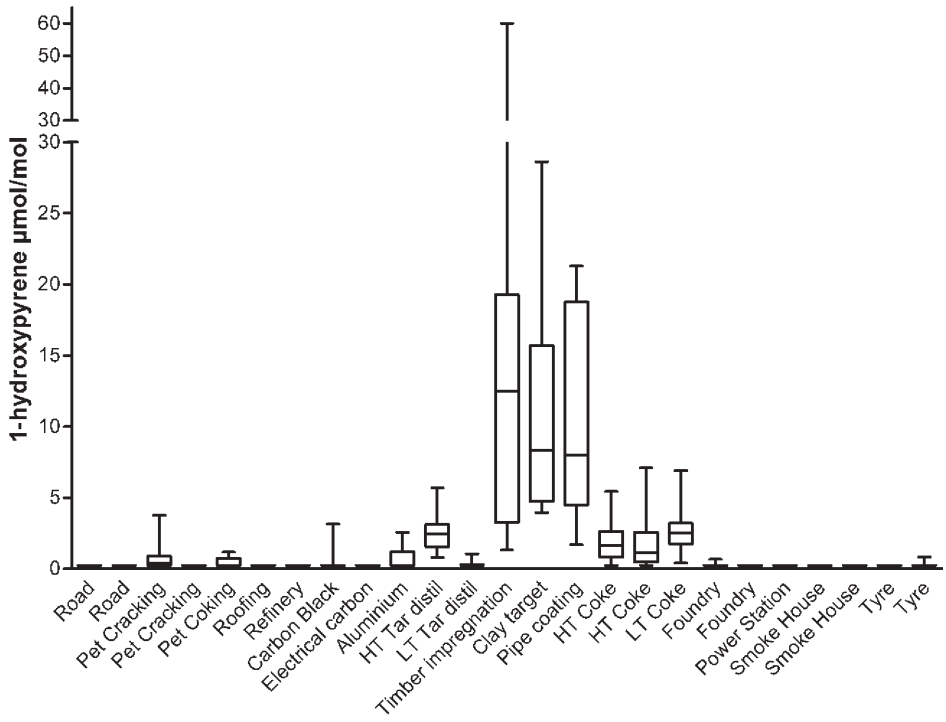

Fig. 3. Summary of urinary 1-OHP levels found at each site. The bar is the median, the box denotes the 25th and 75th percentiles and the whiskers denote the range.

oil-based bitumen and were in contrast to similar workers using coal-tar-based products with urinary 1-OHP levels in the range 1–45 µmol mol^{-1} (Gundel *et al*., 2000). The difference in urinary 1-OHP levels between workers doing similar jobs but with oil rather than coal-tar-based products was also seen with clay target manufacture. Levels in this study with workers using coal tar ranged from 4 to 29 µmol mol^{-1} and contrast with the lower levels found by Lafontaine *et al*. (2000) in workers using oil-based bitumen with urinary 1-OHP levels in the range 2–11 µmol mol^{-1}.

The levels of 1-OHP found in urine of carbon black workers in this study ranged from less than the limit of detection to 3.1 µmol mol^{-1} with a mean of 0.6 µmol mol^{-1}; this compares well with those found by Tsai *et al*. (2000) in carbon black workers (range 0.5–2.1 with a mean of 1.0 µmol mol^{-1}).

The relationship between airborne BaP and urinary 1-OHP initially seems poor ($r^2 \leqslant 0.1$), without taking into account personal protective equipment and occupational hygiene practice. Many of the results show extensive exposure to airborne BaP but with low levels of urinary 1-OHP. This is probably due to workers using RPE. There are also a number of points with significant levels of urinary 1-OHP but with little airborne BaP and this is probably due to dermal absorption. When workers with known use of RPE and sites with significant dermal exposure were removed, the number of data points reduced to 41 but the correlation between airborne BaP and urinary 1-OHP improved (r^2 = 0.77, Fig. 4). Using this relationship and the German TRK value for BaP of 2 µg m^{-3} would predict a urinary 1-OHP value of 23 µmol mol^{-1} creatinine. This compares well with Gundel and Angerer (1999) who found a slightly worse correlation (r = 0.56, n = 16) between BaP and 1-OHP but which gave a urinary 1-OHP value of 24 µmol mol^{-1} creatinine after exposure to 2 µg m^{-3} BaP.

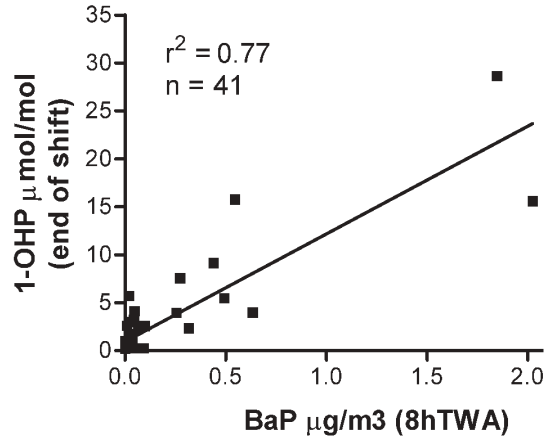

Fig. 4. Relationship between BaP and 1-OHP ($y = 11.1x + 1.13$)—on sites without respiratory protection or significant dermal exposure.

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Excluding the timber impregnation (Site 5) where biological monitoring may be better based on urinary naphthol, the 207 samples collected post-shift ranged from below the detection limit to 29 μmol mol^{-1} with a mean and median of 1.8 and 0.25 μmol mol^{-1}, respectively. Ninety percent of the values were <4 μmol mol^{-1} and this figure was adopted by HSE as a UK biological monitoring benchmark value for exposure to PAHs (HSE, 2005). This control-based approach to proposing biological monitoring guidance values differs from the more usual approach where the guidance value is based on the level of a biomarker found after inhalation at the airborne occupational exposure limit or a direct relationship to the absence of ill health. For comparison, the levels of 1-OHP found in urine of people not occupationally exposed to PAHs are generally <0.25 and 0.5 μmol mol^{-1} in non-smokers and smokers, respectively (Jongeneelen 1994; Gundell and Angerer, 1999). The 95th percentiles for background levels of urinary 1-OHP are 0.76 and 0.24 μmol mol^{-1} for smokers and non-smokers, respectively, and these levels could be viewed as guidance values marking the boundary between background exposures and occupational exposure to PAHs (Jongeneelen, 2001). This approach is the basis of the BEI® proposed by ACGIH® for monitoring PAH exposure based on urinary 1-OHP (ACGIH®, 2005). Because the composition of PAH mixtures is very variable ACGIH® also proposed adjusting the 'benchmark' value for 1-OHP of 1 μg l^{-1} (~0.5 μmol 1-OHP/mol creatinine) if the ratio of airborne pyrene:BaP differs significantly from the default value of 2.5 found in air samples around coke ovens and coal tar pitch processes. In the study reported here 86 samples (39%) had 1-OHP values above the BEI. Only 63 (29%) of the measured airborne exposures (mostly from coke ovens and CTPV exposures) were sufficiently high to give a reliable pyrene:BaP ratio. The Pyrene to BaP ratio ranged from 0.2 to 38 with a median ratio of 2.2 close to the ACGIH® default value of 2.5. Adjustment of the BEI took the values observed in this study over the adjusted BEI® in two cases and below it in three cases.

At the upper end of the occupational exposure spectrum are urinary 1-OHP levels associated with exposure to BaP at the German TRK level of 2 μg m^{-3}. Based on the work of Angerer *et al*. (1997) and Gundel and Angerer (1999) with workers making graphite electrodes and the work reported here in a range of industries the 1-OHP level in urine associated with inhalation of 2 μg m^{-3} BaP would be ~20 μmol mol^{-1}. Lower levels of 1-OHP have been associated with inhalation of 2 μg m^{-3} BaP in other studies. Van Rooij *et al*. (1993) reported a value of 3.2 μmol mol^{-1}, Jongeneelen *et al*. (1990) reported a value of 2.3 μmol mol^{-1} and Pan *et al*. (1998) reported a value of 9.1 μmol mol^{-1} in studies of coke oven workers. The differences may be explained in part by differences in sample collection times, variable ratios of pyrene to BaP and different proportions of dermal and inhalation exposures. However, in view of the carcinogenic nature of many PAHs it would be prudent to keep exposure as low as reasonably practical and the work reported here shows 90% of urine samples from workers in a wide range of industries have levels of 1-OHP <4 μmol mol^{-1} and that in many industries levels well below 4 μmol mol^{-1} and much closer to background levels are achievable.

Using the correlation from the observed relationship between BaP and urinary 1-OHP the value of 4 μmol mol^{-1} would correspond to an airborne BaP level of 0.26 μg m^{-3} BaP.

CONCLUSIONS

In an occupational hygiene study of 25 workplaces with exposure to PAHs, 219 personal airborne samples were collected and showed the levels of nine carcinogenic PAHs ranged from 0.01 to 35.93 μg m^{-3} 8 h TWA with a median of 0.12 μg m^{-3} 8 h TWA. Levels of total PAH (the sum of 17 compounds) were found to be dominated by naphthalene and were insensitive to changing levels of nine carcinogenic PAHs. Measurement of BaP, a single PAH, was well correlated to levels of the nine carcinogenic PAHs and is a marker of exposure and risk. In this study the levels of BaP ranged from <0.01 to 6.21 μg m^{-3} 8 h TWA with a median of 0.01 μg m^{-3} 8 h TWA. Highest BaP exposures were seen in coke ovens where control of exposure relied on RPE.

A control strategy based on an airborne exposure limit for BaP was not subsequently adopted by HSE for several reasons. Although the occupational hygiene data collected here suggests that BaP would be a useful tool as a marker for total PAH exposure in some industries, particularly those involving CTPV, it is a poor predictive marker for exposure to gas-phase compounds (2–4 ring compounds), which represent by far the largest group of most highly exposed workers. Control of exposure for coke ovens can best be achieved in a cost effective way with the use of RPE and specific guidance rather than enforce engineering solutions which, given current coke oven technology, may not be achievable. For these reasons the HSE thought it inappropriate to recommend an airborne occupational exposure standard based on BaP (HSE, 2003a, b).

Urinary 1-OHP showed a good correlation with airborne BaP if dermal exposure and use of RPE were taken into account. Urinary 1-OHP levels ranged from less than the detection limit (0.5 μmol mol^{-1}) to 60 μmol mol^{-1} with a mean of 2.5 μmol mol^{-1} and a median less than the detection limit.

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Highest levels of urinary 1-OHP were found in timber impregnators using creosote and workers using coal tar where there was both dermal and inhalation exposure. Excluding timber impregnation, 90% of urinary 1-OHP levels from 207 workers in 24 workplaces were <4 μmol mol^{-1} and this has been adopted as a biological monitoring guidance value in the UK. Using the observed correlation between urinary 1-OHP and airborne BaP, a level of 1-OHP of 4 μmol mol^{-1} is roughly equivalent to an airborne BaP level of 0.26 μg m^{-3}. Biological monitoring for PAHs may be particularly useful where there is potential for dermal exposure or where control relies on RPE; however, a combination of biological and airborne measurements will be required to determine the effectiveness of control systems more fully.

Acknowledgements—The authors would like to thank the HSE for funding this work and Stuart Whiteley and Bob Guiver for their contribution to the collection of the measurement data. Funding to pay the Open Access publication charges for this article was provided by the Health and Safety Executive.

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