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## ENVIRONMENTAL CHEMISTRY, POLLUTION & WASTE MANAGEMENT | RESEARCH ARTICLE

# Trends and concentrations of selected polycyclic aromatic hydrocarbons in general US population: Data from NHANES 2003–2008

Ram B. Jain<sup>1,2\*</sup>

**Abstract:** Polycyclic aromatic hydrocarbons (PAH) are potentially mutagenic and carcinogenic, and as such their exposure is of serious concern. I aimed to study the trends in the levels of selected PAHs in US for the period 2003–2008 and their distribution by gender, race/ethnicity, socioeconomic and smoking status, and by exposure to second-hand smoke (SHS) at home and work. Using data from the National Health and Nutrition Examination Survey, regression models were fitted for 10 individual urinary PAH metabolites. Smoking was statistically significantly associated with higher levels of naphthalene, fluorene, phenanthrene, and pyrene metabolites when compared to non-smokers. SHS exposure at home was also statistically significantly associated with higher concentrations of naphthalene, fluorene, phenanthrene, and pyrene metabolites. There was a statistically significant increase in the concentrations of total naphthalene and fluorene, 2-hydroxynaphthalene, 2- and 9-hydroxyfluorene, 2- and 4-hydroxyphenanthrene, and 1-hydroxypyrene during the study period of 2003–2008. Females were found to have statistically significantly higher concentrations of total naphthalene and phenanthrene metabolites as well as 1- and 2-hydroxynaphthalene, 1-hydroxyphenanthrene, and 1-hydroxypyrene. For most of the PAH metabolites, non-Hispanic whites had the highest adjusted concentrations and Mexican Americans had the lowest adjusted concentrations. For the concentrations of 2-hydroxynaphthalene, however, the reverse was true.

### ABOUT THE AUTHORS

Ram B. Jain has been involved in environmental research since 2002. His current interests include impact of pregnancy on the concentration of environmental contaminants, impact of environmental contaminants on the thyroid function, and impact of exposure to second-hand smoke on the levels of environmental contaminants. Recently, he has published in such journals as *Environmental Toxicology and Pharmacology*, *Journal of Trace Elements in Medicine and Biology*, *Environmental Research*, and *International Journal of Environmental Health Research*. He is currently a private consultant and continues doing environmental research in his spare time.

### PUBLIC INTEREST STATEMENT

Human organ systems that are adversely affected by the exposure to polycyclic aromatic hydrocarbons (PAHs) include dermal, hepatic, and immunological. Humans can be exposed to PAHs during smoking, grilling, broiling, or other high temperature food processing. Tobacco smoke has also been shown to have PAHs as one of its constituents. This communication has shown how smoking, both mainstream and second-hand, can affect exposure to PAHs. In addition, the factors that affect PAH concentration levels have also been delineated. Finally, the change in the levels of PAHs during the period 2003–2008 has been investigated.

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**Subjects: Environment & Health; Research Methods in Environmental Studies; Statistics & Probability**

**Keywords: naphthalene; fluorene; phenanthrene; pyrene; smoking; NHANES**

### 1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) are a group of more than 100 chemicals usually produced during incomplete combustion of organic materials. Some of the many sources of PAHs are motor vehicle exhaust, residential and industrial heating sources, coal, crude oil and natural gas processing, waste incineration, and tobacco smoke. Organ systems affected by exposure to PAHs include dermal, hepatic, and immunologic (ATSDR, 2014). Smoking, grilling, broiling, or other high temperature processing can result in the formation of PAHs. The emitted PAHs can form or bind to particles in the air, and particle size depends in part on the source of the PAHs (CDC, 2013). Relatively higher concentrations of PAHs are associated with smaller particulates (Boström et al., 2002; Rehwagen, Müller, Massolo, Herbarth, & Ronco, 2005). Uncooked foods and vegetables generally contain low concentrations of PAHs but can be contaminated by airborne particle deposition (CDC, 2013). Some leafy vegetables, may, however, have relatively higher concentrations of PAHs (Li, Li, et al., 2008).

Typically, urinary concentrations of 1- and 2-hydroxynaphthalene among smokers are about 2 to 3 times higher than non-smokers in both occupationally exposed and general populations (Campo et al., 2006; Nan et al., 2001; Serdar, Egeghy, Waidyanatha, Gibson, & Rappaport, 2003; Serdar, Waidyanatha, Zheng, & Rappaport, 2003). Serdar, Waidyanatha, et al. (2003) showed that interactions between cigarette smoking and PAH exposure affects metabolism of individual PAHs.

Children aged 6–15 years old exposed to higher concentrations of fluorene metabolites had a twofold odds of having been enrolled in special education (Abid, Herbstman, & Ettinger, 2014). Total urinary PAH metabolites and naphthalene metabolites are associated with higher body mass index, waist circumference, and obesity in children 6–11 years of age (Scinicariello & Buser, 2014). Everett et al. (2010) analyzed association between serum C-reactive protein and nine urinary PAH metabolites. They suggested a role for monohydroxy PAHs in progression of atherosclerosis. However, after controlling for tobacco use, Clark et al. (2012) did not find an association between PAH exposure and serum biomarkers of cardiovascular disease. Huang, Caudill, Grainger, Needham, and Patterson (2006) found children aged 6–11 years to have higher concentrations of 1-hydroxypyrene than adolescents aged 12–19 years and adults. Smokers and persons exposed to second-hand smoke (SHS) were also found to have higher concentrations of 1-hydroxypyrene than non-smokers.

A limited number of studies have evaluated the association between SHS exposure and PAH metabolite concentrations. Scherer, Frank, Riedel, Meger-Kossien, and Renner (2000), in a field study of 69 subjects, evaluated the influence of smoking, diet, SHS, and location of residence on urinary excretion of 1-hydroxypyrene and benzo[a]pyrene. They found that diet and smoking were the major sources of PAH exposure for persons not occupationally exposed to PAH but the influence of SHS exposure was negligible. Suwan-ampai, Navas-Acien, Strickland, and Agnew (2009) evaluated the impact of active and involuntary smoking on the concentrations of 23 PAHs using data from National Health and Nutrition Examination Survey (NHANES) for the years 1999–2002. Involuntary smoking was found to be associated with 1-hydroxypyrene, 2-hydroxyfluorene, 3-hydroxyfluorene, 9-hydroxyfluorene, 1-hydroxyphenanthrene, 2-hydroxyphenanthrene, and 3-hydroxyphenanthrene.

Relatively higher concentrations of PAHs have been reported in high-density traffic areas (Fischer et al., 2000; Tuntaviroon, Mahidol, Navasumrit, Autrup, & Ruchirawat, 2007). Tuntaviroon et al. (2007) reported total PAH concentrations to be 30-fold higher in high-density traffic areas of Bangkok as compared to roadsides in proximity to the low-density traffic areas near provincial schools. Outdoor concentration of total PAH was reported to be substantially higher (Fischer et al., 2000) in high intensity streets of Amsterdam as compared to low traffic intensity streets.

While there have been studies that have assessed the association of PAH concentrations with a limited number of factors, for example, body mass index (Scinicariello & Buser, 2014), a study that has evaluated association between PAH concentrations with factors like gender, race/ethnicity, and SHS exposure while controlling for the effects of other factors, using recent data are lacking. Consequently, this study was undertaken to assess the effect of various factors that may affect concentrations of PAH in US adults aged 20 years and over. Also, an important objective was to assess the trends in the concentrations of PAH over time. A study of trends over time is necessary to estimate the variability in adverse health risks associated with exposure to PAHs. This can help public health professionals estimate the resources that may be needed to provide general public information necessary to protect themselves from unnecessary exposure to PAHs. Data from National Health and Nutrition Examination Survey (NHANES) for the years 2003–2008 were selected for this purpose.

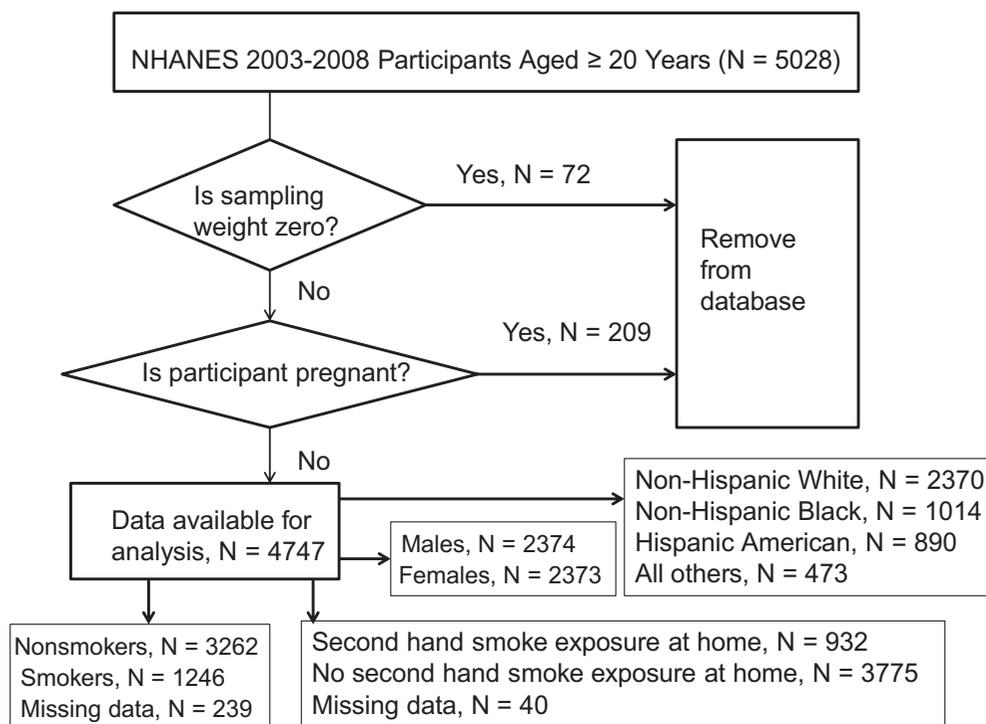
## 2. Materials and methods

Data from NHANES ([www.cdc.gov/nchs/nhanes.htm](http://www.cdc.gov/nchs/nhanes.htm)) for the years 2003–2008 for demographics, PAH metabolites, body measures, serum cotinine, urine creatinine, and family smoking and occupational questionnaire data files were downloaded and match merged. The function of match merge is to assemble all data on each specific NHANES participant in one data file for the purpose of analysis. For example, gender of the participant XYZ which is provided in the data file on demographics need to be matched with his/her observed concentrations for PAH metabolites which are provided in a different data file. NHANES provides a unique participant ID, labeled as SEQN for each NHANES participant. SEQN was used to match and merge data from different data files. The sampling plan for NHANES is a complex, stratified, multistage, probability cluster designed to be representative of the civilian, non-institutionalized US population. NHANES provides sampling weights to account for the complex survey design, including oversampling, survey non-response, and post-stratification. All analyses incorporated information on sampling design variables.

For PAH, data for 10 metabolites were available in urine samples, namely, 1-hydroxynaphthalene, 2-hydroxynaphthalene, 2-hydroxyfluorene, 3-hydroxyfluorene, 9-hydroxyfluorene, 1-hydroxyphenanthrene, 2-hydroxyphenanthrene, 3-hydroxyphenanthrene, 4-hydroxyphenanthrene, and 1-hydroxypyrene. However, for 4-hydroxyphenanthrene, data were available for years 2003–2006 only. In addition, three aggregated variables, namely,  $\Sigma\text{NAP}$  = 1-hydroxynaphthalene + 2-hydroxynaphthalene,  $\Sigma\text{FLU}$  = 2-hydroxyfluorene + 3-hydroxyfluorene + 9-hydroxyfluorene, and  $\Sigma\text{PHE}$  = 1-hydroxyphenanthrene + 2-hydroxyphenanthrene + 3-hydroxyphenanthrene + 4-hydroxyphenanthrene, presenting total concentrations of naphthalene, fluorene, and phenanthrene metabolites, respectively, were also generated. Percent-weighted observations at or above the limit of detection for all 10 PAH metabolites were close to 100% and as such, all of them were selected for further analysis. All observations below the limit of detection (LOD) were set at  $\text{LOD}/\sqrt{2}$ . Laboratory methods used to detect and measure various PAH metabolites for the years 2007–2008 are available at [http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/PAH\\_E.htm#Description\\_of\\_Laboratory\\_Methodology](http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/PAH_E.htm#Description_of_Laboratory_Methodology).

In a household smoking questionnaire ([http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/SMQFAM\\_E.htm](http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/SMQFAM_E.htm)) administered at the time of the home interview, questions were asked if somebody smoked at home, and if so, how many persons smoked cigarettes inside home and how many cigarettes were smoked inside home every day (CPD\_Home). Using the data from this questionnaire, a categorical variable, presence and absence of SHS exposure at home (HmE) was created. Based on the results of preliminary analysis, only CPD\_Home was selected for analysis for this research. For those who had no HmE, CPD\_Home was set at zero. Using an occupational questionnaire ([http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/OCQ\\_E.htm](http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/OCQ_E.htm)), the respondent was asked if the NHANES participant was working at a job or business, and if so, how many hours per day, tobacco smoke was inhaled from other people's cigarettes, cigars, or pipes. Using the responses to the questions, if participants inhaled tobacco smoke at work, an indicator variable representing SHS exposure at work (WkE) was created. Number of hours smoke was inhaled per day from other people's cigarettes, cigars, or pipes (Hours\_Smk) was the continuous variable available for use. For this study, smokers were defined as those who had serum cotinine concentrations  $\geq 10$  ng/mL and non-smokers were defined as those who had

**Figure 1. Sample selection process for data analysis and final sample distribution by gender, race/ethnicity, smoking status, and exposure to second-hand smoke at home.**



serum cotinine concentrations < 10 ng/mL. A consideration was given to use WkE as a categorical variable but it would have increased the number of data cells from 32 to 64 possibly resulting in certain number of empty data cells and thus, negatively affecting the stability and reliability of statistical estimates and decreasing the number of degrees of freedom available for error terms. In the interest of having stable and reliable estimates, a decision was made to use WkE as an indicator variable.

A total of 5,028 NHANES participants aged 20 years and older were available for analysis. There were 72 participants for whom sampling weight was recorded as zero in the NHANES database. These 72 participants were removed from the database. There were 209 females who were pregnant at the time of NHANES participation. Since pregnancy may affect PAH concentrations, these 209 participants were removed from the database for this study leaving a sample size of 4,747 available for analysis (Figure 1). Data were available for 2,374 males, 2,373 females; 2,370 non-Hispanic whites (NHW), 1,014 non-Hispanic blacks (NHB), 890 Mexican Americans (MA), 473 participants for whom race/ethnicity was not classified (OTH); 3,262 non-smokers, 1,246 smokers; 932 participants who were exposed to SHS at home and 3,775 participants who were not exposed to SHS at home (Figure 1). Data were missing for 239 participants for smoking status and for 40 participants for SHS exposure status at home (Figure 1). However, actual sample size used in the analyses was much smaller because of missing values for dependent and independent variables. General characteristics of the study population are given in Table 1.

The independent variables considered for regression modeling were age as a continuous variable, gender (males, females), race/ethnicity (NHW, NHB, MA, OTH), smoking status (nonsmoker, smoker), body mass index (BMI), HmE, WkE, CPD\_Home, and Hours\_Smk. It is important to consider the association of BMI with the concentration concentrations of PAHs because PAHs are transported to all tissues of the human body containing fat and are strongly lipophilic (Scinicariello & Buser, 2014). In addition, since a positive association between obesity (BMI ≥ 30 kg/m<sup>2</sup>) and PAHs has been previously reported among children (Scinicariello & Buser, 2014), it is possible that an association may be found among adults too. Other reasons to include BMI as a covariate may be increased caloric intake and increased volume of distribution possibly resulting in lower concentrations of target chemicals. Family poverty income ratio (PIR) was used as a surrogate variable for socioeconomic status. PIR

**Table 1. General characteristics of the study population**

Study characteristic	Sample size		Range	Mean
	N	%		
Age in years	4,747	100.0	20–85	50.4
Body mass index in kg/m <sup>2</sup>	4,747	100.0	13.4–130.2	28.8
Urine creatinine	4,747	100.0	8–882	129.1
Poverty Income Ratio	4,747	100.0	0–5	2.6
Number of cigarettes smoked at home per day	4,747	100.0	0–40	3.1
Number of hours per day tobacco smoke inhaled at work	4,747	100.0	0–16	0.4
Gender				
Males	2,374	50.0		
Females	2,373	50.0		
Race/Ethnicity				
Non-Hispanic white	2,370	49.9		
Non-Hispanic black	1,014	21.4		
Mexican American	890	18.7		
All others	473	10.0		
Smoking status				
Non-smoker	3,262	68.7		
Smoker	1,246	26.2		
Missing data	239	5.0		
Second hand smoke exposure at home				
Exposed	932	19.6		
Not exposed	3,775	79.5		
Missing data	40	0.8		
Second-hand smoke exposure at work				
Exposed	507	10.7		
Not exposed	4,240	89.3		

Source: Data from National Health and Nutrition Examination Survey 2003–2008.

should be expected to have association with the concentrations of PAHs because of the possible association of PIR with higher smoking levels and/or higher levels of coal burning for heating. Data for PAH metabolites were log<sub>10</sub> transformed before being used as dependent variables in the regression models. A total of 13 regression models were fitted.

A consideration was given to use consumption of broiled/smoked meat/fish as an independent variable as has been used by Suwan-ampai et al. (2009). I identified those participants from the 24-h recall dietary databases for NHANES years 2003–2008 ([http://www.cdc.gov/nchs/nhanes/nhanes2005-2006/DR1IFF\\_D.htm](http://www.cdc.gov/nchs/nhanes/nhanes2005-2006/DR1IFF_D.htm)) who specifically were reported to have eaten broiled and/or smoked food items. However, in certain cases, it was not possible to determine if the food that was eaten was broiled or baked. For example, for the United States Department of Agricultural (USDA) food code database, the description provided in the NHANES 24-h recall dietary database for USDA food code 21101120 was “BEEF STEAK, BROILED OR BAKED, LEAN & FAT”. All such food items were not considered to be broiled or smoked. I identified a total of 37 participants who specifically reported having consumed broiled/smoked food items. Since non-availability of complete data did not allow creation of a variable representing consumption of broiled/smoked food, these 37 participants were removed from the analysis.

All data were analyzed using SAS 9.2 ([www.sas.com](http://www.sas.com)) and SUDAAN 11.0 ([www.rti.org/sudaan](http://www.rti.org/sudaan)). SUDAAN Proc DESCRIPT was used to compute unadjusted geometric means and SUDAAN Proc REGRESS was used to fit regression models and to compute adjusted geometric means. It should be

noted that unadjusted geometric means are simply the means of the log-transformed PAH variables, for example, for males irrespective of how these unadjusted geometric means may be affected by other factors like race/ethnicity. On the other hand, adjusted geometric means are means of log-transformed variables after mathematical adjustments have been made for the contribution of other variables in the regression models. Two-way interactions between gender, race/ethnicity, HmE, and smoking status were also considered but were retained in the final model only if one or more of them were statistically significant at  $\alpha = 0.05$ . An interaction term between HmE and smoking status was considered because of the possibility that HmE may affect PAH metabolite concentrations differently among smokers and non-smokers, and retained in the final model if the interaction was found to be statistically significant at  $\alpha = 0.05$ .

### 3. Results

In the interest of journal space, results for individual naphthalene, fluorene, and phenanthrene metabolites are presented as supplemental material. Specifically, regression coefficients with associated *p*-values are presented in Table S1 and adjusted geometric means (AGM) in Table S2. Results for  $\Sigma$ NAP,  $\Sigma$ FLU,  $\Sigma$ PHE, and 1-hydroxypyrene are presented here.

Actual sample sizes used in regression models for  $\Sigma$ NAP,  $\Sigma$ FLU,  $\Sigma$ PHE, and 1-hydroxypyrene were 3,804, 3,821, 2,133, and 3,858, respectively. *R*<sup>2</sup> for the models for  $\Sigma$ NAP,  $\Sigma$ FLU,  $\Sigma$ PHE, and 1-hydroxypyrene was 39.5, 63.3, 45.5, and 50.2%, respectively. No statistically significant interactions were found for the models for  $\Sigma$ NAP and  $\Sigma$ PHE. For the model for  $\Sigma$ FLU, statistically significant interactions were found between gender and smoking status (*p* < 0.01), and between smoking status and HmE (*p* < 0.01). For the model for 1-hydroxypyrene, statistically significant interactions were found between gender and race/ethnicity (*p* < 0.01), and between smoking status and HmE (*p* = 0.03).

Unadjusted geometric means (UGM) with 95% confidence intervals based on unweighted data for  $\Sigma$ NAP,  $\Sigma$ FLU,  $\Sigma$ PHE, and 1-hydroxypyrene by survey period, gender, and race/ethnicity are given in Table 2. While no formal statistical tests were conducted, it is quite obvious that the levels of  $\Sigma$ NAP,

**Table 2. Unadjusted geometric means with 95% confidence interval for selected polycyclic aromatic hydrocarbons (PAH) by survey year, gender, and race/ethnicity**

Survey year	PAH (ng/L)			
	$\Sigma$ NAP*	$\Sigma$ FLU**	$\Sigma$ PHE***	1-hydroxypyrene
2003–2008	8,022.2 (7,714.3–8,342.3)	841.0 (812.3–870.8)	348.1 (338.1–358.5)	94.3 (91.1–97.6)
2003–2004	7,232.2 (6,764.3–7,732.5)	782.6 (733.8–834.6)	356.6 (338.6–375.5)	79.6 (74.7–84.9)
2005–2006	9,045.5 (8,345.7–9,804.0)	900.8 (845.2–960.0)	359.9 (339.2–381.8)	95.6 (74.6–122.4)
2007–2008	7,996.1 (7,542.7–8,476.9)	846.0 (801.4–893.0)	334.7 (320.2–350.0)	106.8 (101.4–112.5)
Gender				
Males	7,468.9 (7,041.2–7,922.5)	1,054.2 (1,005.5–1,105.3)	346.1 (331.9–360.9)	111.2 (106.0–116.7)
Females	8,623.5 (8,193.0–9,076.6)	669.9 (637.7–703.7)	350.2 (336.1–364.8)	79.9 (76.0–83.9)
Race/Ethnicity				
Non-Hispanic white	7,477.8 (7,053.0–7,928.3)	831.5 (790.4–874.7)	348.7 (334.3–363.7)	88.1 (83.7–92.6)
Non-Hispanic black	11,229.3 (10,326.0–12,211.7)	1,231.4 (1,143.6–1,325.9)	431.6 (405.8–459.1)	115.6 (107.1–124.8)
Mexican Americans	6,930.4 (6,412.2–7,490.5)	623.4 (581.6–668.2)	285.9 (269.4–303.5)	88.93 (82.7–95.6)
All others	7,222.8 (6,459.6–8,076.3)	693.0 (624.4–769.0)	307.7 (281.8–335.9)	95.1 (86.0–105.2)

\* $\Sigma$ NAP = 1-hydroxynaphthalene + 2-hydroxynaphthalene.

\*\* $\Sigma$ FLU = 2-hydroxyfluorene + 3-hydroxyfluorene + 9-hydroxyfluorene.

\*\*\* $\Sigma$ PHE = 1-hydroxyphenanthrene + 2-hydroxyphenanthrene + 3-hydroxyphenanthrene + 4-hydroxyphenanthrene for 2003–2006,  $\Sigma$ PHE = 1-hydroxyphenanthrene + 2-hydroxyphenanthrene + 3-hydroxyphenanthrene for 2007–2008.

Source: Data from National Health and Nutrition Examination Survey 2003–2008.

$\Sigma$ FLU, and 1-hydroxypyrene were substantially higher during 2007–2008 than during 2003–2004. For example, for  $\Sigma$ FLU, while UGM for 2003–2004 was 782.6 ng/L, for 2007–2008, it was 846 ng/L for about 9% increase. Males had higher UGM for  $\Sigma$ FLU (1,054.2 vs. 669.9 ng/L) and 1-hydroxypyrene (111.2 vs. 79.9 ng/L) than females and females had higher UGM than males for  $\Sigma$ NAP (8,623.5 vs. 7,468.9 ng/L). Among the three major racial/ethnic groups, the order in which UGMs for  $\Sigma$ NAP,  $\Sigma$ FLU, and 1-hydroxypyrene were observed was: NHB > NHW > MA.

Age was positively associated with the concentrations of  $\Sigma$ NAP (Table 3,  $\beta = 0.0021$ ,  $p = 0.001$ ) and  $\Sigma$ PHE ( $\beta = 0.0013$ ,  $p = 0.018$ ) but negatively associated with the concentrations of 1-hydroxypyrene ( $\beta = -0.0029$ ,  $p < 0.001$ ). However, the concentrations of 2-hydroxynaphthalene were not associated with age ( $\beta = 0.0003$ ,  $p = 0.522$ , Table S1) and the concentrations of 2-hydroxyphenanthrene as well as 4-hydroxyphenanthrene were also not associated with age ( $p \geq 0.389$ , Table S1). On the other hand, while the concentrations of  $\Sigma$ FLU were not associated with age ( $\beta = 0.0005$ ,  $p = 0.127$ , Table 3), the concentrations of 3-hydroxyfluorene were negatively associated with age ( $\beta = -0.0011$ ,  $p = 0.001$ , Table S1), and the concentrations of 9-hydroxyfluorene were positively associated with age ( $\beta = 0.0017$ ,  $p < 0.001$ , Table S1).

BMI was positively associated with the concentrations of  $\Sigma$ FLU (Table 3,  $\beta = 0.0043$ ,  $p < 0.001$ ). However, the concentrations of 3-hydroxyfluorene had a statistically insignificant negative association with BMI ( $\beta = -0.0002$ ,  $p = 0.869$ , Table S1). Statistically significant negative association of the concentrations of 1-hydroxynaphthalene ( $\beta = -0.0038$ ,  $p = 0.046$ ) with BMI in combination with statistically significant positive association of the concentrations of 2-hydroxynaphthalene with BMI ( $\beta = 0.0005$ ,  $p < 0.001$ , Table S1) resulted in statistically insignificant association of the concentrations of  $\Sigma$ NAP with BMI ( $\beta = 0.0022$ ,  $p = 0.137$ ). Similarly, while the concentrations of  $\Sigma$ PHE did not show any association with BMI ( $\beta = 0.0023$ ,  $p = 0.092$ , Table 3), the concentrations of 1-hydroxyphenanthrene ( $\beta = 0.0025$ ,  $p = 0.01$ , Table S1), 2-hydroxyphenanthrene ( $\beta = 0.007$ ,  $p < 0.01$ , Table S1), and 4-hydroxyphenanthrene ( $\beta = 0.0063$ ,  $p = 0.001$ , Table S1) had statistically significant positive associations with BMI.

Concentrations of  $\Sigma$ NAP ( $p < 0.001$ ),  $\Sigma$ FLU ( $p < 0.001$ ), and 1-hydroxypyrene ( $p < 0.001$ ) increased during the study period of 2003 to 2008. On the individual metabolite concentrations, 2-hydroxynaphthalene, 2-hydroxyfluorene, 9-hydroxyfluorene, 2-hydroxyphenanthrene, and

**Table 3. Regression slopes ( $\beta$ ) for continuous variables with associated  $p$ -values for selected polycyclic aromatic hydrocarbon metabolites used in the analysis for participants aged  $\geq 20$  years**

	$\beta$ ( $p$ -value) for			
	$\Sigma$ NAP	$\Sigma$ FLU	$\Sigma$ PHE	1-hydroxypyrene
Age	0.0021 (0.001)	0.0005 (0.127)	0.0013 (0.018)	-0.0029 (<0.001)
Body mass index	0.0022 (0.137)	0.0043 (<0.001)	0.0023 (0.092)	0.0009 (0.37)
NHANES cycle	0.0515 (0.001)	0.0369 (<0.001)	0.0264 (0.26)	0.0929 (<0.001)
Urine creatinine	0.0032 (<0.001)	0.0033 (<0.001)	0.0032 (<0.001)	0.0034 (<0.001)
Poverty income ratio	0.0031 (0.677)	-0.0123 (0.004)	-0.0036 (0.485)	-0.0184 (<0.001)
Exposed to SHS at work	-0.0896 (0.012)	-0.0076 (0.776)	-0.0107 (0.748)	0.0389 (0.135)
Number of cigarettes smoked at home per day	0.0031 (0.003)	0.003 (0.001)	0.0033 (0.002)	0.0024 (0.036)
Number of hours inhaling tobacco smoke at work	0.0181 (0.005)	-0.0005 (0.911)	-0.0003 (0.956)	-0.0059 (0.288)

Source: Data from National Health and Nutrition Examination Survey 2003–2008.

4-hydroxyphenanthrene had similar statistically significant increases over the study period of 2003–2008 (Table S1).

Concentrations of  $\Sigma$ NAP,  $\Sigma$ FLU,  $\Sigma$ PHE, and 1-hydroxypyrene were positively associated with urine creatinine concentrations (Table 3). Statistically significant positive associations were also observed between urine creatinine and every metabolite of naphthalene, fluorene, and phenanthrene (Table S1).

Lower PIR was associated with increasing concentrations of  $\Sigma$ FLU ( $\beta = -0.0123$ ,  $p = 0.004$ , Table 3), and 1-hydroxypyrene ( $\beta = -0.0184$ ,  $p < 0.001$ , Table 3). Similar results were observed for every metabolite of naphthalene, fluorene, and phenanthrene though associations were not always statistically significant (Table S1).

Exposure to SHS at work was associated with decreased concentrations of  $\Sigma$ NAP ( $\beta = -0.0896$ ,  $p = 0.012$ , Table 3). On an individual metabolite level, 1-hydroxynaphthalene was the only metabolite for which statistically significant negative association with SHS at work was observed ( $\beta = -0.1324$ ,  $p = 0.002$ , Table S1). Concentrations of  $\Sigma$ NAP increased with an increase in number of hours smoke was inhaled at work from other people's cigarettes, cigars, and pipes (Table 3,  $\beta = 0.0181$ ,  $p = 0.005$ ). These results were also observed for both 1-hydroxynaphthalene ( $\beta = 0.0159$ ,  $p = 0.004$ , Table S1) and 2-hydroxynaphthalene ( $\beta = 0.018$ ,  $p = 0.01$ , Table S1). However, number of hours smoke was inhaled at work from other people's cigarettes, cigars, and pipes was negatively associated with the concentrations of 2-hydroxyphenanthrene ( $\beta = -0.0093$ ,  $p = 0.038$ , Table S1).

CPD\_Home or the number of cigarettes smoked inside home every day was associated with increased concentrations of  $\Sigma$ NAP ( $\beta = 0.0031$ ,  $p = 0.003$ , Table 3),  $\Sigma$ FLU ( $\beta = 0.003$ ,  $p = 0.001$ , Table 3),  $\Sigma$ PHE ( $\beta = 0.0033$ ,  $p = 0.002$ , Table 3), and 1-hydroxypyrene ( $\beta = 0.0024$ ,  $p = 0.036$ , Table 3). CPD\_Home had statistically significant positive associations with almost every metabolite of naphthalene, fluorene, and phenanthrene (Table S1).

Females had statistically significantly higher adjusted geometric means (AGM) for  $\Sigma$ NAP (8,901 ng/L vs. 7,146.2 ng/L for a difference of about 25%, Table 4,  $p < 0.01$ ),  $\Sigma$ PHE (392.8 ng/L vs. 364 ng/L, for a difference of about 8%,  $p = 0.04$ ), and 1-hydroxypyrene (98.4 ng/L vs. 92.3 ng/L,  $p = 0.049$ ) than males. In fact, females had higher concentrations of every metabolite of naphthalene, fluorene, and phenanthrene except 2-hydroxyphenanthrene and 3-hydroxyphenanthrene (Table S2). Statistically significant differences were observed for 1-hydroxynaphthalene (3,249 vs. 2,939 ng/L,  $p < 0.01$ ,

**Table 4. Adjusted geometric means with 95% confidence intervals in ng/L for selected polycyclic aromatic hydrocarbon metabolites by age, gender, race/ethnicity, and smoking status for participants  $\geq 20$  years**

	$\Sigma$ NAP*	$\Sigma$ FLU**	$\Sigma$ PHE***	1-hydroxypyrene****
Males (M)	7,146.2 (6,780.9–7,531.2)	846.9 (810.7–884.7)	364 (342–387.4)	92.3 (88.1–96.7)
Females (F)	8,901 (8,283.6–9,564.5)	855.3 (814.2–898.4)	392.8 (366.5–421)	98.4 (93.4–103.6)
Non-Hispanic white (NHW)	7,920.7 (7,426–8,448.3)	868.7 (830.7–908.5)	391.5 (366.9–417.7)	96.8 (92.2–101.6)
Non-Hispanic black (NHB)	7,506.3 (6,817.6–8,264.5)	825 (769.3–884.8)	338.6 (308.5–371.6)	81.5 (75.6–87.9)
Mexican American (MA)	8,786.4 (7,911.7–9,757.9)	747.8 (705–793.1)	337.8 (307.6–371)	97.7 (91.3–104.6)
Others (OTH)	8,454.9 (7,292.9–9,802)	786.4 (712–868.6)	358.9 (313.3–411.1)	99.2 (89.8–109.5)
Non-smokers (NSM)	5,205.2 (4,890–5,540.7)	574.9 (550.1–600.7)	323.2 (303.1–344.6)	74.2 (70.5–78.2)
Smokers (SM)	17,556.7 (15,919.8–19,361.8)	1,964.9 (1,818.3–2,123.3)	531.5 (484.8–582.7)	160.7 (147.9–174.6)
SHS exposure at home (SHSY)	7,669.8 (6,481.8–9,075.5)	1,036.9 (942–1,141.5)	438.2 (387.9–495)	109.7 (98.1–122.7)
No SHS exposure at home (SHSN)	7,412 (6,975.4–7,875.9)	775.4 (739.9–812.6)	363.8 (340.4–388.7)	89.6 (85.1–94.3)

\*M < F ( $p < 0.01$ ), NHB < MA ( $p = 0.03$ ), NSM < SM ( $p < 0.01$ ).

\*\*NHW > MA ( $p < 0.01$ ), NHW > OTH ( $p = 0.04$ ), NHB > MA ( $p = 0.02$ ), NSM < SM ( $p < 0.01$ ), SHSY > SHSN ( $p < 0.01$ ).

\*\*\*M < F ( $p = 0.04$ ), NHW > NHB ( $p = 0.01$ ), NHW > MA ( $p < 0.01$ ), NSM < SM ( $p < 0.01$ ), SHSY > SHSN ( $p = 0.01$ ).

\*\*\*\*M < F ( $p = 0.049$ ), NHW > NHB ( $p < 0.01$ ), NHB < MA ( $p < 0.01$ ), NHB < OTH ( $p < 0.01$ ), NSM < SM ( $p < 0.01$ ), SHSY > SHSN ( $p < 0.01$ ).

Source: Data from National Health and Nutrition Examination Survey 2003–2008.

Table S2), 2-hydroxynaphthalene (4,184.2 vs. 3,329.5 ng/L,  $p < 0.01$ , Table S2), and 1-hydroxyphenanthrene (161.7 vs. 144 ng/L,  $p < 0.01$ , Table S2) only.

MA had the highest AGM for  $\Sigma$ NAP and 1-hydroxypyrene. However, there were substantial differences in how the metabolites of naphthalene were distributed across three major racial/ethnic groups. While, as compared to other racial/ethnic groups, MA had the lowest concentrations of 1-hydroxynaphthalene, they had the highest concentrations of 2-hydroxynaphthalene. NHB had statistically significantly lower AGM than MA for  $\Sigma$ NAP (7,506.3 vs. 8,786.4 mg/L,  $p = 0.03$ , Table 4). However, while MA had statistically significantly higher AGM than NHW for 2-hydroxynaphthalene (4,713.1 vs. 3,562.6 ng/L,  $p = 0.01$ , Table S2), they had statistically lower AGM than NHW for 1-hydroxynaphthalene (2,457.8 vs. 3,165.2 ng/L,  $p = 0.01$ , Table S2). MA had the lowest AGM for  $\Sigma$ FLU as well as the three metabolites of fluorene (Table S2). NHW had the highest AGM for  $\Sigma$ FLU (Table 4) as well as for 2- and 9-hydroxyfluorene (Table S2). Both NHW (868.7 ng/mL,  $p < 0.01$ ) and NHB (825 ng/L,  $p = 0.02$ ) had statistically significantly higher AGM for  $\Sigma$ FLU than MA (747.8 ng/L, Table 4). Similarly, for 2-, 3-, and 9-hydroxyfluorene, both NHW and NHB had statistically significantly higher AGM than MA (Table S2,  $p < 0.01$ ).

MA had the lowest and NHW the highest AGM for  $\Sigma$ PHE. NHW (391.5 ng/L, Table 4) had statistically significantly higher AGM than both NHB (338.6 ng/L,  $p = 0.01$ ) and MA (337.8 ng/L,  $p < 0.01$ ) for  $\Sigma$ PHE. NHW also had the statistically significantly higher AGM than NHB for 1-, 2-, and 4-hydroxyphenanthrene ( $p < 0.01$ , Table S2). NHW also had the statistically significantly higher AGM than MA for 1-, 2-, and 3-hydroxyphenanthrene ( $p < 0.01$ , Table S2). NHB had statistically significantly lower AGM for 1-hydroxyphenanthrene than MA (120.6 vs. 136.5 ng/L,  $p = 0.02$ , Table S2) and statistically significantly higher AGM than MA for 3-hydroxyphenanthrene (107.8 vs. 88.9 ng/L,  $p < 0.01$ , Table S2). Both NHW (96.8 ng/L,  $p < 0.01$ ) and MA (97.7 ng/L,  $p < 0.01$ ) had statistically significantly higher AGM than NHB (81.5 ng/L, Table 4) for 1-hydroxypyrene.

Smokers had statistically significantly higher AGM for  $\Sigma$ NAP,  $\Sigma$ FLU,  $\Sigma$ PHE as well as 1-hydroxypyrene than non-smokers ( $p < 0.01$ , Table 4). In fact, for both  $\Sigma$ NAP and  $\Sigma$ FLU, AGMs for smokers were more than three times of what they were for non-smokers (17,556.7 vs. 5,205.2 ng/L for  $\Sigma$ NAP, and 1,964.9 vs. 574.9 ng/L for  $\Sigma$ PHE). Non-smokers had statistically significantly lower AGMs for every metabolite of naphthalene, fluorene, and phenanthrene than smokers ( $p < 0.01$ , Table S2). It should be noted that for 3-hydroxyfluorene, AGM for smokers was more than six times of what it was for non-smokers (450.9 vs. 73.1 ng/L, Table S2).

Exposure to SHS at home was associated with higher concentrations of  $\Sigma$ NAP,  $\Sigma$ FLU,  $\Sigma$ PHE as well as 1-hydroxypyrene as compared to no exposure to SHS at home. However, statistical significance (Table 4,  $p \leq 0.01$ ) was reached for  $\Sigma$ FLU,  $\Sigma$ PHE, and 1-hydroxypyrene only. For almost every metabolite of naphthalene, fluorene, and phenanthrene, exposure to SHS at home was associated with statistically significantly higher AGM concentrations than when there was no exposure to SHS at home ( $p < 0.01$ , Table S2). The only exception was 2-hydroxynaphthalene for which statistical significance was not reached.

When interaction between smoking status and HmE was considered for  $\Sigma$ NAP, while non-smokers did have statistically significantly lower AGM than smokers (4,531.2 vs. 26,788.5 ng/L with SHS exposure at home, 5,382.2 vs. 15,856 ng/L with no SHS exposure at home) irrespective of SHS exposure at home, it was only for smokers that SHS exposure at home was associated with higher AGM than when there was no SHS exposure at home (26,788.5 vs. 15,856 ng/L, Figure 2, Panel A,  $p < 0.01$ ). However, for  $\Sigma$ FLU (Figure 2, Panel B), SHS exposure at home was associated with higher AGM than when there was no SHS exposure at home for both smokers (2,952.8 vs. 1,779.6 ng/L) and non-smokers (675.5 vs. 552.7 ng/L). There were no statistically significant gender differences in AGM for  $\Sigma$ FLU for non-smokers (593.2 for males vs. 557.9 ng/L for females, Figure 3, Panel A) but for smokers, males had statistically significantly lower AGM than females (1,791.7 vs. 2,145.8 ng/L,  $p < 0.01$ , Figure 3, Panel A). The racial/ethnic differences in the concentrations of 1-hydroxypyrene were limited to males only (96.9 ng/L for NHW, 65.4 ng/L for NHB, 92.5 ng/L for MA, and 93.4 ng/L for OTH,

Figure 2. Adjusted geometric means with 95% confidence intervals in ng/L by smoking status and second-hand smoke (SHS) exposure at home (NSM\_SHSY = nonsmokers with SHS exposure at home, NSM\_SHSN = non-smokers with no SHS exposure at home, SM\_SHSY = smokers with SHS exposure at home, SM\_SHSN = smokers with no SHS exposure at home those aged  $\geq 20$  years) (A)  $\Sigma$ NAP, and (B)  $\Sigma$ FLU.

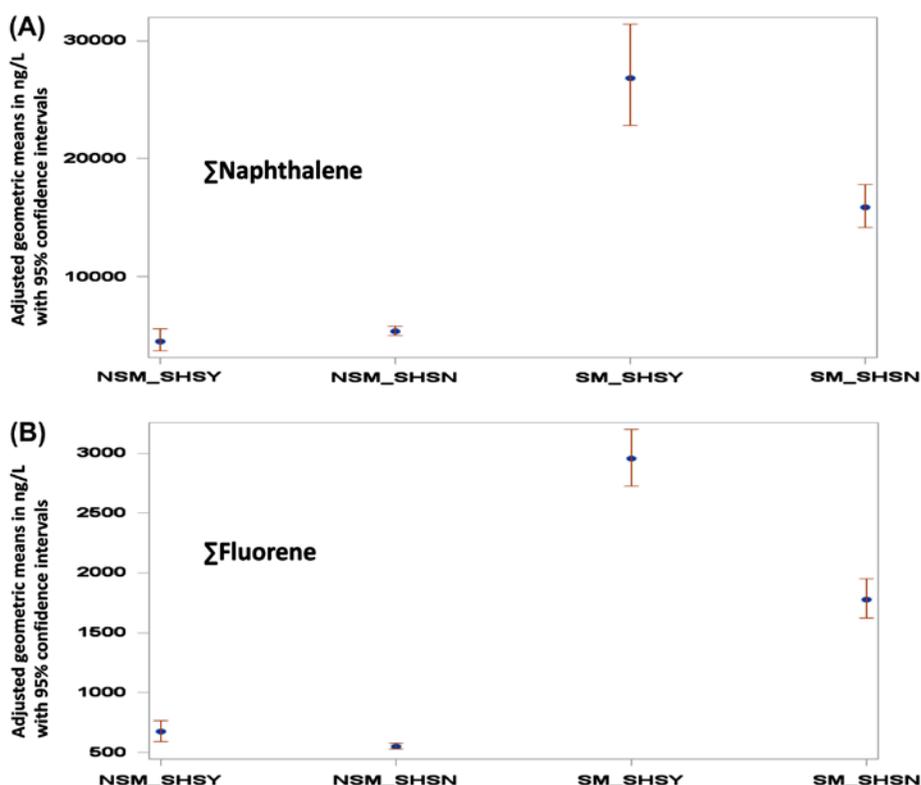


Figure 3. Adjusted geometric means with 95% confidence intervals in ng/L for  $\geq 20$  years old by (A) gender and smoking status for  $\Sigma$ FLU (M\_NSM = male nonsmokers, M\_SM = male smokers, F\_NSM = female nonsmokers, F\_SM = female smokers) (B) by gender and race/ethnicity for 1-hydroxypyrene (M\_NHW = non-Hispanic white males, M\_NHB = non-Hispanic black males, M\_MA = Mexican American males, M\_OTH = other males, F\_NHW = non-Hispanic white females, F\_NHB = non-Hispanic black females, F\_MA = Mexican American females, F\_OTH = other females).

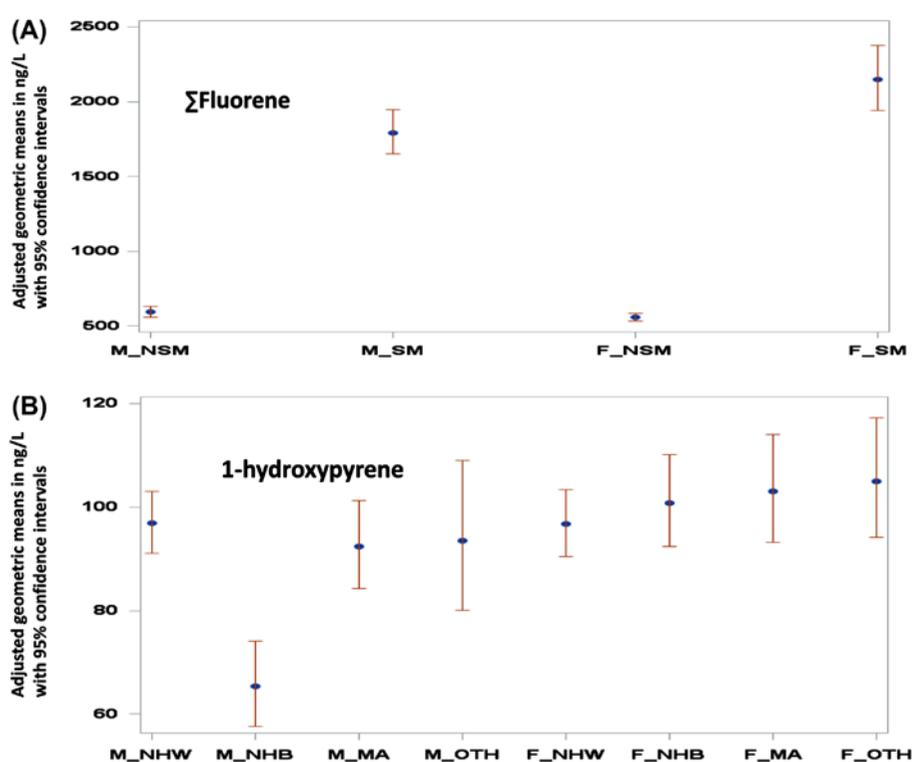


Figure 4. Adjusted geometric means with 95% confidence intervals in ng/L those aged  $\geq 20$  years by smoking status and second-hand smoke (SHS) exposure at home for 1-hydroxypyrene (NSM\_SHSY = non-smokers with SHS exposure at home, NSM\_SHSN = nonsmokers with no SHS exposure at home, SM\_SHSY = smokers with SHS exposure at home, SM\_SHSN = smokers with no SHS exposure at home).

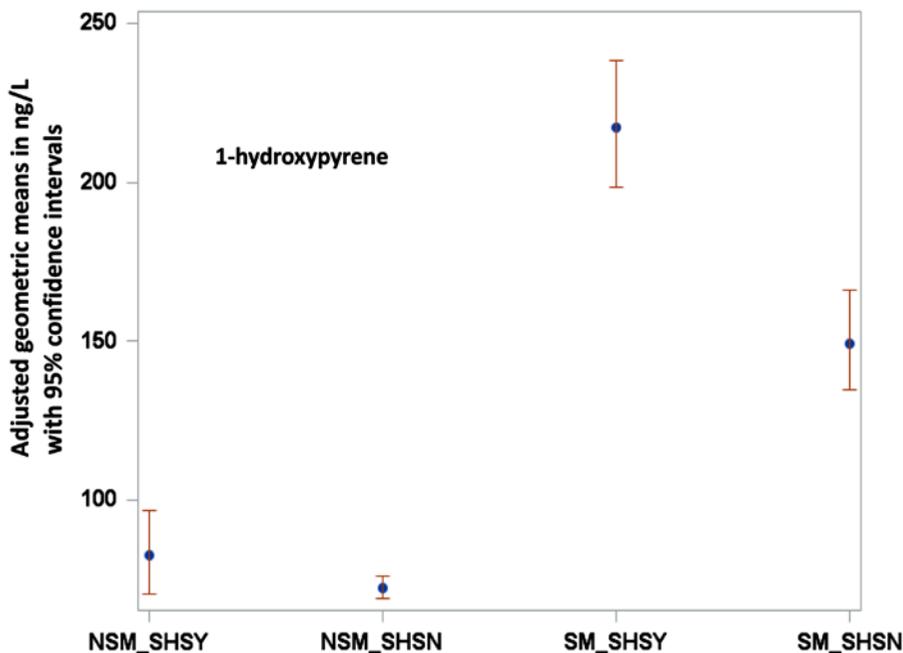


Figure 3, Panel B) and gender differences were limited to NHB only (65.4 ng/L for males vs. 100.8 ng/L for females, Figure 3, Panel B). In addition, while non-smokers had lower AGM for 1-hydroxypyrene than smokers irrespective of SHS exposure at home (82.5 vs. 217.3 ng/L with SHS exposure at home, 72.3 vs. 149.3 ng/L with no SHS exposure at home, Figure 4), it was for only smokers that SHS exposure at home was associated with statistically significantly higher AGM than when there was no exposure to SHS at home (217.3 vs. 149.3 ng/L, Figure 4).

#### 4. Discussion

Ding, Trommel, Yan, Ashley, and Watson (2005) presented data on 14 PAHs present in the mainstream smoke of various US cigarette brands. Naphthalene was found to be the most abundant PAH irrespective of cigarette brand followed by fluorene and phenanthrene. As provided in Supporting Information to Ding et al. (2005), the mean concentrations of naphthalene, fluorene, phenanthrene, and pyrene in full flavor Marlboro were found to be 386 ng/cigarette, 182 ng/cigarette, 145 ng/cigarette, and 59.1 ng/cigarette respectively. AGMs for these four PAHs observed in this study followed the same pattern not only for smokers but for non-smokers also (Table 4). The same was true for those who were and were not exposed to SHS at home (Table 4). St.Helen et al. (2012) found hydroxyfluorene metabolites to be most discriminative of smokers from non-smokers followed by metabolite of 2-naphthalene and 1-pyrene. Similar results were observed in this study also. The ratios of AGMs for smokers divided by non-smokers were found to be 1.45, 3.42, 1.64, and 2.17 for naphthalene, fluorene, phenanthrene, and 1-pyrene metabolites respectively. The ratios of these AGMs for 1-naphthalene and 2-naphthalene metabolites were 4.2 and 3.43 respectively.

In this study, for both aggregated (Table 4) as well as individual metabolite levels (Table S2) for naphthalene, fluorene, phenanthrene, and pyrene, smokers were found to have statistically significantly higher concentrations than non-smokers ( $p < 0.01$ ) indicating the excess risk of exposure to PAHs carried by smokers. Suwan-ampai et al. (2009) also found active smokers (defined as those who self-reported themselves to be current smokers or had serum cotinine levels  $\geq 10$  ng/mL) to have statistically significantly higher concentrations of all PAH metabolites than those who were not exposed to tobacco smoke (defined as those who did not self-report themselves to be current smokers, were not living with a smoker, and had serum cotinine levels below LOD).

Also, for both aggregated (Table 4) as well as individual metabolite levels (Table S2) for fluorene, phenanthrene, and pyrene, SHS exposure at home was associated with statistically significantly higher concentrations than when there was no exposure to SHS at home. The same was true for 2-hydroxynaphthalene. Thus, even the exposure to SHS at home is associated with excess risk of exposure to PAHs. Suwan-ampai et al. (2009) also found involuntary smokers (defined as those who did not self-report themselves to be current smokers but were living with a smoker in the home and had detectable but < 10 ng/mL level of serum cotinine) to have statistically significantly higher levels of 1-hydroxypyrene, 1-, 2-, and 3-hydroxyphenanthrene, and 2- and 3-hydroxyfluorene than those who were not exposed to tobacco smoke. I, in addition to evaluating the effect of SHS exposure at home, also evaluated the impact of the extent of SHS exposure at home. The variable that indicated the extent of SHS exposure at home was the number of cigarette smokers inside home every day. Suwan-ampai et al. (2009) did not consider the role of the extent of SHS exposure at home in their study. In addition, instead of generating adjusted geometric means as I generated, they generated ratios of geometric means which may not have a practical value if someone is looking for the numeric concentrations of PAH exposure. I considered the interactions between various categorical variables in models in our analyses, they did not. I evaluated the co-exposure of SHS at both home and work in our analyses for adults, they did not.

As would be expected, the concentrations of  $\Sigma$ NAP increased with increase in the number of hours per day tobacco smoke was inhaled at work ( $p = 0.005$ , Table 3) but contrary to what should be expected, those who were exposed to SHS at work were found to have lower concentrations of  $\Sigma$ NAP than those who were not exposed to SHS at work ( $p = 0.012$ , Table 3). The reason for this is unknown but there may be one possibility. All those who were not at a job and/or were not working were coded as being not exposed to SHS at work while, in fact, they may have been exposed. This may have resulted in a false statistically significant finding. Thus, caution should be exercised in interpreting this finding. It should be noted only when 1-hydroxynaphthalene was statistically significantly ( $p = 0.002$ , Table S1) negatively associated with exposure to SHS at work.

When the primary source of exposure to PAHs is through inhalation, the concentrations of exposure can be enhanced with increase in physical activity which may be associated with increased inhalation rate and of course, exposure levels do depend upon the concentration of these chemicals in the air. While, exposure to other PAHs can occur through multiple sources, exposure to naphthalene occurs mainly through inhalation (Li, Sandau, et al., 2008). When route of exposure is through diet, enhanced physical activity may lead to relatively higher energy intake which depending upon the concentrations of PAHs in the diet may lead to higher levels of exposure and which may affect the rate of metabolism.

Out of 10 PAH metabolites for which data were available for this study, concentrations of 6 metabolites increased statistically significantly over the study period of 2003–2008 (Tables 3 and S1). The increasing trends seen in this study should be of concern because of established mutagenicity and carcinogenicity (Castano-Vinyals et al., 2008; Perera, Tang, Whyatt, Lederman, & Jedrychowski, 2005; Petruzzelli et al., 1998) associated with exposure to certain PAHs. Since the source of exposure to PAHs was not available, it is not possible to speculate the reasons for these increases. However, diet is one source of exposure that could be a possible reason. Unfortunately, in spite of the availability of data on diet from 24-h recall questionnaires, it was not possible to assess this as a source of exposure to PAHs because of the unavailability of complete data on the consumption of the smoked and broiled food.

Suwan-ampai et al. (2009) used data from NHANES for the period 1999–2002 and reported females to have higher model adjusted geometric means than males for 1-hydroxyphenanthrene and 2-hydroxynaphthalene. The geometric means for other metabolites analyzed by them were similar for males and females. Some of the results reported by Suwan-ampai et al. (2009) could not be confirmed by me because of the differences in design between this study and study by Suwan-ampai et al. (2009). Suwan-ampai et al. (2009) used participants aged 6 years and older in their study. This study used participants aged 20 years and older. There may be several reasons why for this study,

females were found to have statistically significantly higher concentrations of 1- and 2-hydroxynaphthalene, 1-hydroxyphenanthrene, 1-hydroxypyrene, and total naphthalene, fluorene, and phenanthrene metabolites. First, though unlikely, females may be consuming diets cooked at high temperatures more often and/or in larger quantities than males. Adjustments for diets that expose consumers to relatively higher levels of exposure to PAHs could not be made for reasons previously mentioned. Secondly, which seems to be a more likely explanation, there may be differences in how certain PAHs are metabolized by males and females. It appears that females metabolize certain PAHs like naphthalenes more slowly than males or in other words, females have longer half-life for these PAHs than males. Gender differences in PAH-induced CYP1A1 expression have been reported by Mollerup, Ryberg, Hewer, Phillips, and Haughen (1999) among other authors.

It should be noted that UGMs for males were higher for  $\Sigma$ FLU and 1-hydroxypyrene (Table 2) than females but when adjustments were made for differential contributions of race/ethnicity, poverty income ratio and other factors, these differences disappeared for  $\Sigma$ FLU and the differences were reversed for 1-hydroxypyrene (Table 4).

Some of the racial/ethnic differences and associations with socioeconomic status noted by Suwani-ampai et al. (2009) also could not be confirmed in this study. In this study, family poverty income ratio (PIR) as a continuous variable was used as an indicator of socioeconomic status. Low income was associated with statistically significantly higher concentrations for  $\Sigma$ FLU and 1-hydroxypyrene (Table 3) as well as 3-hydroxyphenanthrene (Table S1). Suwani-ampai et al. (2009) used both respondents' education as well as PIR as indicators of socioeconomic status but instead of using PIR as a continuous variable, they used it as a categorical variable. Suwani-ampai et al. (2009) found low PIR as compared with medium and/or high PIR to be associated with higher concentrations of 1-hydroxypyrene, 1-, 2-, and 3-hydroxyphenanthrene, 1- and 2-hydroxynaphthalene, 2- and 3-hydroxyfluorene. Once again, the differences in results in this and their study may be due to differences in study design. They found NHW to have higher concentrations of all PAH metabolites except 2-hydroxynaphthalene than MA with or without statistical significance having been reached. Also, they found NHW to have higher concentrations of all PAH metabolites except 2-hydroxynaphthalene and 9-hydroxyfluorene than NHB with or without statistical significance having been reached. Similar results were observed in this study also except for distribution of the concentrations of 1-hydroxynaphthalene and 2-hydroxynaphthalene (Table S2). While MA had the lowest concentrations of 1-hydroxynaphthalene at 2,457.8 ng/L and NHW had the highest concentrations at 3,165.2 ng/L, for 2-hydroxynaphthalene, MA had the highest concentrations at 4,713.1 ng/L, and NHW had the lowest concentrations at 3,562.6 ng/L. Racial/ethnic differences in concentrations for various PAHs seen in this study are also probably due to differences in how various PAHs are metabolized by different race/ethnicities. More work will be needed to explain why MA had the highest concentrations of 2-hydroxynaphthalene and lowest concentrations of 1-hydroxynaphthalene or, in other words, metabolize them differently. However, racial differences in CYP1A1 gene inducibility have been reported by Cosma et al. (1993).

This study furthers the work of Suwan-ampai et al. (2009) in several ways. First, it provides updates on the concentrations of PAH metabolites for the recent years 2003–2008 as compared to the data for 1999–2002 provided by Suwan-ampai et al. (2009). From the public health perspective, it is important to know how the risk of exposure to environmental contaminants as determined by observed concentrations in either urine or serum varies over time. Even though Suwan-ampai (2009) used data from two different cycles of NHANES, namely, 1999–2000 and 2001–2002, they did not study if the concentrations of PAH metabolites varied from 1999–2000 to 2001–2002. I used model-based methodology to estimate how concentrations of each of the 10 metabolites (Tables S1 and 3) as well as total naphthalene, fluorene, and phenanthrene metabolites (Table 3) varied over time from 2003–2004 to 2007–2008. In addition, I also computed unadjusted concentrations of three aggregated metabolites for each of the three NHANES cycles (Table 2), namely, 2003–2004, 2005–2006, and 2007–2008. Second, while Suwan-ampai (2009) did use data on exposure to SHS at home to define involuntary smokers, they did not evaluate if the extent of exposure to SHS at home may also affect PAH concentrations. I used model-based approach to determine if the extent of exposure to SHS at

home defined by the number of smokers smoking inside home may affect PAH concentrations (Table S1 and 3). The results indicate that both the exposure as well the extent of exposure to SHS at home affect PAH concentrations. Third, while Suwan-ampai (2009) did not determine if the SHS exposure at work may affect PAH concentrations, I used model-based approach to determine if the exposure as well the extent of exposure to SHS at work may affect PAH concentrations (Tables S1 and 3).

In summary, (i) statistically significantly higher concentrations of selected metabolites of naphthalene, fluorene, phenanthrene, and pyrene are associated with exposure to SHS at home, (ii) smoking was associated with statistically significantly higher concentrations of every metabolite of naphthalene, fluorene, phenanthrene, and pyrene analyzed in this study, (iii) there was a statistically significant increase in the concentrations of total naphthalene and fluorene, 2-hydroxynaphthalene, 2- and 9-hydroxyfluorene, 2- and 4-hydroxyphenanthrene, and 1-hydroxypyrene during the study period of 2003–2008, and (iv) poverty income ratio was negatively associated with the concentrations of 2-, 3-, and 9-hydroxyfluorene, 3-hydroxyphenanthrene, and 1-hydroxypyrene.

The limitations of this study should be carefully considered. Consumption of food cooked at high temperatures like broiled and smoked food has been found to be associated with elevated concentrations of PAHs. An attempt was made to include the consumption of such food as an independent variable in this study but since it was not fully possible to identify all those who consumed smoked or broiled food, a few data points where it was possible to identify smoked or broiled food were deleted from the analysis. To what degree, the results of this study could have been affected because of this limitation is unknown. Neither the magnitude nor the source of exposure to PAHs was available. The timing of the exposure was also not available for either SHS or PAHs. Data on exposure to PAHs from traffic related sources were also not available and as such, exposure to PAHs from traffic sources could not be included as one of the independent variables in the regression models. A relatively long-term follow-up study (or may be a short term well designed study) that can keep track of the ongoing PAH and SHS exposure and observed urinary PAH metabolite concentrations may better be able to evaluate association between PAH exposure and SHS exposure.

#### List of abbreviations

AGM	Adjusted geometric mean
BMI	Body mass index
CPD_Home	Number of cigarettes smoked inside home every day
FLU	Fluorene
HmE	Exposure to second-hand smoke inside home
Hours_Smk	Number of hours per day tobacco smoke inhaled per day at work from other people's cigarettes, cigars, or pipes
LOD	Limit of detection
MA	Mexican American
NAP	Naphthalene
NHANES	National Health and Nutrition Examination Survey
NHB	Non-Hispanic black
NHW	Non-Hispanic white
OTH	Other unclassified race/ethnicity
PAH	Polycyclic aromatic hydrocarbons
PHE	Phenanthrene
PIR	Poverty income ratio
PYE	Pyrene
SHS	Second-hand smoke
UGM	Unadjusted geometric mean
USDA	United States Department of Agriculture
WKE	Exposure to second hand smoke at work

### Supplementary material

Supplementary material for this article can be accessed here <http://dx.doi.org/10.1080/23311843.2015.1031508>.

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