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# Urinary Metabolites of Polycyclic Aromatic Hydrocarbons in Saudi Arabian Schoolchildren in Relation to Sources of Exposure

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**ABSTRACT**

Polycyclic aromatic hydrocarbons contain a number of known carcinogenic compounds, and urinary biomarkers have been widely used as a measure of exposure but quantitative relationships with exposure variables have proved elusive. This study aimed to quantify the relationship between exposures to phenanthrene and pyrene from atmospheric and dietary sources with the excretion of 1-hydroxypyrene and hydroxyphenanthrenes in urine as biomarkers of exposure. The study population consisted of 204 male schoolchildren attending three schools in different parts of Jeddah, Saudi Arabia who provided urine samples on each of three consecutive days. Outdoor air measurements of polycyclic aromatic hydrocarbons were made at the schools and the children provided information on diet, exposure to environmental tobacco smoke and incense, and various lifestyle factors through a questionnaire. Mixed models with random effects for subjects nested within site were fitted in order to examine the relationship between exposure variables and urinary PAH metabolites. A unit increase (1 ng $m^{-3}$) in ambient pyrene (particulate plus gaseous phase) was associated with a 3.5% (95% CI: 1.01, 5.13) increase in urinary 1-hydroxypyrene concentration. A unit increase in ambient phenanthrene caused a 1.01% (95% CI: 0.03, 2.02) increase in total hydroxyphenanthrene concentrations. Consumption of chargrilled food increased the hydroxypyrene and hydroxyphenanthrene concentrations by 24% (95% CI: 11, 37) and 17% (95% CI: 8, 26) respectively. We did not find evidence of association for environmental tobacco smoke exposure or incense burning. It is concluded that both respiratory exposure and consumption of chargrilled food are considerable sources of PAH exposure as reflected by concentrations of urinary biomarkers.

**Keywords:** Polycyclic aromatic hydrocarbons; atmosphere; diet; exposure

## 1. INTRODUCTION

A number of polycyclic aromatic hydrocarbons (PAH) are known genotoxic carcinogens. The compounds arise from combustion sources and evaporation of petroleum-derived fuels and are hence widespread in the environment, and exposure occurs by inhalation (Mari et al., 2010), ingestion (Martorell et al., 2012; Perello et al., 2009), and potentially by dermal absorption. PAH occur in the environment as a complex mixture of individual compounds, referred to as congeners. Exposure to polluted air through inhalation poses a risk of lung cancer (Hamra et al., 2014), and PAH exposure appears to make an appreciable contribution to that risk (Harrison et al., 2004). The European Union has set a target value of 1 ng $m^{-3}$ of benzo(a)pyrene (B(a)P), taken as representative of the PAH mixture, and in the United Kingdom, an air quality objective of 0.25 ng $m^{-3}$ of B(a)P has been adopted. PAH are also carcinogenic in animal models as a result of ingestion in the diet or drinking water. Consequently the European Food Safety Authority has made a detailed assessment of human exposure from dietary sources (EFSA, 2008). In addition to the cancer risk, PAH exposure has been linked to the onset of diabetes mellitus (Yang et al. 2014; Alshaarawy, et al. 2014), metabolic syndrome (Brocato et al. 2014; Hu et al. 2015), and cardiovascular conditions (Xu et al. 2010; Feng et al. 2014). Since all exposure sources can contribute to the body burden of PAH, quantitative evaluation of exposure pathways is important if health risk is to be minimised.

While exposure can be evaluated by separate chemical analysis of PAH in exposure media such as food, water and air, such monitoring is highly resource intensive and the use of urinary biomarkers of exposure is a more practicable means of exposure estimation, although without additional information, it cannot quantify the contribution of different exposure pathways. PAHs are oxidised in the body by P450 enzymes ultimately to form hydroxylated metabolites. Of these, urinary 1-hydroxypyrene (1-OHPyr) has been used most extensively as an exposure biomarker in both occupational cohorts and the general population. It has the advantages of strong correlation with

metabolites of other PAH (Li et al. 2008) and of having a relatively short biological half life, and hence being representative of recent exposure. Half-lives from inhalation exposure are reported as between 6-35h (Brzeznicki et al. 1997; Jongeneelen et al. 1990), 4.4h and 12h after oral ingestion (Buckley and Lioy, 1992; Viau et al. 1995) and 3.5-5.1h for the first phase (Li et al., 2012).

Hansen et al. (2008) have reviewed 132 studies addressing the use of 1-hydroxypyrene as a biomarker of both occupational and environmental exposure to PAH. Of these, 25 studies addressed environmental exposure, and only nine included children. Studies of children not included in that review include those of Vyskocil et al. (2000) and Freire et al. (2009). It is notable that rather few environmental studies have made measurements of exposure, either through chemical analysis of air or diet, or by questionnaire in relation to diet. Several have used a cross-sectional design in which subjects from areas deemed to be more polluted are compared to groups from less polluted areas (Vyskocil et al. 2000; Wilhelm et al. 2007; Lee et al. 2007; Hansen et al. 2005). This can provide useful insights into relative exposures, but does not quantify the contribution of different pathways to intake. Some studies of adults have used benzo(a)pyrene or a sum of PAH in air concentrations as an indication of airborne PAH exposure (Fiala et al. 2001; Merlo et al. 1998), but since the relative amounts of PAH congeners in mixtures vary from place-to-place, this does not provide direct information on the relationship between exposure to pyrene and urinary 1-hydroxypyrene concentrations. Very few studies have used measurements of pyrene in exposure media (Vyskocil et al. 2000; Cavanagh et al. 2007), and not all have fully recognised the importance of measuring both the vapour and particulate components of airborne pyrene (Suzuki and Yoshinaga, 2007), as unlike benzo(a)pyrene, it is the vapour phase component which is typically dominant. Other studies have used nitrogen dioxide or $NO_x$ as a marker of road traffic exposure (Freire et al. 2009; Kanoh et al. 1993), but this is not necessarily reflective of total atmospheric PAH exposure, as PAH have other important sources (Jang et al., 2013; Alghamdi et al., 2015).

In this study, the PAH exposure of children of median age 11 years attending three schools in Jeddah, Saudi Arabia has been evaluated through analysis of hydroxylated metabolites of phenanthrene and pyrene in urine. Urinary metabolite concentrations have been related to sources of exposure through a questionnaire on diet, passive smoking and other lifestyle factors, and chemical analysis of airborne concentrations.

## 2. MATERIALS AND METHODS

### 2.1 Data Collection

A total of 204 school boys in Jeddah city were recruited for the study. A questionnaire was used to obtain data on baseline characteristics including age, gender, passive smoking, socio-economic indicators, housing conditions and current health status. The questionnaire was validated through trials on adult subjects and children with additional questions to gauge comprehension. The children were enrolled in three different schools located in differing environments; the first school was located near to an oil refinery (Site A), the second to a major highway (Site B) and the last to the Red Sea (Site C). Daily ambient atmospheric PAH concentrations were measured in both particle-associated and vapour phases for each site on consecutive days. A summary of covariates appears in Table 1. Full details of the sampling and analytical methods, and measured concentrations have been reported elsewhere (Alghamdi et al. 2015). For sites A and B air sampling data for 23, 24 and 25 February 2013 was used in conjunction with the corresponding urine samples collected on 24, 25 and 26 February 2013 respectively. For site C the air and urine samples used in the data analysis were collected on 20, 21 and 22 April and 21, 22 and 23 April 2013 respectively. The questionnaire was updated on each urine sampling occasion by asking additional information about the previous day's dietary and cooking patterns as well as use of incense**.** Urine samples were provided from the first morning micturition event, and were rapidly frozen and stored at -80°C before being transported in dry ice to the analytical laboratory. They were analysed for hydroxyphenanthrenes and 1-hydroxypyrene by HPLC according to a recommended method of the German Research

Foundation (DFG, 1999) as described by Hemat et al. (2012). Very low values fell below the limit of detection. For these, a value of LOD$\div \sqrt{2}$ was adopted, following the recommendation of Hornung and Reed (1990). Creatinine in urine was determined photometrically as picrate according to the Jaffé method (Taussky, 1954). A total of five biomarkers, both raw and after creatinine correction, were available. These included 1-OH-phenanthrene, sum of 2-/9-OH-phenanthrene, 3-OH-phenanthrene, 4-OH-phenanthrene and 1-OH-pyrene. From the 204 students enrolled, 170 presented three urine samples (58 from School A; 66 from School B and 46 from School C). The statistical analysis was based upon these 510 (i.e. 170 x 3) samples.

### 2.2 Statistical Analysis

Ambient and biomarker PAH concentrations were summarised by site and date. Correlations between each PAH pair were explored using plots and their magnitudes were computed. PAH concentrations from urine sample were transformed to the log (natural) scale for statistical analysis. Mixed models with random effects for subjects nested within site (Verbeke and Molenberghs, 2000) were fitted in order to examine the relationship between ambient and biomarker (urinary) PAH concentrations. We controlled for potential confounding by passive smoking, use of incense, consumption of char-grilled and fried food (all as categorical variables), age and BMI. In order to avoid multiple testing, we opted to use only 1-OH-Pyrene (1-OHPyr) and the sum of the all the OH-Phenanthrenes (OH-Phen) family (but separately for raw and normalised samples) as the outcomes of interest in our models. We supplemented the analysis by exploring all pairwise associations between individual biomarker and ambient PAHs. A likelihood ratio test was used to assess the effect of smoking and Chi-square statistics and *p*-values were reported.

## 3. RESULTS AND DISCUSSION

The median age among the boys was 11 years with interquartile range (IQR) between 10 and 12 years. The median BMI was 17.3 with an IQR between 15.4 and 21.3. The proportion of the boys

who consumed chargrilled food was lower (37%) compared to fried food (70%). Most houses were with no smokers (64%) while 28% had one smoker and 41% used incense in the house. A summary of questionnaire responses appear in Table 1.

The OH-Phen isomers were strongly correlated with one another with the minimum and maximum observed correlation coeffients at 0.59 and 0.95 respectively. Likewise reasonably strong correlations for OH-Phen isomers were observed with hydroxy-pyrene measurements. The total OH-Phen showed very strong correlation with all the individual biomarkers with measured correlations ranging between 0.86-0.98 (Table S1). Thus our main analysis was based on using total hydroxyphenanthrenes as a proxy for all hydroxyphenanthrene compounds, and 1-OHPyr.

Table 2 presents a summary of both the urinary biomarkers and ambient phenanthrene and pyrene concentrations for each sampling day as well as by site. The results indicate that particulate-associated PAH concentrations were very low for all the three sampling days and sites. Total ambient phenanthrene and pyrene were used as the atmospheric exposure metric.

There have been many more reported measurements of 1-hydroxypyrene than those of the hydroxyphenanthrenes. Consequently, data from other studies of children for urinary 1-hydroxypyrene normalised by creatinine are shown for comparison in Table 3. Because of the strong age-dependence of creatinine excretion (Remer et al., 2002; Mage et al., 2008), studies of children within a comparable age group have been selected. The concentrations in the present study are high in relation to other groups studied. Both the median and highest concentrations (creatinine normalised) exceed those in most earlier work. We can only speculate as to the reasons. As the PAH body burden is determined by intake and metabolism/excretion, the most plausible explanation is that intakes were relatively high, and both the airborne concentration data, which appear high in comparison with many other studies, and the frequency of chargrilled food

consumption are likely to be influential factors. Comparative data for hydroxyphenanthrenes are far fewer in the literature and a few comparative values appear in Table 4.

As a comparison between sites, Figure 1 shows geometric mean (and 95% confidence interval) biomarker concentrations for each school for both raw and creatinine normalised data. The appreciably lower hydroxy-PAH values at site C with lower airborne PAH are clearly seen. This is despite other exposure factors varying little between the locations (Table 1). In Table 5, inter-site differences are shown, using site C as the background reference site. Both the t-test and Chi squared test show a significant ($p<0.05$) difference between urinary hydroxypyrene concentrations at both sites A and B, when compared with site C. This difference is significant both for raw and creatinine-normalised concentrations. On the other hand, results for the sum of hydroxy-phenanthrenes do not show significant differences for either site (Table 5).

Inhalation exposures make an important contribution to hydroxy-PAH (Table 6). The total hydroxyphenanthrene concentration (raw data) showed an increase of 1.01% (95% CI: 0.03, 2.02) and 2.02% (95% CI: 0.1, 4.08) for each unit increase (1 ng $m^{-3}$) in ambient phenanthrene and pyrene concentration respectively after controlling for age, body mass index (BMI) and consumption of chargrilled food. However, a significant association could not be shown for the corresponding hydroxyphenanthrene levels normalised by creatinine concentration; the percentage increases were 1.01% (95% CI: -0.04, 1.01) and 1.01% (95% CI: -0.3, 3.05) for a unit increase (1 ng $m^{-3}$) in ambient phenanthrene and pyrene concentrations respectively (Table 6). Similarly a unit increase in ambient phenanthrene and pyrene concentrations was associated with 1.01% (95% CI: 0.3, 2.02) and 3.05% (95% CI: 1.01, 5.13) increase in raw urinary hydroxypyrene concentrations respectively. Again such an association was not apparent for the normalised samples (Table 6). The mean air concentrations averaged across all three sites are 17.2 ng $m^{-3}$ of phenanthrene and 4.2 ng $m^{-3}$ for pyrene. These surrogate airborne exposures account for 19% of the hydroxyphenanthrenes

concentration and 13% of 1-hydroxypyrene as the models are log-linear. For the most polluted site A, these percentages rise to 30% for hydroxyphenanthrene and 26% for 1-hydroxypyrene.

Many studies have drawn attention to diet, and particularly to certain cooking methods as a source of PAH intake (Perello et al., 2009). Consumption of chargrilled food increased the raw and normalised hydroxyphenanthrene concentrations by about 25% and 19% respectively compared to those who did not consume such food showing consistently strong association (p-value<0.01 in all cases, Table 7). Moreover, BMI tended to increase hydroxyphenanthrene concentrations for the creatinine normalised sample (p-value = 0.03) but not for the raw sample (p-value >0.05). Although not significant, age was negatively associated with hydroxyphenanthrene concentration in both raw and corrected samples (Table 7). For pyrene models, BMI did not show material association while consumption of chargrilled food remained a strong predictor of urinary concentration (Table 6), accounting for 24% and 17% respectively in raw and creatinine normalised 1-hydroxypyrene. Incense burning was not found to be a significant influence upon either hydroxyphenanthrenes or 1-hydroxypyrene concentration (results not shown). Passive smoking (ETS exposure) was accounted for by the data for the number of smokers in the household (n = 0,1,2,3,>4). As there was only one subject in the >4 group, this was combined with the n = 3 group. ETS exposure was then added to the model presented and its significance checked through a Chi square probability test (results are in Table S4). There was no evidence of association with hydroxy-PAH, so ETS was excluded from the final model.

Vyskocil et al. (2000) studied children of 3-6 years of age in Montreal, Canada, living at two sites with outdoor pyrene concentrations of 2.7 ng $m^{-3}$ and 0.4 ng $m^{-3}$ respectively (each the mean of three measurements). These exposures are substantially lower than those in our study (Table 1). They estimated that the respiratory dose accounted for 5-7% of total absorbed intake, the remainder arising from the diet, taking account of both indoor and outdoor atmospheric exposures (Vyskocil et

al. 2000). No relationship was found between absorbed doses of pyrene in diet, air, or both combined, with 1-hydroxypyrene in urine, which was attributed to uncertainties in the food uptake or the limited statistical power of the study (n = 10-13). Fiala et al. (2001) studied children of 3-6 years in the Czech Republic. They measured outdoor pyrene concentrations in the range 0.43-1.50 ng $m^{-3}$, also appreciably lower than in the current study. Air inhalation was calculated to account in summer for 4-5% of absorbed dose (assuming 12.5% gastrointestinal absorption) or 0.4-0.7% of absorbed dose (assuming 100% G.I. absorption) at the more polluted site. The equivalent figures for the less polluted site were 2-3% and 0.3% respectively. In the winter data, the equivalent figures were 4-15% and 0.6-2.1% for the polluted site, and 4-8% and 0.5-1.0% for the less polluted site (Fiala et al. 2001). Relationships of both ingestion and inhalation doses with 1-hydroxypyrene in urine were both very weak. However, the contribution of soil ingestion as an exposure pathway was estimated to be negligible (Fiala et al. 2001).

Lee et al. (2007) applied a multiple regression model to evaluate the influences on urinary 1-hydroxypyrene in samples taken from child residents living in two separate areas at different distances from a steel mill. The study showed a highly significant association with location of residence suggesting an important contribution of airborne exposures, but these were not measured in their study. Other associations significant at the 5% level were with age, sex, monthly income and consumption of smoked ham. A positive association was also found with consumption of charbroiled pork, but this was not significant (p = 0.188).

Many studies have emphasised the importance of diet as a pathway for PAH exposure, but in most cases this has been based upon chemical analysis. The studies summarised above show that it is difficult to demonstrate a clear relationship between dietary exposure and urinary excretion of 1-hydroxypyrene. Grilled, fried, smoked and baked meals can contain particularly high levels of PAH (Ludovici, et al. 1995; Fiala et al. 2001) and our results emphasise the importance of

chargrilled food as a PAH source. In common with our study, others have also failed to find a relationship between environmental tobacco smoke exposure and urinary excretion of PAH biomarkers (Siwinska et al. 1998; Hansen et al. 2005).

From Table 6 it may be seen that results for creatinine normalised hydroxy-PAH data showed positive associations with airborne concentrations but failed the $p<0.05$ significance criterion. In the case of chargrilled food consumption, associations were significant with both normalised and un-normalised data (Table 7). Creatinine normalisation is frequently conducted to allow for variations in urine flow rate. The question of the appropriateness of normalising urinary biomarkers with creatinine has been widely discussed. While Viau et al. (2004) advocate creatinine adjustment, Boeniger et al. (1993) and Barr et al. (2005) point out that creatinine excretion is influenced by meat intake, diurnal variations, age, gender and other factors. According to Remer et al. (2002) and Mage et al. (2008), daily creatinine excretion is a strong function of age. Heavner et al. (2006) point out that the general relationship between a component's excretion rate and urine flow is indicative of the renal excretion mechanism. They report a zero slope for excretion rate/urine flow for creatinine, suggesting no urine flow effect on creatinine excretion rate (Heavner et al., 2006). Consequently, for biomarkers excreted mainly by passive diffusion, a non-zero slope would be expected, and creatinine normalisation would not be appropriate. In reporting data from non-smokers, Aquilina et al. (2010) found that correlations of the urinary tobacco smoke markers creatinine and trans-3′-hydroxycotinine with exposures to tobacco smoke components 3-ethenylpyridine, 1,3-butadiene and some PAH were higher in non-creatinine normalised data. We therefore feel confident in regarding the associations with the un-normalised hydroxy-PAH data as meaningful. It is also notable that in the comparison between sites shown in Table 5, the larger and more significant difference between sites are seen for the creatinine-normalised data.

There are some weaknesses to the study. As noted in the Introduction, some estimates of the biological half life of hydroxy-PAH are quite short, and collection of the first morning samples of urine may not have been ideal. A clearer dietary signal might have been given by an evening sample. It would have been optimal to integrate daily hydroxy-PAH excretion, but this would have been well beyond the capabilities of this study. Airborne exposures were measured outdoors over 24-hour periods, and the use of indoor and/or personal samplers would have better represented the exposures to airborne PAH, but were not practicable within the study.

A companion study using the same PAH and hydroxy-PAH dataset (Trasande et al., 2015) examined brachial artery distensibility and blood pressure in the same school children, finding a positive association between prehypertension and proximity to the oil refinery in Jeddah.

## 4. CONCLUSIONS

This study has analysed urine samples from a far larger number of subjects than most earlier studies. It has compared airborne concentrations in three different areas of Jeddah and has benefitted from day-to-day changes in airborne concentrations. The large number of samples and the wide range of exposures has contributed to a greater statistical power than in most earlier studies which has shown marked benefits in terms of quantifying the gradient between airborne concentrations of pyrene and phenanthrene and the urinary concentrations of their hydroxy metabolites. This has given quantitative support to the significance of airborne exposures in influencing the body burden of PAH previously inferred from qualitative cross-sectional studies, and points to the benefits which could accrue from measures designed to reduce airborne concentrations.

The major advance achieved by this study is the elucidation of a quantitative relationship between airborne concentrations and hydroxy-PAH excretion, and quantification of the contribution from

chargrilled food. While many other studies have inferred that the contribution of chargrilled food to the body burden of pyrene is important, this study has quantified the average contribution of the consumption of chargrilled food to overall PAH exposure as represented by hydroxypyrene and hydroxyphenanthrenes urinary excretion. The contribution is substantial and serves to highlight the potential risks associated with a diet rich in chargrilled foods.

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**Ethical Approval:** The study proposal was scrutinised and approved by King Abdulaziz University Research Ethics Committee (protocol no. 700-12, approval date 4/01/2012).

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## TABLE LEGENDS

**Table 1:** Summary of covariates by site.

**Table 2:** Median (IQR) biomarker (ng $L^{-1}$) and ambient concentrations (ng $m^{-3}$) by site and sampling date.

**Table 3:** Comparison between 1-OHPyr results (median and range) of the present study and previous studies.

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**Table 6.** Percentage increase in biomarker PAH concentration per unit increase in ambient atmospheric concentrations.

**Table 7.** Detailed results with coefficients for confounders.

## FIGURE LEGEND

**Figure 1:** Geometric means (95% CI) for biomarker PAHs of the three sites A, B and C (sum of hydroxyphenanthrenes and 1-hydroxypyrene only).

**Table 1:** Summary[¶] of covariates by site

| Variable | | Site | | |
|---|---|---|---|---|
| | | A | B | C |
| Consumed chargrilled food | *No* | 102 (61.08) | 117 (62.57) | 87 (65.91) |
| | *Yes* | 65 (38.92) | 70 (37.43) | 45 (34.09) |
| Consumed fried food | *No* | 60 (35.71) | 33 (17.19) | 54 (40.6) |
| | *Yes* | 108 (64.29) | 159 (82.81) | 79 (59.4) |
| Use of incense | *No* | 89 (53.29) | 99 (52.11) | 98 (74.81) |
| | *Yes* | 78 (46.71) | 91 (47.89) | 33 (25.19) |
| Smokers in the house | *None* | 118 (64.13) | 119 (60.41) | 103 (66.88) |
| | *1* | 48 (26.09) | 66 (33.5) | 38 (24.68) |
| | *2* | 9 (4.89) | 9 (4.57) | 10 (6.49) |
| | *≥3* | 9 (4.89) | 3 (1.52) | 3 (1.95) |
| Age, Median (IQR) | | 11 (2) | 11 (1) | 11 (2) |
| BMI, Median (IQR) | | 16.64 (6.16) | 16.64 (4.47) | 19.29 (6.9) |

[¶]N (%)

**Table 2:** Median (IQR) biomarker (ng $L^{-1}$) and ambient concentrations (ng $m^{-3}$) by site and sampling date

| | Site | | | Sample | | |
|---|---|---|---|---|---|---|
| PAHs | A | B | C | 1 | 2 | 3 |
| 1-hydroxyphenanthrene | 189.8 (211.8) | 132.4 (127.5) | 148.1 (171.5) | 147.8 (162.8) | 166.7 (152.9) | 181.2 (220.2) |
| 1-hydroxyphenanthrene (norm) (ng $g^{-1}$) | 209.3 (189.6) | 193.8 (150.7) | 168.5 (134.3) | 184.8 (149.9) | 189.2 (167.9) | 199.7 (188.7) |
| 2+9-hydroxyphenanthrene | 85.2 (79.9) | 68.9 (64) | 76.6 (82.7) | 73 (70.4) | 75.9 (66.5) | 80.7 (88.3) |
| 2+9-hydroxyphenanthrene (norm) (ng $g^{-1}$) | 89.6 (81.5) | 100.2 (69.2) | 80 (86.6) | 96.8 (77.9) | 87.6 (77.7) | 91.9 (84.9) |
| 3-hydroxyphenanthrene | 178.6 (182.8) | 147.8 (148.7) | 141.9 (158.9) | 146.8 (170.1) | 159.9 (152.9) | 165.6 (184) |
| 3-hydroxyphenanthrene (norm) (ng $g^{-1}$) | 194.9 (181.6) | 216.8 (154.8) | 146.2 (114.1) | 187.1 (147.5) | 180.2 (178.9) | 194.1 (172) |
| 4-hydroxyphenanthrene | 40.9 (38.6) | 33.9 (29.5) | 28.7 (36.8) | 32.9 (34.4) | 34 (34.5) | 37.3 (40.8) |
| 4-hydroxyphenanthrene (norm) (ng $g^{-1}$) | 47.2 (42.2) | 48.3 (36.4) | 34.9 (32.1) | 45.7 (44) | 43 (37.7) | 44.6 (38.4) |
| Sum of hydroxyphenanthrene | 489.1 (495.1) | 406.3 (364) | 423.8 (450.1) | 411.5 (425.7) | 459.3 (408.2) | 470.3 (520.7) |
| Sum of hydroxyphenanthrene (norm) (ng $g^{-1}$) | 525.9 (486.4) | 565.5 (397) | 443.6 (377.5) | 523.2 (455.7) | 502.5 (425.4) | 523 (489.3) |
| 1-hydroxypyrene | 304.7 (307.6) | 191 (252.3) | 168.3 (216.2) | 216.3 (248.3) | 210.3 (233.5) | 228.8 (322) |
| 1-hydroxypyrene (norm) (ng $g^{-1}$) | 339.3 (304.7) | 278.1 (237.5) | 189.1 (216.1) | 268 (292.7) | 261.6 (273.4) | 268.5 (272.3) |
| Phenanthrene (vapour) (ng $m^{-3}$) | 27.1 (7.7) | 18.1 (16.9) | 4.2 (5.5) | 12.7 (13.9) | 18.1 (22.9) | 29.5 (27) |
| Pyrene (vapour) (ng $m^{-3}$) | 7.3 (4.2) | 2.1 (5.9) | 0.5 (0.9) | 2.1 (6) | 1.9 (5.2) | 7.8 (9.5) |
| Phenanthrene (particulate) (ng $m^{-3}$) | 0.14 (0.1) | 0.14 (0.04) | 0.03 (0.02) | 0.14 (0.1) | 0.11 (0.09) | 0.14 (0.19) |
| Pyrene (particulate) (ng $m^{-3}$) | 0.12 (0.2) | 0.25(0.1) | 0.04 (0.02) | 0.12 (0.26) | 0.11 (0.14) | 0.25 (0.29) |
| Phenanthrene (total) (ng $m^{-3}$) | 27.2 (7.7) | 18.3 (16.9) | 4.2 (5.5) | 12.8 (14) | 18.3 (23) | 29.7 (27.2) |
| Pyrene (total) (ng $m^{-3}$) | 7.4 (4.4) | 2.4 (6) | 0.5 (0.9) | 2.4 (6.1) | 2.1 (5.3) | 8.1 (9.8) |

**Table 3:** Comparison between 1-OHPyr results (median and range) of the present study and previous studies

| Country | Period | Age (years) | Particulars | N | Urinary 1-OHPyr concentration [ng/g creatinine][a] | Reference |
|---|---|---|---|---|---|---|
| Denmark | 1994-1995 | 4 - 13 | Urban residence | 100 | 193 (19 – 1215) | Hansen et al. (2005) |
| Denmark | 1994-1995 | 3 - 13 | Rural residence | 97 | 135 (19 – 3203) | Hansen et al. (2005) |
| USA | 2001-2002 | 6 - 11 | National survey | 387 | 67 (58 – 78) [b] | Li et al. (2008) |
| USA | 2001-2002 | 12 - 19 | National survey | 735 | 44 (38 – 53) [b] | Li et al. (2008) |
| Korea | 2004 | 11.3 ± 2.4 | Industrial area | 406 | 93 (17 – 992) | Lee et al. (2007) |
| Korea | 2004 | 10.8 ± 2.6 | Remote area | 606 | 69 (<LOD – 1829) | Lee et al. (2007) |
| New Zealand | 2004 | 12 - 18 | Autumn season | 88 | 37 (8 – 197) | Cavanagh et al. (2007) |
| New Zealand | 2004 | 12 - 18 | Winter season | 79 | 83 (12 – 276) | Cavanagh et al. (2007) |
| Thailand | 2004-2005 | 8 - 12 | Urban residence | 115 | 309 (58 – 1910) | Tuntawiroon et al. (2007) |
| Thailand | 2004-2005 | 9 - 13 | Rural residence | 69 | 212 (39 – 829) | Tuntawiroon et al. (2007) |
| Mexico | 2009 | 6 - 12 | Urban area | 37 | 251 (39-1660) | Martinez-salinas et al. (2012) |
| Congo | 2009 | 6 - 14 | Urban residence | 56 | 1700 (300 – 14800) | Tuakuila et al. (2013) |
| Mongolia | 2011-12 | 11-15 | Urban area (warm season) | 320 | 269 (404) [c] | Chen et al. (2015) |
| Mongolia | 2011-12 | 11-15 | Urban area (cold season) | 320 | 577 (1096) [c] | Chen et al. (2015) |
| Saudi Arabia | 2013 | 10 - 12 | Urban area | 204 | 264 (15 – 2476) | Present study |

[a] Median (range)
[b] Geometric mean and 95% CI
[c] Mean (standard deviation)

**Table 4:** Geometric mean concentrations for hydroxyphenanthrenes (ng $L^{-1}$)

| Country | Age | Concentration (ng $L^{-1}$) | | | | | | | Reference |
|---|---|---|---|---|---|---|---|---|---|
| | | 1-OH | 2-OH | 3-OH | 4-OH | 3+4-OH | 9-OH | 2+9-OH | |
| USA | 6-11 | 165 | 55 | 148 | 43 | | 38 | | Li et al. (2008) |
| USA | 12-19 | 122 | 46 | 98 | 39 | | 30 | | Li et al. (2008) |
| USA | >20 | 146 | 56 | 106 | 43 | | 35 | | Li et al. (2008) |
| UK | Adult | 220 | 140 | | | 220 | | | Aquilina et al. (2010) |
| Saudi Arabia | 10-12 | 155 | | 156 | 35 | | | 78 | This study |

**Table 5:** Differences in biomarker PAHs associated with site

| PAHs | Site | Difference[¶] (95%CI) | $\chi 2$(P-value)[b] |
|---|---|---|---|
| Sum of hydroxyphenanthrene (raw) | A | 0.15 (0.15, 0.49) | 3.2 (0.2) |
| | B | -0.07 (-0.07, 0.2) | |
| Sum of hydroxyphenanthrene (norm) | A | 0.24 (0.24, 0.55) | 4.16 (0.12) |
| | B | 0.22 (0.22, 0.53) | |
| 1-hydroxypyrene (raw) | A | 0.43 (0.43, 0.92) | 6.99 (0.03) |
| | B | 0.08 (0.08, 0.45) | |
| 1-hydroxypyrene (norm) | A | 0.55 (0.55, 1.05) | 9.91 (0.01) |
| | B | 0.42 (0.42, 0.88) | |

[¶]Differences are on the log scale; reference category is site C

[b]P-value (from likelihood ratio test) for including site in the model adjusted for char grilled food, age and BMI

**Table 6.** Percentage increase in biomarker PAH concentration per unit increase in ambient atmospheric concentrations*

| Variable | Difference (%) | 95% CI | P-value |
|---|---|---|---|
| **Sum of hydroxyphenanthrenes** | | | |
| phenanthrene (total) | 1.01 | (0.03, 2.02) | 0.04 |
| pyrene (total) | 2.02 | (0.1, 4.08) | 0.04 |
| **Sum of hydroxyphenanthrenes (norm)** | | | |
| phenanthrene (total) | 1.01 | (-0.04, 1.01) | 0.07 |
| pyrene (total) | 1.01 | (-0.3, 3.05) | 0.11 |
| **1-hydroxypyrene** | | | |
| phenanthrene (total) | 1.01 | (0.3, 2.02) | 0.01 |
| pyrene (total) | 3.05 | (1.01, 5.13) | 0.01 |
| **1-hydroxypyrene (norm)** | | | |
| phenanthrene (total) | 0.3 | (-0.4, 1.01) | 0.40 |
| pyrene (total) | 1.01 | (-1.0, 3.05) | 0.19 |

*Models adjusted for consumption of chargrilled food, age and BMI

**Table 7.** Detailed results with coefficients for confounders[¶]

| Variable | Difference | 95% CI | P-value |
|---|---|---|---|
| **Sum of hydroxyphenanthrenes** | | | |
| phenanthrene (air total) | 0.01 | (0.0003, 0.02) | 0.04 |
| charfood | 0.22 | (0.09, 0.35) | <0.01 |
| age | -0.02 | (-0.12, 0.07) | 0.61 |
| BMI | 0.02 | (-0.002, 0.04) | 0.08 |
| pyrene (air total) | 0.02 | (0.001, 0.04) | 0.04 |
| **Sum of hydroxyphenanthrenes (norm)** | | | |
| phenanthrene (total) | 0.01 | (-0.0004, 0.01) | 0.07 |
| charfood | 0.17 | (0.08, 0.26) | <0.01 |
| age | -0.05 | (-0.13, 0.03) | 0.24 |
| BMI | 0.02 | (0.002, 0.04) | 0.03 |
| pyrene (air total) | 0.01 | (-0.003, 0.03) | 0.11 |
| **1-hydroxypyrene** | | | |
| pyrene (air total) | 0.03 | (0.01, 0.05) | 0.01 |
| charfood | 0.24 | (0.11, 0.37) | <0.01 |
| age | 0.001 | (-0.11, 0.11) | 0.99 |
| BMI | -0.01 | (-0.03, 0.01) | 0.38 |
| phenanthrene (air total) | 0.01 | (0.003, 0.02) | 0.01 |
| **1-hydroxypyrene (norm)** | | | |
| pyrene (air total) | 0.01 | (-0.01, 0.03) | 0.19 |
| charfood | 0.17 | (0.08, 0.26) | <0.01 |
| age | -0.03 | (-0.13, 0.07) | 0.57 |
| BMI | -0.01 | (-0.03, 0.02) | 0.63 |
| phenanthrene (air total) | 0.003 | (-0.004, 0.01) | 0.4 |

[¶]Results are differences in hydroxy-PAH levels (on the log scale) for a unit change in exposure

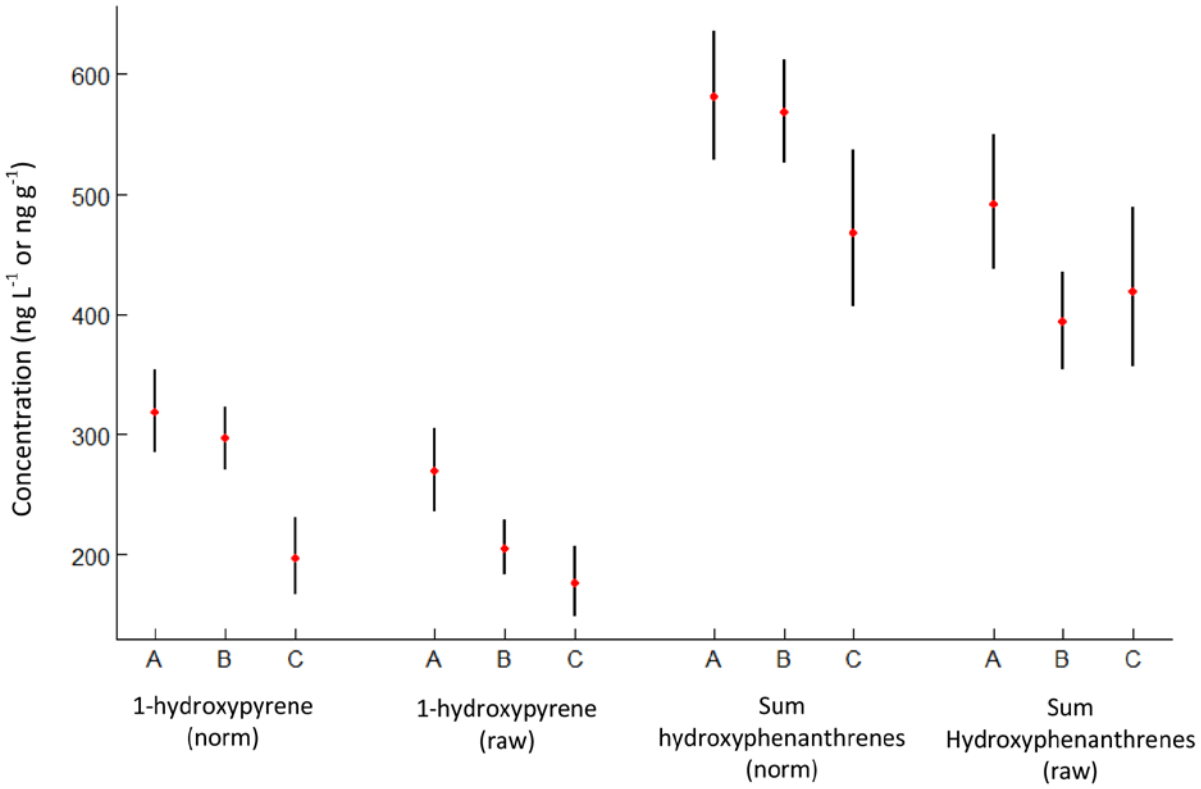


**Figure 1:** Geometric means (95% CI) for biomarker PAHs of the three sites A, B and C (sum of hydroxyphenanthrenes and 1-hydroxypyrene only).