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Urinary naphthalene and phenanthrene as biomarkers of occupational exposure to polycyclic aromatic hydrocarbons

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ABSTRACT

Objectives: The study investigated the utility of unmetabolised naphthalene (Nap) and phenanthrene (Phe) in urine as surrogates for exposures to mixtures of polycyclic aromatic hydrocarbons (PAHs).

Methods: The report included workers exposed to diesel exhausts (low PAH exposure level, n = 39) as well as those exposed to emissions from asphalt (medium PAH exposure level, n = 26) and coke ovens (high PAH exposure level, n = 28). Levels of Nap and Phe were measured in urine from each subject using head space-solid phase microextraction and gas chromatography–mass spectrometry. Published levels of airborne Nap, Phe and other PAHs in the coke-producing and aluminium industries were also investigated.

Results: In post-shift urine, the highest estimated geometric mean concentrations of Nap and Phe were observed in coke-oven workers (Nap: 2490 ng/l; Phe: 975 ng/l), followed by asphalt workers (Nap: 71.5 ng/l; Phe: 54.3 ng/l), and by diesel-exposed workers (Nap: 17.7 ng/l; Phe: 3.60 ng/l). After subtracting logged background levels of Nap and Phe from the logged post-shift levels of these PAHs in urine, the resulting values (referred to as ln(adjNap) and ln(adjPhe), respectively) were significantly correlated, suggesting a common exposure source in each case. Surprisingly, multiple linear regression analysis of ln(adjNap) on ln(adjPhe) showed no significant effect of the source of exposure (cope ovens, asphalt and diesel exhaust) and further suggested that the ratio of urinary Nap/Phe (in natural scale) decreased with increasing exposure levels. These results were corroborated with published data for airborne Nap and Phe in the coke-producing and aluminium industries. The published air measurements also indicated that Nap and Phe levels were proportional to the levels of all combined PAHs in those industries.

Conclusion: Levels of Nap and Phe in urine reflect airborne exposures to these compounds and are promising surrogates for occupational exposures to PAH mixtures.

Polycyclic aromatic hydrocarbons (PAHs) comprise a class of chemicals composed of two or more fused aromatic rings. Since PAHs are produced by the incomplete combustion of organic matter, including petroleum, coal and other carbonaceous materials (for example, wood, tobacco products, food products), they are ubiquitous contaminants of the human environment. Humans are exposed to PAHs via inhalation, ingestion and dermal contact. Moderate to high-level PAH exposures stemming from coke ovens, aluminium production and asphalt use have been associated with cancers of the lung, skin and bladder.1 Exposure to diesel exhausts, containing lower levels of PAHs, has been associated with increased lung cancer risks.2-5

The quantitative assessment of PAH exposure has been complicated by the large number of individual compounds in a given mixture and by the presence of PAHs in both the gas phase (two- and three-ring compounds) and the particulate phase (four- to six-ring compounds). While certain particulate-phase PAHs (notably benzo(a)pyrene) have been classified as known or probable human carcinogens,6 air concentrations of these four to six ring PAHs tend to be very low and difficult to measure. Recently, attention has focused upon the more abundant gas-phase PAHs, notably naphthalene (Nap, two rings) and phenanthrene (Phe, three rings), as possible surrogates for PAH exposure.7-8 Naphthalene is typically the most abundant PAH measured from a given source9 and air levels of Nap tend to be highly correlated with the sum of all measured PAH levels (hereafter “total PAHs”) in workplaces.10 Since Nap is a known carcinogen of the lung in rodents,11-12 it is important to characterise human exposure to Nap. Phenanthrene is also present at high concentrations in PAH emissions and, while not classified as a carcinogen, is the smallest PAH to contain a four-ring PAH and has been associated with increased lung cancer risks.13-18

Small amounts of Nap and Phe are eliminated unchanged in the urine.19 Here we report levels of unmetabolised Nap and Phe in urine from workers exposed to three sources of PAHs that had been classified a priori as having low, medium and high levels of PAHs, namely, diesel-exhausts, asphalt emissions and coke-oven emissions, respectively.14 We show that urinary levels of Nap and Phe in these workers followed the expected low, medium and high designations of the sources and that they were highly correlated, suggesting a common source of exposure to Nap and Phe in each group. We further compare results from measurements of urinary Nap and Phe with published data representing air concentrations of Nap, Phe and total PAHs in the coke-producing and aluminium-producing industries.15-18

METHODS

Chemicals and supplies

Naphthalene (99%), phenanthrene (99.5%), (2H8)naphthalene (98%), (2H10)phenanthrene (Phe) in urine as surrogates for exposures to mixtures of polycyclic aromatic hydrocarbons (PAHs).
measurements (including area and personal samples) for up to plant, three measurements (grouped personal samples from 24 personal samples) for up to 39 PAHs in a coke production. The data include 32 measurements (area, breathing-zone and reported along with various other PAHs in several workplaces. Analysis, we consider air levels of both Nap and Phe that were investigated. Urine samples were also obtained from 26 asphalt workers in road paving crews in the northeastern US. This group included 20 paving workers who applied hot mix asphalt to roads and six milling workers who removed old asphalt from roads. Urine samples were collected from asphalt workers on either two (milling workers) or three consecutive days (paving workers) both before and after work shifts, starting at the beginning of the work week. Finally, urine samples were obtained from 39 diesel-exposed workers who performed various tasks in trucking terminals throughout the US. (While workers in trucking terminals may have experienced PAH exposures from ambient air, diesel exhaust is believed to be their primary source of PAH exposure and we refer to this group as “diesel-exposed workers” for simplicity.) This group included 27 loading-dock workers who drove propane forklifts and loaded trailers, eight truck-repair-shop workers involved with truck maintenance and refuelling activities and four office workers who had only background exposure to diesel exhausts. Pairs of urine samples were collected from each diesel-exposed worker before and after a work shift. The smoking status of all workers was obtained by questionnaires.

Air levels of Nap, Phe and total PAHs were derived from published data reporting exposures in the coke-producing and aluminium industries. The data were previously summarised by Rappaport et al., who focused upon Nap exposures. For this analysis, we consider air levels of both Nap and Phe that were reported along with various other PAHs in several workplaces. The data include 52 measurements (area, breathing-zone and personal samples) for up to 39 PAHs in a coke production plant, three measurements (grouped personal samples from 24 individuals) for 16 PAHs in a coke-production plant, 28 measurements (including area and personal samples) for up to 36 PAHs in an aluminium reduction plant and six measurements (midrange of five personal samples from 6 workers) for 26 PAHs in a carbon anode plant. All subjects included in this study provided informed consent to participate according to protocols approved by ethics committees at the Harvard University School of Public Health (Boston, Massachusetts, USA) and the Institute for Occupational Medicine (Beijing, China).

Analysis of urinary naphthalene and phenanthrene

Urine samples from asphalt and diesel-exposed workers were analysed for Nap and Phe as previously described, with minor modifications.

Samples were stored at either –20°C or –80°C before analysis. After thawing, 0.7-ml portions were transferred into 2-ml crimp top vials containing 0.5 g of NaCl. Urine samples were spiked with 1.0 μl of an internal standard mixture containing (H3)Nap and (H10)Phe in methanol, to give a final concentration of 0.5 μg/l of urine. Samples were immediately capped and stored at –20°C for up to 24 hours before analysis. Before use, vials, caps and NaCl were conditioned at 160°C to remove background Nap and Phe.

Headspace solid-phase microextraction (HS-SPME) was performed to extract Nap and Phe from urine using a CombiPal autosampler (CTC Analytics, Zwingen, Switzerland). Before analysis, samples were brought to room temperature and were incubated at 55°C for 3 minutes. Analytes were sampled from urine headspace using a PDMS fibre (10 mm, 100-μm film thickness). Adsorption and desorption times were 30 minutes and 20 minutes, respectively. Levels of Nap and Phe were measured with a model 6890N gas chromatograph (GC) coupled to a model 5973N mass spectrometer (MS) (Agilent, Palo Alto, CA, USA). The MS was operated with electron impact ionisation at an ionisation voltage of 70 eV. The MS transfer line was maintained at 280°C, the source temperature at 200°C and the quadrupole at 100°C. A DB-1 (J&W Scientific Inc, Folsom, CA, USA) fused silica capillary column (60 m, 0.25-mm internal diameter, 0.25-μm film thickness) was used with helium as the carrier gas. A 0.75-mm internal diameter SPME injection sleeve was used in the injector port, with the temperature maintained at 250°C. The GC oven was held at 75°C for 8 minutes and then ramped at 5°C/min to 260°C, where it was held for 10 minutes. Ions selected for analysis included m/z 128 (Nap), m/z 156 [(H9)Nap], m/z 178 (Phe) and m/z 188 [(H10)Phe]. Quantitation was based on response ratios of the analytes to the corresponding internal standards [(H3)Nap or (H10)Phe]. Standard curves were prepared with pooled urine from human volunteers, which had been spiked with Nap and Phe at concentrations of 0.40 ng/l, 2.0 ng/l, 10 ng/l, 25 ng/l, 50 ng/l, 75 ng/l and 100 ng/l and the same levels of internal standards used for experimental samples. The estimated coefficients of variation for Nap and Phe were 0.25 and 0.26, respectively and the estimated limit of detection (LOD) was 0.40 ng/l for each analyte. Two observations, with analyte levels below the LOD, were assigned levels of LOD/2 for statistical analyses.

Statistical analyses

Statistical analyses were performed after (natural) logarithmic transformation of urinary levels, to remove heteroscedasticity and satisfy normality assumptions, using SAS statistical software v. 9.1. A p value <0.05 was considered significant (two-tailed test). For asphalt workers, subject-specific means of logged pre-shift and post-shift Nap and Phe levels (from urine samples collected on two or three consecutive days) were used for statistical analyses. General linear models were used with dummy variables to test for effects on analyte levels of the source of exposure (diesel exhausts, asphalt emissions and coke-oven emissions), the job category (coke-top, coke-side, coke-control, asphalt-paver, asphalt-miller, diesel-office, diesel-shop and diesel-dock workers), smoking status and interaction terms for job category and smoking status (Proc GLM). Correlation and regression analyses of subject-specific urinary analyte levels were based upon background-adjusted values as follows. Since asphalt and diesel-exposed workers had paired pre-shift and post-shift urine samples, background adjustment was performed by subtracting logged pre-shift analyte levels from logged post-shift analyte levels. Background adjustment for coke-oven workers, who had only post-shift urine samples, was performed by subtracting the mean logged level estimated in factory control workers from the logged level observed for each

(Original article)

(98+%) and methanol (purge and trap grade) were obtained from Aldrich Chemical Company (Milwaukee, WI, USA). Sodium chloride was obtained from Fisher Scientific (Pittsburgh, PA, USA). Head space-solid phase microextraction supplies were obtained from Supelco (Bellefonte, PA, USA) and MicroLeter Analytical Supplies, Inc (Suwanee, GA, USA).

Sources of urine samples and published air measurements

Post-shift urine samples were obtained from 28 coke-oven workers (15 top workers and 15 side and bottom workers) and 22 control workers (office and hospital workers) from a single steel-producing complex in northern China. Since levels of Nap and Phe had previously been determined in urine from these subjects, here we compare the original data with those obtained from other groups of workers in the current investigation. Urine samples were also obtained from 26 asphalt workers in road paving crews in the northeastern US. This group included 20 paving workers who applied hot mix asphalt to roads and six milling workers who removed old asphalt from roads. Urine samples were collected from asphalt workers on either two (milling workers) or three consecutive days (paving workers) both before and after work shifts, starting at the beginning of the work week. Finally, urine samples were obtained from 39 diesel-exposed workers who performed various tasks in trucking terminals throughout the US. (While workers in trucking terminals may have experienced PAH exposures from ambient air, diesel exhaust is believed to be their primary source of PAH exposure and we refer to this group as “diesel-exposed workers” for simplicity.) This group included 27 loading-dock workers who drove propane forklifts and loaded trailers, eight truck-repair-shop workers involved with truck maintenance and refuelling activities and four office workers who had only background exposure to diesel exhausts. Pairs of urine samples were collected from each diesel-exposed worker before and after a work shift. The smoking status of all workers was obtained by questionnaires.

Air levels of Nap, Phe and total PAHs were derived from published data reporting exposures in the coke-producing and aluminium industries. The data were previously summarised by Rappaport et al., who focused upon Nap exposures. For this analysis, we consider air levels of both Nap and Phe that were reported along with various other PAHs in several workplaces. The data include 52 measurements (area, breathing-zone and personal samples) for up to 39 PAHs in a coke production plant, three measurements (grouped personal samples from 24 individuals) for 16 PAHs in a coke-production plant, 28 measurements (including area and personal samples) for up to 36 PAHs in an aluminium reduction plant and six measurements (midrange of five personal samples from 6 workers) for 26 PAHs in a carbon anode plant.

All subjects included in this study provided informed consent to participate according to protocols approved by ethics committees at the Harvard University School of Public Health (Boston, Massachusetts, USA) and the Institute for Occupational Medicine (Beijing, China).
coke-oven worker. Since background adjustment was based upon subtraction of logged pre-shift or control values of Nap and Phe from logged post-shift values, the adjusted values, designated adjNap and adjPhe, in natural scale, represent the ratios of post-shift analyte levels to pre-shift or control levels. Pairwise correlations between ln(adjNap) and ln(adjPhe) were estimated using Pearson correlation coefficients (Proc CORR). Least squares multiple linear regression was used to investigate the relation between ln(adjNap) and ln(adjPhe) using dummy variables to test for effects of worker group and job category (Proc REG). Individual observations were investigated as possible outliers based upon influence as measured by leverage, Studentised residuals, Cook’s distance and change in adjusted R^2. Considering these criteria, one observation (out of a total of 93 observations) was rejected from the final model (removing this outlier increased the adjusted R^2 of the final model by 7.7%). Least squares multiple linear regression was also used to investigate the relation between logged air concentrations of Nap and Phe using dummy variables for sources of exposure (coke-producing and aluminium-producing industries) (Proc REG). Three observations of air data (out of a total of 85 observations) were not included in the analysis, because they were reported as containing little or no Nap and/or Phe in the gas phase, a physical impossibility for the environments in question.

RESULTS AND DISCUSSION

Effects of job category on urinary analyte levels

Summary statistics (geometric means (GMs), geometric standard deviations (GSDs) and numbers of subjects) of Nap and Phe levels in pre-shift urine samples (asphalt and diesel-exposed workers only) and post-shift urine samples are presented in table 1, for workers classified by source of PAHs and job category. Results from general linear models showed a significant effect of job category on Nap and Phe levels in both pre-shift (p<0.0005) and post-shift urine (p<0.0001). After adjustment by job category, smoking status did not significantly affect Nap and Phe levels in either pre-shift or post-shift urine (p>0.05).

Significant differences in pre-shift urine levels suggest job-specific variations in background exposures stemming from variability in ambient air exposures, diet, lifestyle factors and/or geographical location. The post-shift data point to an approximate

Table 1 Urinary naphthalene and phenanthrene levels * for workers grouped by source of polycyclic aromatic hydrocarbons (PAHs) and job category

<table>
<thead>
<tr>
<th>Source of PAHs</th>
<th>Job category</th>
<th>No of subjects</th>
<th>Pre-shift (ng/l)</th>
<th>Post-shift (ng/l)</th>
<th>Pre-shift (ng/l)</th>
<th>Post-shift (ng/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diesel exhausts</td>
<td>Dock workers</td>
<td>27</td>
<td>21.2 (1.69)</td>
<td>17.1 (1.71)</td>
<td>3.39 (2.02)</td>
<td>2.67 (1.87)</td>
</tr>
<tr>
<td></td>
<td>Office workers</td>
<td>4</td>
<td>14.9 (1.51)</td>
<td>18.0 (1.42)</td>
<td>4.05 (2.20)</td>
<td>5.18 (1.40)</td>
</tr>
<tr>
<td></td>
<td>Shop workers</td>
<td>8</td>
<td>13.0 (1.55)</td>
<td>19.6 (2.28)</td>
<td>4.40 (2.16)</td>
<td>8.20 (2.31)</td>
</tr>
<tr>
<td>Asphalt emissions</td>
<td>Road milling workers</td>
<td>6</td>
<td>33.3 (1.30)</td>
<td>34.4 (2.01)</td>
<td>10.1 (2.01)</td>
<td>23.8 (2.87)</td>
</tr>
<tr>
<td></td>
<td>Road paving workers</td>
<td>20</td>
<td>32.3 (1.90)</td>
<td>89.1 (1.90)</td>
<td>11.0 (2.61)</td>
<td>69.6 (2.01)</td>
</tr>
<tr>
<td>Coke-oven emissions</td>
<td>Office and hospital workers (factory controls)</td>
<td>22</td>
<td>NA</td>
<td>765 (2.31)</td>
<td>NA</td>
<td>58.2 (3.27)</td>
</tr>
<tr>
<td></td>
<td>Coke-oven workers (side and bottom)</td>
<td>13</td>
<td>NA</td>
<td>1710 (3.39)</td>
<td>NA</td>
<td>735 (4.56)</td>
</tr>
<tr>
<td></td>
<td>Coke-oven workers (top)</td>
<td>15</td>
<td>NA</td>
<td>3450 (5.14)</td>
<td>NA</td>
<td>1250 (7.65)</td>
</tr>
</tbody>
</table>

*Geometric mean (geometric standard deviation) levels are displayed. NA, not available.

Table 2 Background-adjusted levels of naphthalene and phenanthrene in the urine of groups of workers exposed to polycyclic aromatic hydrocarbons (PAHs) from three different sources

<table>
<thead>
<tr>
<th>Source of PAHs</th>
<th>No of subjects</th>
<th>GM (GSD)</th>
<th>Min</th>
<th>Max</th>
<th>GM (GSD)</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diesel exhaust</td>
<td>39</td>
<td>0.959 (2.00)</td>
<td>0.159</td>
<td>7.61</td>
<td>0.989 (2.74)</td>
<td>0.228</td>
<td>17.5</td>
</tr>
<tr>
<td>Asphalt</td>
<td>26</td>
<td>2.20 (2.19)</td>
<td>0.651</td>
<td>10.4</td>
<td>5.04 (3.35)</td>
<td>1.21</td>
<td>219</td>
</tr>
<tr>
<td>Coke ovens</td>
<td>28</td>
<td>3.26 (4.38)</td>
<td>0.144</td>
<td>32.8</td>
<td>16.7 (6.06)</td>
<td>0.228</td>
<td>330</td>
</tr>
</tbody>
</table>

These background-adjusted values represent the ratios of levels of naphthalene and phenanthrene in post-shift urine to either pre-shift urine levels (diesel-exhaust and asphalt sources) or factory-control urine levels (coke-oven source).

GM, geometric mean; GSD, geometric standard deviation.
200-fold range of Nap levels and a 470-fold range of Phe levels across the different job categories, with the highest levels observed in the top coke-oven workers and the lowest levels observed in the diesel-exposed workers. The ranks of observed Nap and Phe levels are consistent with a priori reports of PAH exposures, where coke-oven workers had the highest concentrations (top > side and bottom > steel-factory controls), followed by asphalt workers (paving > milling), followed by diesel-exposed workers. We note that the asphalt workers’ Nap and Phe levels in urine are similar to median levels measured by Campo et al in road paving workers and road construction workers. We also note that the control workers from the Chinese steel-making complex had higher levels of Nap and Phe in their urine than even the asphalt workers, indicating a significant source of background exposure to PAHs in the air of that factory.

Background-adjusted levels of urinary naphthalene and phenanthrene

Summary statistics for Nap and Phe levels are shown in table 2 by source of PAHs, after adjustment for background concentrations of Nap and Phe. Because (in natural scale) the adjusted values represent ratios of post-shift levels to background levels, values of adjNap and adjPhe of approximately one for diesel-exposed workers imply little difference between post-shift and background levels for a typical subject. Adjusted post-shift levels for asphalt workers indicate an approximate twofold and fivefold increase over background levels for Nap and Phe, respectively, and results for coke-oven workers suggest threefold and 17-fold increases over background levels for Nap and Phe, respectively. While background-adjusted values follow the same rankings as the unadjusted levels of Nap and Phe ( coke-oven workers > asphalt workers > diesel-exposed workers), the ranges are considerably smaller—that is, about threefold for adjNap and 17-fold for adjPhe. This reflects the large range of background levels of Nap and Phe observed for each worker group. (We recognise that our use of the large median values of urinary Nap and Phe measured in factory controls to adjust the corresponding urinary levels for the Chinese coke-oven workers could have introduced uncertainty into this analysis.) Since Nap is typically the most abundant PAH measured from a given source, the larger adjPhe values suggest greater urinary excretion of Phe compared to Nap and/or lower relative background concentrations of Phe compared to Nap. This result is consistent with previous results estimating the percentages of excreted Nap and Phe in the coke oven-workers to be 4% and 13%, respectively, of the urinary levels of the metabolites of these PAHs.

Relations between urinary levels of naphthalene and phenanthrene

Significant correlations between ln(adjNap) and ln(adjPhe) levels were observed in all three groups of workers (p<0.0001), suggesting common sources of exposure to Nap and Phe in each case. Pearson correlation coefficients for ln(adjNap) and ln(adjPhe) increased from r = 0.71 for diesel-exposed workers, to r = 0.82 for asphalt workers, to r = 0.89 for coke-oven workers (one outlier excluded).

Multiple linear regression analyses of ln(adjNap) on ln(adjPhe) showed no significant effect of the source of PAHs (coke ovens, asphalt, diesel exhaust) or the job category (p>0.05). Since the sources of PAHs were dramatically different, this finding was unexpected. However, it is clear from figure 1 that the overall relation between ln(adjNap) and ln(adjPhe) was essentially the same for each of the three groups of workers.

Figure 1 also shows that the intra-group variability of the data pairs [ln(adjNap), ln(adjPhe)] was very large for each of the three sources of PAHs. The final regression model (after removal of one outlier) is given as: ln(adjNap) = −0.121 + 0.551 × ln(adjPhe) (n = 92), with an adjusted R² value of 0.751. The log-scale regression coefficient of 0.551, with 95% confidence limits of [0.485, 0.617], was significantly less than 1. This suggests that, in natural scale, the ratio of Nap/Phe diminished with increasing levels of Phe.

Relations between air levels of naphthalene, phenanthrene and total PAHs

In selecting Nap and Phe as possible surrogates for PAH exposure, it is important that urinary levels accurately reflect air concentrations of total PAHs at the time of urine collection. Rappaport et al reported high correlations between logged levels of airborne Nap and logged levels of total airborne PAH in published data from several industries—namely, creosote impregnation (Pearson r = 0.815), coke production (r = 0.917), an iron foundry (r = 0.854) and aluminium production (r = 0.933). Furthermore, the estimated slopes of the log-scale relations between ln(Nap) and ln(total PAH) ranged from 0.824 to 1.19, indicating that air concentrations of Nap were roughly proportional to those of total measured PAHs in each case.

Using the same data previously analysed by Rappaport et al, we investigated the straight-line relations between logged levels of airborne Phe and logged levels of total PAHs. Datasets reporting air Phe levels were only available in studies of the aluminium-producing and coke-producing industries. Across four datasets, 60 out of 66 observations of total PAH levels included measurements of Phe. We note that, for studies from Bjorseth et al, some observations specified both gaseous and particulate PAH measurements; open circles, data from Strunk et al.11

Figure 2 Air phenanthrene concentration (µg/m³) vs total PAH concentration (µg/m³) (minus phenanthrene concentration) in aluminium-production facilities (A) and coke-production facilities (B). Solid triangles, data from Bjorseth et al (gas plus particulate measurements); open triangles, data from Bjorseth et al (particulate measurements only); plus symbols, data from Petry et al; solid circles, data from Bjorseth et al (gas plus particulate measurements); open circles, data from Bjorseth et al (particulate measurements only); ×, data from Strunk et al.
particulate air levels, while others specified only particulate levels. We distinguished between these types of observations in figure 2, where we examined the log-log relations of Phe and total PAHs. As shown in figure 2, the estimated slopes of the log-log relations were 1.09 and 1.15 for aluminium-producing and coke-producing industries, respectively, indicating that levels of airborne Phe were roughly proportional to those of total PAHs in these industries.

We used these same data to investigate the relation between air concentrations of Nap and Phe in the aluminium-producing and coke-producing industries. The final analysis included six observations from Bjsorset et al., 15 observations from Bjorseth et al., 16 three observations from Strunk et al. 17 and six observations from Petry et al. 18 As shown in figure 3, the relation between air Nap and air Phe is given by the regression model: ln[Nap (µg/m³)] = 0.723 + 0.769 × ln[Phe (µg/m³)] + 1.50[source]; where coke-production source = 1 and aluminium-production source = 0; adjusted R² = 0.796.

CONCLUSIONS

We conclude that levels of Nap and Phe in urine from diesel-exposed workers, asphalt workers and coke-oven workers followed the expected low, medium and high exposure designations and were highly correlated with each other, suggesting common sources of the two PAHs. Regression analyses of ln(adjNap) values on the corresponding ln(adjPhe) values showed no significant effect of the source of PAH in these three cases. Furthermore, these analyses indicated that the ratio of urinary Nap/Phe decreased with increasing levels of urinary Phe. Since the ratio of airborne Nap/Phe was also found to decrease with increasing airborne Phe, in independent sets of data from the coke-producing and aluminium-producing industries, we conclude that urinary levels of Nap and Phe very probably reflect the corresponding levels of airborne Nap and Phe at the time of urine collection. Also, we observed that air concentrations of Nap and Phe were both proportional to total PAHs in the coke-producing and aluminium -producing industries. Taken together, these findings, plus the ease and sensitivity of measuring Nap and Phe in urine, lead us to conclude that urinary levels of Nap and Phe are promising surrogates for occupational exposures to total PAHs. Of the two urinary analytes, Phe may be a more useful surrogate than Nap, given the much larger fold increases above background values observed for Phe (onefold to 17-fold) compared to Nap (onefold to threefold) across the three sources of PAHs in our study (see table 2).

Main messages

Urinary naphthalene and phenanthrene are promising surrogates for occupational exposures to polycyclic aromatic hydrocarbons.

Policy implications

Measurement of urinary naphthalene and phenanthrene could simplify the assessment of occupational exposures to polycyclic aromatic hydrocarbons.

REFERENCES


