Published in final edited form as:

Environ Int. 2016 March ; 88: 30–35. doi:10.1016/j.envint.2015.11.019.

Monitoring exposure to polycyclic aromatic hydrocarbons in an Australian population using pooled urine samples

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Abstract

Integrated exposure to polycyclic aromatic hydrocarbons (PAHs) can be assessed through monitoring of urinary mono-hydroxylated PAHs (OH-PAHs). The aim of this study was to provide the first assessment of exposure to PAHs in a large sample of the population in Queensland, Australia including exposure to infant (0–4 years). De-identified urine specimens, obtained from a pathology laboratory, were stratified by age and sex, and pooled (n = 24 pools of 100) and OH-PAHs were measured by gas chromatography-isotope dilution-tandem mass spectrometry. Geometric mean (GM) concentrations ranged from 30 ng/L (4-hydroxyphenanthrene) to 9221 ng/L (1-naphthol). GM of 1-hydroxypyrene, the most commonly used PAH exposure biomarker, was 142 ng/L. The concentrations of OH-PAHs found in this study are consistent with those in developed countries and lower than those in developing countries. We observed no association between sex and OH-PAH concentrations. However, we observed lower urinary concentrations of all OH-PAHs in samples from infants (0–4 years), children (5–14 years) and the elderly (>60 year old) compared with samples from other age groups (15–29, 30–44 and 45–59 years) which may be attributed to age-dependent behaviour-specific exposure sources.

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Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.envint.2015.11.019.

Keywords

OH-PAHs; Urinary metabolite; Biomonitoring; Infant; Exposure monitoring

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs), a class of hazardous pollutants, are produced predominantly during the incomplete combustion of organic materials naturally or by human activities (e.g., vehicular emissions, power plants, wood smoke, bush fires). Several PAHs have been classified as probable human carcinogens (IARC, 2010; Kim et al., 2013). Recent findings suggest a relationship between PAHs in placenta and the risk of neural tube defects, and with alteration of the immune system (Langlois et al., 2012; Walker et al., 2013). Other epidemiological studies also revealed associations between exposure to PAHs and childhood obesity and behavioural changes (Perera et al., 2014; Scinicariello and Buser, 2014).

Human exposure to PAHs may occur via inhalation, ingestion, and dermal absorption. The major sources of exposure to PAHs in non-occupational settings are smoking, grilled food and air pollution (Srogi, 2007). Biomonitoring provides an aggregate exposure estimate, integrating exposures from all sources and routes, including those that are hard to measure such as hand to mouth transfer in children (Sexton et al., 2004). Biomonitoring of PAH exposure has been carried out through the measurement of urinary mono-hydroxylated PAH (OH-PAH), a group of PAH metabolites (Jacob and Seidel, 2002), with 1-hydroxypyrene being the most commonly used PAH biomarker of exposure (Hansen et al., 2008).

There have been many biomonitoring studies using PAH metabolites for assessing PAH exposure, but these studies are typically limited to occupational settings, or specific sub-populations (Srogi, 2007). Fewer than 10 countries have conducted large-scale monitoring of PAH exposure in the general population (Bartolomé et al., 2015). Systematic monitoring of environmental pollutants in the general population provides the basis for future epidemiological studies to examine burden of disease or to provide evidence for the effectiveness of policies aiming at reducing chemical exposure (CDC, 2015; Health Canada, 2013; Toms et al., 2014; Toms et al., 2012). For example, US National Health and Nutrition Examination Survey (NHANES) data (2001–2006) for OH-PAHs have been used to investigate associations between exposure to PAHs and childhood obesity (Scinicariello and Buser, 2014). Although there have been some biomonitoring studies conducted in the general Australian population for other chemicals, including polyfluorinated alkyl substances and brominated flame retardants (Toms et al., 2014; Toms et al., 2012), to our knowledge there are no data on PAH exposure except for a small group of nine people (Thai et al., 2015).

While biomonitoring is regarded as the gold standard for exposure assessment (Sexton et al., 2004), measuring individual samples in general population studies involves substantial financial investment. Appropriately pooled biological samples can be used in cross-sectional studies to monitor temporal and spatial trends at the population level with considerably fewer resource requirements (Heffernan et al., 2014a; Toms et al., 2014). The aim of this study is to provide the first assessment of PAH exposure in a large subsection of the

Australian population through the quantification of OH-PAH metabolites in pooled urine samples.

2. Materials and methods

2.1. Study population, sample collection and pooling protocol

De-identified urine samples were obtained from a community-based pathology laboratory from surplus stored specimens that had been collected and analysed as part of routine clinical pathology testing in Queensland, Australia. Urine samples were collected in sterile polyethylene specimen containers, refrigerated for up to three days, and then frozen. Descriptive information about each sample included donor's sex, birthdate, and date of sample collection. Before pooling, samples were stratified by age (calculated from the birthdate and date of urine collection) and sex into six age strata: 0–4, 5–14, 15–29, 30–44, 45–59, and ≥60 years. The mean age of each pool was calculated from the average age of the individuals making up that pool. A total of 2400 individual samples were combined into 24 pools, with 100 individual samples contributing to each pool, with two replicate pools for each sex by age strata (Table 1). Samples were pooled based on volume, where each individual in the pool contributed the same volume (1 mL), thus the concentration measured in each pool is equivalent to the arithmetic mean of the concentration in each individual sample contributing to the pool (Caudill, 2010; Mary-Huard, 2007). To make the pools, individual urine samples were thawed, mixed well and aliquoted, after which the pooled sample was homogenised, divided into smaller aliquots and frozen until analysis. No measurements of creatinine or specific gravity were available. Sample collection and pooling occurred from November 2012 to November 2013. This work was approved by the University of Queensland ethics committee (approval number 2013000397). The involvement of the Centers for Disease Control and Prevention (CDC) laboratory (Atlanta, GA, USA) was determined not to constitute engagement in human subject research.

2.2. Urine analysis

Pooled urine aliquots were shipped on dry ice to the CDC and analysed for ten OH-PAHs using a modification of the gas chromatography–isotope dilution-tandem mass spectrometry method described previously (Li et al., 2014). Briefly, urine samples (1 mL) were spiked with ^{13}C-labelled internal standards and sodium acetate buffer containing β-glucuronidase/sulfatase to hydrolyse urinary conjugates overnight at 37 °C. The target analytes were then extracted through semi-automated liquid–liquid extraction using a Gilson 215 Liquid Handler (Gilson Inc., Middleton, WI, USA). The extracts were evaporated, and the target analytes were derivatised, separated, and quantified on an Agilent 7890 gas chromatograph coupled with an Agilent 7000B triple quadrupole mass spectrometer (Agilent Technologies, Santa Clara, CA, USA). The 10 OH-PAHs determined were metabolites of naphthalene, 1-naphthol (1-NAP) and 2-naphthol (2-NAP); of fluorene, 2-hydroxyfluorene (2-FLU), 3-hydroxyfluorene (3-FLU), 9-hydroxyfluorene (9-FLU); of phenanthrene, 1-hydroxyphenanthrene (1-PHE), 2-hydroxyphenanthrene (2-PHE), 3-hydroxyphenanthrene (3-PHE), 4-hydroxyphenanthrene (4-PHE); and of pyrene, 1-hydroxypyrene (1-PYR). The limits of detection were 44 ng/L for 1-NAP, 40 ng/L for 2-NAP, and 10 ng/L for the remaining OH-PAHs. As described elsewhere (Li et al., 2014), analyses were subject to a

series of quality control (QC) and quality assurance checks, including a multi-rule QC evaluation for each analytical run, and relative retention time and ^{13}C-internal standard spiking accuracy checks for each analyte in each sample.

3. Results

The 10 PAH metabolites measured were detected in all samples. Individual results are presented in Table 1. The concentrations varied between different OH-PAHs with geometric means (GM) ranging from 30 ng/L (4-PHE) to 9221 ng/L (1-NAP). GM mean concentration of 1-PYR, the most commonly used PAH exposure biomarker, was 142 ng/L.

For each metabolite the range of concentrations among age groups was relatively small, with the ratio of maximum to minimum concentrations ranging from 4 to 8, except for 1-NAP for which concentrations varied widely (924 to 375,182 ng/L). The concentration of 1-NAP in two pooled samples (Pools 15 and 19, Table 1) was two orders of magnitude higher than the GM (9221 ng/L). The highest concentrations of 1-NAP were detected in the 45–59 years age strata (7900–375,000 ng/L, Fig. 1A).

Representative plots of concentration versus age are shown for 1-NAP and 1-PYR in Fig. 1. In general, concentrations of OH-PAHs appeared to be higher in the 15–59 years adolescents and adult demographic groups, with lower concentrations in infants (0–4 years), children (5–14 years) and the elderly (>60 years). Age-concentration profiles for the remaining metabolites can be viewed in the supplementary material (Fig. S1). Visual inspection of the data suggests a non-linear relationship between urinary concentration and age for the studied PAH metabolites. There appears to be no significant differences in concentration of male and female pools.

The total concentration of OH-PAHs (ΣOH-PAHs — sum of all measured metabolites) among pools ranged from 4195 to 383,457 ng/L with the combined concentration of 1-NAP and 2-NAP accounting for 82–99% of ΣOH-PAHs value (Table 1). Contribution of metabolites from each parent PAH, e.g. 1-NAP and 2-NAP for naphthalene, to ΣOH-PAHs was naphthalene > fluorene > phenanthrene > pyrene, and was similar to the profile reported for the US population (Li et al., 2008; CDC, 2015).

4. Discussion

4.1. Concentration and age/sex trends

This study presents the first OH-PAH biomonitoring data for infants <3 years and together with the Canadian Health Measures Survey (CHMS) provides some of the first data on PAH exposures in young children (3–6 years) (Health Canada, 2013). Infants and young children are often excluded from biomonitoring campaigns due to logistical challenges with sample collection and recruitment. For example, NHANES in the USA only measured urinary OH-PAHs in participants > 6 years (CDC, 2015); other studies (e.g., in Korea) did not include infants and children (Sul et al., 2012).

Urinary concentrations of all OH-PAHs measured were lower in infants (0–4 years), children (5–14 years) and the elderly (>60 years) than in adolescent and adult(15–29; 30–44; 45–59

years) demographic groups (Fig. 1). Similar age-dependent profiles have been reported in CHMS II (Health Canada, 2013). The lower concentrations at these ages may reflect lower exposure to PAHs and thus potentially reduce the risk of adverse health effects among infants and the elderly, two population groups who are more susceptible than adults to negative impacts following pollutant exposure (Perera et al., 2014).

The non-linear relationship between OH-PAH concentration and age suggests adult-specific behaviours or exposure sources to PAHs that are not experienced by the other age groups. For example, smoking rates are higher in adolescents and adults than among the other age groups in Australia (ABS, 2013). Because smoking is a major source of PAH exposure among the non-occupationally exposed general population (Srogi, 2007), the different smoking rates by age groups may have contributed to the age-dependent profile of OH-PAH concentrations seen in this study. Regrettably, we could not assess the actual impact of smoking on the measured concentrations of OH-PAHs because measurement of urinary cotinine (nicotine's major metabolite) for individual specimens was unavailable.

We observed no association between sex and OH-PAH urinary concentrations. This finding is consistent with studies from Israel, Spain and the United States that reported no difference in OH-PHEs and OH-PYR among males and females (Levine et al., 2015; Bartolomé et al., 2015; CDC, 2015). However, in Korea, males had significantly higher OH-PAH urinary concentrations than females, likely due to higher incidence of smoking among Korean males (Sul et al., 2012). In Australia, the proportion of smokers among males (20.4%) and females (16.3%) is similar (ABS, 2013), and hence sex-trends with smoking status are unlikely to impact the relative distribution of OH-PAH concentrations between the two sexes.

4.2. Comparison with international data

With the exception of 1-NAP, the GM concentrations of urinary OH-PAH measured in this Australian population are comparable with those reported in other developed countries including the USA (CDC, 2015) and Canada (Health Canada, 2013), but are several times lower than in populations in developing countries such as Afghanistan, China, India, and Vietnam mainly for OH-PHEs (1, 2 and 3-PHE) and 1-PYR (Guo et al., 2013; Hemat et al., 2012) (Table 2). These differences are likely a reflection of lower exposure to PAHs in ambient air in Australia than in those relatively more polluted developing countries where emission of PAHs from industry and traffic contributes substantially to air pollution (Hopke et al., 2008).

4.2.1. Metabolite of pyrene—1-PYR is the most commonly used OH-PAH biomarker, and has been used to compare exposure to PAHs between occupational and non-occupational populations (Srogi, 2007; Hansen et al., 2008). Geometric mean concentrations of 1-PYR measured in this Australian population (142 ng/L) are comparable with those reported in other developed countries including the USA (111 ng/L, n = 2487) (CDC, 2015), Germany (140 ng/L, n = 963) (Wilhelm et al., 2008), Canada (110 ng/L, n = 2422) and Korea (150 ng/L, n = 4702) (Table 2), but considerably lower than those reported in developing countries such as China (378 ng/L, n = 84), India (424 ng/L, n = 38) and Vietnam (463 ng/L, n = 23) (Guo et al., 2013). Of interest, 1-PYR concentration in this study

is significantly lower (more than 10 times) than among people in developing countries who were regularly exposed to biomass fuel burning (Hemat et al., 2012; Li et al., 2011; Riojas-Rodriguez et al., 2011) or traffic pollution (Wertheim et al., 2012); concentrations of 1-hydroxypyrene were as high as 1646 ng/L for biomass burning (Hemat et al., 2012) or 1020 ng/g creatinine (~1000 ng/L) for traffic pollution (Wertheim et al., 2012).

4.2.2. Metabolites of naphthalene—Concentrations of 1-NAP varied widely—up to two orders of magnitude (Table 1). Of note, 10 of the 24 pooled samples had 1-NAP concentrations >20,000 ng/L; the highest concentration was 375,182 ng/L. Those values are near or higher than the 95th concentrations reported in CHMS II (15,000 ng/L, n = 2522) (Health Canada, 2013) and the US NHANES 2011–2012 (22,100 ng/L, n = 2492) (CDC, 2015). Unlike 1-NAP, the concentration profile of 2-NAP presented a narrow range of less than one order of magnitude. This profile is also seen for the other OH-PAH metabolites. The GM value of 2-NAP in this study (4100 ng/L) coincided with the value reported for the United States NHANES 2011–2012 survey cycle (4100 ng/L) (CDC, 2015). It is also comparable to the values reported in Canada (3830 ng/L, n = 2422) (Health Canada, 2013), Korea (3840 ng/L, n = 4702) (Sul et al., 2012), India (3780 ng/L, n = 38), Japan (3250 ng/L, n = 34) and Vietnam (4900 ng/L, n = 23) (Guo et al., 2013) (Table 2).

While urinary concentrations of 1-NAP and 2-NAP both arise from exposure to naphthalene, 1-NAP is also a major urinary metabolite of carbaryl (1-naphthyl-N-methylcarbamate), a broad-spectrum carbamate insecticide that can be used in domestic, gardening and agricultural settings, excluding fruits and vegetables in home gardens (APVMA, 2014). On the other hand, 2-NAP is a unique biomarker for naphthalene, and has been used in many studies to assess exposure to naphthalene. Meeker et al. (2007) suggested the use of 1-NAP/2-NAP ratio to evaluate the contribution of carbaryl exposure, where a ratio >2 would indicate sources other than naphthalene contributing to the 1-NAP urinary concentration. In this study, 11 pools had ratios of 1-NAP/2-NAP ranging from 2.26 to 16.04; ten of the pools also had 1-NAP concentrations >20,000 ng/L (Table 1). It is therefore probable that exposure to sources other than naphthalene contributed to the measured 1-NAP concentrations in some pools.

Interestingly, there are a few studies in the literature reporting similarly high ratios of 1-NAP/2-NAP. Zhang et al. (2014) report 1-NAP/2-NAP ratio in the range of 10–20 in a Chinese population. In that study, the average 1-NAP concentration in participants was >50,000 ng/L after ingestion of various food including rice, cabbage, braised chicken, pork chops, and grilled lamb; the average concentration of 2-NAP was 2000 ng/L in males and 5000 ng/L in females. No information is available about exposure to other chemicals but all participants lived and worked on campus at Peking University during the study (Zhang et al., 2014). High 1-NAP concentrations and 1-NAP/2-NAP ratios were also reported in a controlled dietary exposure study on excretion profile and half-lives of OH-PAHs among nine persons who consumed barbecued chicken (Li et al., 2012). The higher than expected 1-NAP/2-NAP ratios (91–442) in three participants, attributed to exposure to carbaryl, led to the recommendation of using 2-NAP, not 1-NAP, as the biomarker to naphthalene exposure in future biomonitoring studies (Li et al., 2012). Further, a commercially available Standard Reference Material for urinary OH-PAHs in non-smokers also had a 1-NAP/2-NAP ratio of

> 150, with the certified value of 1-NAP being 140 times higher than the GM value for US non-smokers (Schantz et al., 2015). To explain these findings the authors suggested that sources other than naphthalene (e.g., carbaryl) may have contributed to the urinary concentrations of 1-NAP. The poor correlation between 1-NAP and other OH-PAHs as shown in Table 3 provides further evidence that a non-PAH exposure source may have contributed to the relatively high 1-NAP urinary concentrations measured in this study.

4.2.3. Other PAH metabolites—In biomonitoring studies on PAH exposures, the metabolites of fluorene and phenanthrene are not measured as frequently as 1-PYR, 1-NAP and 2-NAP. Fluorene and phenanthrene metabolites were included in a number of population exposure studies including NHANES 2001–02 (for OH-PHEs) and NHANES 2003–04 (for OH-FLUs) in the United States (CDC, 2015), and later in the United Kingdom (Aquilina et al., 2010), Canada (Health Canada, 2013) and some Asian countries (Guo et al., 2013; Yang et al., 2015). Studies in Afghanistan, Israel, and Spain only included OH-PHEs (Bartolomé et al., 2015; Hemat et al., 2012; Levine et al., 2015).

The concentrations of OH-FLUs and OH-PHEs in the above-mentioned studies usually correlate with the 1-PYR concentration. Such correlations (OH-FLUs and OH-PHEs with 1-PYR) were demonstrated again in this study (Table 3), which supports the use of 1-PYR as a representative biomarker for human exposure to PAHs (Hansen et al., 2008; Wilhelm et al., 2008). Moreover, information on other OH-PAHs provides a broad assessment of the exposure to PAHs.

4.3. Limitations

The study population consisted of samples of convenience collected during the course of routine pathology testing and creatinine or specific gravity measures were not available. The samples are not statistically representative of the Australian population as a whole but of Queensland, where exposures to PAHs are likely to be similar to those of the general Australian population. Pooled pathology urine specimens have been used successfully in previous studies to measure urinary bisphenol A, another ubiquitous environmental chemical (Heffernan et al., 2013; Heffernan et al., 2014b), and a discussion of the opportunities and limitations of using pooled samples for biomonitoring has been published recently (Heffernan et al., 2014a).

5. Conclusions

This study provides the first data on PAH exposure of a large Australian population including infants. With the exception of 1-NAP, concentrations of urinary OH-PAHs in this study are comparable to the concentrations reported in similar populations from developed countries, and lower than those reported from selected developing countries. The relatively high urinary concentration of 1-NAP in some samples suggests that alternate exposure sources other than exposure to naphthalene also exist. While there were no differences in concentrations of OH-PAHs by sex, adolescent and adult groups (15–49 years) had higher urinary concentrations of OH-PAHs than the young and the elderly, most likely reflective of behaviour-specific exposure sources, such as smoking. This study can serve as a valuable reference for future studies to evaluate temporal trends in PAH exposures.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The authors wish to thank Soumini Vijayasarathy, Andrew Banks, Beatrix Fletcher, Nhung Dang and the staff at Sullivan Nicolaides Pathology Taringa for assistance with sample collection and pooling. We also gratefully acknowledge US CDC staff for technical assistance in chemical analysis and statistical analysis. PT is partly funded by a UQ Postdoctoral Fellowship and a QUT VC Research Fellowship. LMLT is partly funded by an ARC DECRA (DE120100161). JFM is funded by an ARC Future Fellowship. The authors would like to thank the Australian Government Department of the Environment for their financial support. The authors declare no conflict of interest. Entox is a joint venture of the University of Queensland and the Queensland Department of Health.

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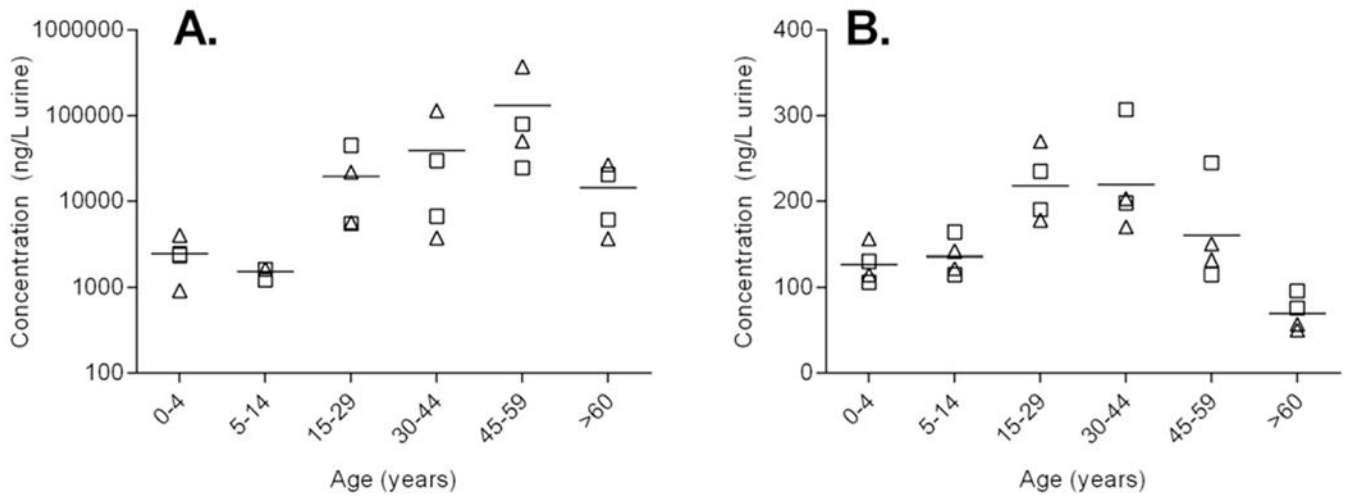

Fig. 1.

Urinary concentration versus age (years) for 1-NAP (A, ng/L) and 1-PYR (B, ng/L). Triangles denote female pools, squares denote male pools. Horizontal line indicates mean concentration of four pools in each age strata. Note log axis for 1-NAP.

Environ Int. Author manuscript; available in PMC 2019 December 30.

Table 1

Summary of pool characteristics and concentrations of OH-PAHs (ng/L) per strata. Each pool represents 100 individuals. Shaded rows are female pools.

Pool #	Age strata (years)	Av. Age (years)	Urinary concentration (ng/L)											
			1–NAP	2–NAP	2–FLU	3–FLU	9–FLU	1–PHE	2–PHE	3–PHE	4–PHE	1–PYR	1–NAP/2–NAP	ΣOH–PAHs
1	0–4	2.93	2365	2804	111	51	196	113	29	54	18	130	0.84	5871
2		2.74	2453	3233	119	54	140	82	26	48	15	106	0.76	6276
3		3.33	4002	2663	129	64	209	141	34	65	24	156	1.50	7487
4		3.24	924	2698	95	50	135	83	28	52	15	115	0.34	4195
5	5–14	8.83	1644	2838	185	89	239	123	53	82	21	164	0.58	5438
6		9.21	1227	3521	135	62	142	79	40	60	12	115	0.35	5393
7		8.74	1654	2456	140	59	204	97	45	63	15	142	0.67	4875
8		9.54	1615	3069	168	78	270	160	47	82	23	122	0.53	5634
9	15–29	24.3	5490	5957	463	261	302	127	82	113	30	190	0.92	13,015
10		24.0	45,308	5461	501	276	328	152	88	117	32	235	8.30	52,498
11		24.0	5683	8104	634	374	478	208	102	119	50	270	0.70	16,022
12		23.4	21,957	6035	347	164	332	131	78	80	36	178	3.64	29,338
13	30–44	37.8	30,136	6122	652	330	501	201	130	151	54	307	4.92	38,584
14		37.3	6660	7107	460	274	432	151	82	114	34	198	0.94	15,512
15		36.7	115,397	7720	513	276	439	157	81	92	47	203	14.95	124,925
16		36.8	3749	5192	390	234	312	147	68	86	39	170	0.72	10,387
17	45–59	52.9	79,452	4955	458	259	358	130	77	87	34	115	16.04	85,925
18		53.2	24,661	5702	555	334	770	183	113	151	50	245	4.33	32,764
19		53.3	375,182	6444	402	236	485	278	104	136	40	150	58.22	383,457
20		53.0	50,690	5673	478	253	570	159	83	101	65	132	8.94	58,204
21	≥60	73.7	20,562	3102	188	80	349	133	54	66	40	76	6.63	24,650
22		71.9	6077	2695	265	117	340	143	63	74	40	96	2.26	9910
23		75.1	26,736	2196	110	46	197	91	40	43	22	57	12.18	29,538
24		76.1	3677	2017	129	60	218	105	37	42	29	50	1.82	6364
Geometric mean			9221	4104	261	132	299	134	60	81	30	142		

Table 2

Concentrations of urinary OH-PAHs in selected populations (geometric mean or median) (ng/L).

	Australia[a]	US 2007–08[b]	US 2011–12[b]	Canada[c]	Korea[d]	Spain[e]	UK[f]	Germany[g]	Israel[h]	China[i]	Vietnam[i]	Japan[i]	India[i]	Malaysia[i]	Kuwait[i]	Afghanistan[j]
N	24 × 100	2581	2487	2422	4702	954	85	963	243	84	23	34	38	29	38	55
1-NAP	9221	2580	1670	1500	n/a	n/a	n/a	n/a	n/a	528	642	266	1110	263	1410	
2-NAP	4104	3830	4140	3800	3840	n/a	n/a	n/a	n/a	2270	4900	3250	3780	1550	7330	
2-FLU	261	303	240	270	n/a	n/a	170	n/a	n/a	893	473	207	346	112	448	
3-FLU	132	116	93.6	96	n/a	n/a	280	n/a	n/a							
9-FLU	299	337	245	160	n/a	n/a	180	n/a	n/a							
1-PHE	134	139	126	150	n/a	200	220	n/a	190	323	157	65	256	36	89	1310
2- PHE	60	63.6	61	67	n/a		140	n/a	95	387	201	121	289	43	137	
3- PHE	81	97.6	62	97	n/a		220	n/a	130	217	279	77	154	33	163	1147
4- PHE	30	29.3	21	25	n/a		n/a	n/a	29	33	32	10	27	6	0	119
1-PYR	142	118	111	110	150	180	n/a	140	172	378	463	75	424	65	220	1646

[a] This study.

[b] CDC (2015).

[c] Health Canada (2013).

[d] Sul et al. (2012).

[e] Bartolomé et al. (2015).

[f] Aquilina et al. (2010).

[g] Wilhelm et al. (2008).

[h] Levine et al. (2015).

[i] Guo et al. (2013).

[j] Hemat et al. (2012).

Table 3

Correlation coefficients (*r*) among urinary concentrations of OH-PAHs.

	2-NAP	2-FLU	3-FLU	9-FLU	1-PHE	2-PHE	3-PHE	4-PHE	1-PYR
1-NAP	0.366	0.239	0.252	0.341	**0.689**	0.388	0.396	0.277	0.077
2-NAP		**0.903** *	**0.912**	**0.703**	**0.662**	**0.827**	**0.782**	**0.683**	**0.750**
2-FLU			**0.991**	**0.822**	**0.691**	**0.946**	**0.889**	**0.813**	**0.807**
3-FLU				**0.822**	**0.697**	**0.928**	**0.891**	**0.782**	**0.800**
9-FLU					**0.748**	**0.868**	**0.843**	**0.881**	**0.592**
1-PHE						**0.796**	**0.818**	**0.692**	**0.569**
2-PHE							**0.950**	**0.805**	**0.781**
3-PHE								**0.692**	**0.826**
4-PHE									0.493

*The numbers in bold indicate statistical significance ($p < 0.001$).