Estimating Exposure to Polycyclic Aromatic Hydrocarbons: A Comparison of Survey, Biological Monitoring, and Geographic Information System–Based Methods

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Abstract

Our objective was to compare polycyclic aromatic hydrocarbon (PAH) exposure estimates based on survey, biological monitoring, and geographic information system (GIS) methods. The 304 participants in this study supplied a urine sample and completed questionnaires about exposure to potential PAH sources. We assayed urine samples for 1-hydroxypyrene-*O*-glucuronide (1-OHPG), the major metabolite of pyrene, a common PAH. We used a GIS to estimate traffic exhaust exposure using vehicle count data at the residence and workplace. The five subjects who reported smoking during the 48-hour period had median 1-OHPG concentrations 10-fold that of nonsmokers (1.6 versus 0.16 pmol/mL; $P = 0.01$). Among nonsmokers, those who reported eating grilled, roasted, or broiled meat had significantly higher 1-OHPG concentrations than those who did not reported eating meat prepared by these methods (0.25 versus 0.06 pmol/mL; $P = 0.02$). Nonsmokers who reported traveling on roads for ≥3 hours during the 48-hour period also had significantly higher 1-OHPG levels than those who traveled <3 hours (0.23 versus 0.11 pmol/mL; $P = 0.03$). 1-OHPG levels were also correlated with hours of secondhand smoke exposure among nonsmokers ($P = 0.04$). In this study, 1-OHPG urine concentrations were not associated with self-reported exposures to cooking smoke, wood burning, or traffic levels near the home or to traffic density or urban/rural status determined using a GIS. Self-reported indicators of residential proximity to high traffic volume were, however, associated with GIS traffic density measures. (Cancer Epidemiol Biomarkers Prev 2006;15(7):1376–81)

Introduction

There has been growing interest in evaluating exposure to vehicle exhaust for epidemiologic studies of lung cancer (1, 2), childhood cancer (3-6), breast cancer (7), asthma (8-10), respiratory symptoms (11), all cause mortality (12), and birth outcomes (13-16). These studies often rely on exposure estimates derived from surveys or a geographic information system (GIS). It is not clear, however, how well these measures capture personal exposures to vehicle exhaust or how they relate to each other. Vehicle exhaust is a major contributor to the concentration of polycyclic aromatic hydrocarbons (PAH) in urban areas and near major roads (17, 18). Several PAHs are classified as probable human carcinogens based on evidence from occupational and laboratory animal studies (19, 20), making these compounds of primary interest for epidemiologic studies of cancer and exposure to vehicle exhaust. PAHs are formed during incomplete combustion of organic compounds; therefore, exposure occurs not only from vehicle exhaust but also from a variety of sources, including diet and tobacco smoke (21).

Personal air monitoring of women in New York City found that pyrene had the highest mean concentration among the PAHs measured (22). Personal air monitoring for PAHs is difficult and expensive and does not capture exposure from dietary sources, making development of a good biological exposure marker highly desirable. 1-Hydroxypyrene-*O*-glucuronide (1-OHPG) is the major urinary metabolite of pyrene, a common PAH (23). Elevated 1-OHPG levels have been measured in steel plant and waste incineration workers exposed to PAHs (24, 25). Because 1-OHPG is more sensitive than other exposure biomarkers, it has been proposed as a biomarker of exposure to environmental PAHs in air (26).

In this cross-sectional study, we determined 1-OHPG concentrations for 24-hour urine samples from a subset of women enrolled in the California Teachers Study cohort. The main objective of our study was to determine whether 1-OHPG urine concentrations were related to self-reported sources of PAH exposure or GIS-based estimates of traffic density. If so, this marker could be used to estimate PAH exposures in larger epidemiologic studies.

Materials and Methods

Study Population. Our study population consisted of 304 women participating in a measurement substudy of the California Teachers Study cohort. The California Teachers Study cohort includes 133,479 women who were active or retired public school teachers or administrators enrolled in the State Teachers Retirement System in 1995 (27).

The measurement substudy, conducted in 2000, included a random sample of 528 California Teachers Study participants who were ≤85 years old when the cohort was originally established (i.e., at baseline) and who resided in the substudy area (i.e., western Alameda, Santa Clara, San Mateo, Santa Cruz, Monterey, or northern San Benito counties in California). Forty-four (8%) of these women were not contacted because they had died, moved out of the substudy area, or could not be located. Of the 484 women invited to participate, 328 (68%) agreed, 138 refused, and 18 were not interviewed for other reasons. Most of the 18 not interviewed were too ill ($n = 9$). Reasons for not interviewing the remaining 9 were varied and included passive refusals (i.e., showed initial interest but failed

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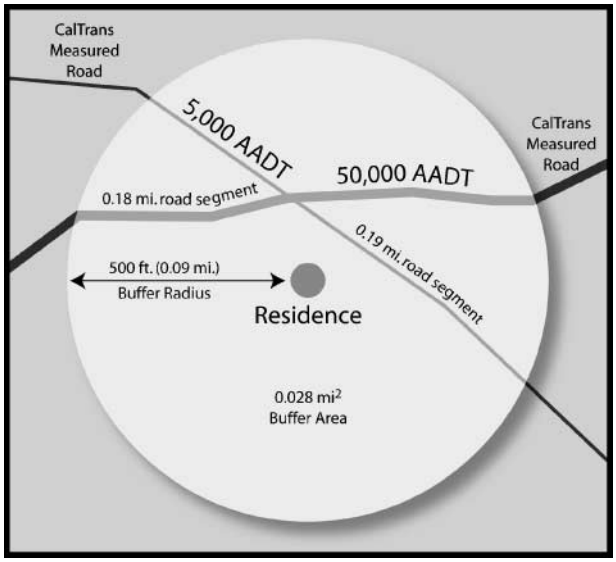

Buffer area = 0.028 square miles

Traffic Density: $TD = \frac{\sum (AADT \times L)}{A_B}$

Where: TD = traffic density (vehicles × miles/day/miles^2)
AADT = annual average daily traffic (vehicles/day)
L = length of road segment (miles)
A_B = buffered area (miles^2)

Example: $TD = \frac{(5{,}000 \times 0.19\,\text{mi}) + (50{,}000 \times 0.18\,\text{mi})}{0.028\,\text{mi}^2} = 355{,}350\ \text{VMT/day/mi}^2$

Figure 1. Method used to estimate traffic density (VMT/d/mi^2) near a residence.

to follow-through with the interview), personal/family issues that interfered with completing the interview, and scheduling problems that precluded interviews from taking place before the end of the study period. Of the 328 participants, 304 (93%) provided a 24-hour urine specimen. Our analysis is based on these 304 women, 124 of whom were urban and 180 of whom were rural residents. The use of human subjects in this study was reviewed by the California Health and Human Services Agency, Committee for the Protection of Human Subjects and found to be in compliance with their ethical standards as well as with the U.S. Code of Federal Regulations, Title 45, Part 46 on the Protection of Human Subjects.

Survey Data. We conducted in-person interviews, covering a variety of topics, including general exposure information and information on selected exposures within the 24 hours preceding urine collection. A self-administered questionnaire collected exposure information during the 24-hour urine collection period. The information we obtained from participants included cigarette smoking; exposure to secondhand tobacco smoke; time spent traveling on roads; traffic levels on their street of residence; presence of a highway within four blocks of their home; use of wood-burning stoves; use of cooking methods that produce smoke, including grilling, frying, or sautéing; and consumption of meat prepared by methods that generate the highest PAH levels (grilling, broiling, or roasting). The in-person interviews covering the 24 hours preceding urine collection used a more detailed questionnaire with meat photographs that was developed to assess exposure to PAHs based on the degree of doneness (28). Subjects also provided their residential address and workplace address if they were currently employed outside their homes.

Biological Monitoring Data. We gave each participant a collection kit and instructed her to collect all urine produced in the 24-hour period starting immediately following the in-person interview. We asked them to use cold packs, placed around the outside of the jug, to keep the sample chilled and, if willing, to refrigerate the samples during the 24-hour collection period. At the end of the 24 hours, interviewers collected each sample and stored it in a −20°C freezer until it was transported to our main laboratory (within 2 weeks) where it was thawed, aliquoted, and frozen at −70°C.

We assayed the urine samples for 1-OHPG, a major urinary metabolite of pyrene. 1-OHPG concentrations were quantified using immunoaffinity chromatography specific for PAH metabolites followed by synchronous fluorescence spectroscopy (23). 1-OHPG levels were determined in pmol/mL. The reporting limit for 1-OHPG was 0.05 pmol/mL urine. We did not adjust for creatinine concentration because the samples were 24-hour voids, making normalization for hydration unnecessary when examining chemical exposures (29, 30). The concentration of 1-OHPG in urine is an indicator of recent exposure to PAHs through all routes. The half-life for urinary excretion of 1-OHPG ranges from 6 to 35 hours and peak urine concentration occurs a few hours following exposure (31).

GIS Data and Analysis. We used a GIS to address-geocode residences and workplace information to spatially referenced road networks (32, 33). The geocoded residential locations were also assigned urban or rural classifications based on U.S. Census data (34). To estimate traffic density around each location, we used traffic-count data for 2000, available from the Highway Performance and Monitoring System of the U.S. Department of Transportation (35). These data provide the annual average daily traffic (AADT) for all highways and most major roads in the state. The AADT represents the average number of vehicles per day traveling in both directions on a road segment. The road segments are spatially referenced in a GIS database. AADT is based on rotating traffic counts conducted every 3 years; during noncount years, the AADT is estimated using traffic trends for that location. The AADT is not typically measured on smaller, residential roads with low traffic volumes; therefore, values for these roads are not included in the database. For our analyses, we set the traffic volumes assigned for these small roads to zero.

For each residence and workplace, we calculated the vehicle miles of travel (VMT) within a 500-foot radius of the residence, for each attributed road segment, by multiplying the AADT by the road segment length within the buffer (Fig. 1). We used a 500-foot radius because this approximates the distance from a road at which pollutant concentrations are the highest based on available monitoring data (36-38). We calculated traffic density by summing the VMT for all road segments and dividing by the buffered area (0.028 mi^2). The resulting units for traffic density are VMT/d/mi^2.

$$TD = \frac{\Sigma(AADT \times L)}{A_B}$$

where TD is traffic density (vehicles × miles/d/mi^2), AADT is annual average daily traffic (vehicles/d), L is length of road segment (miles), and A_B is buffered area (mi^2).

Statistical Analysis. We used nonparametric tests for statistical comparisons because the distributions of both traffic density and 1-OHPG were highly skewed. All analyses were done using SAS version 8 (SAS Institute, Inc., Cary, NC). We evaluated the relationship of survey and GIS-based exposure measures to 1-OHPG levels using both continuous and categorical variables. For continuous variables, we used the Spearman rank correlation coefficient (PROC CORR SPEARMAN); for categorical variables, we used the Kruskal-Wallis

one-way ANOVA by ranks (PROC NPAR1WAY). We also used the Kruskal-Wallis one-way ANOVA by ranks to compare the distribution of GIS-based traffic measures with self-reported traffic levels.

We combined information from the personal interview and self-administered questionnaire to estimate potential PAH exposure during a 48-hour period, including the day before and day of urine collection. We also evaluated data from the self-administered questionnaire covering only the 24-hour urine collection period.

Results

The age of study participants ranged from 27 to 80 years (mean, 51 years). Two hundred fifty-nine (85%) of the 304 participants were non-Hispanic white, 3 (1%) were black, 18 (6%) were Hispanic, 13 (4%) were Asian, and 11 (4%) were classified as other/unknown race/ethnicity. The distributions of these characteristics among the 156 women who were eligible for the study but did not participate were not different. The participation rate was higher among rural residents (75%) than among urban residents (57%).

Table 1 shows the distributions of 1-OHPG levels in urine and residential and workplace traffic density exposures. Forty-five percent of the subjects had 1-OHPG levels below the reporting limit of 0.05 pmol/mL urine. The mean ± SD concentration was 0.58 ± 1.2 pmol/mL urine. The 75th percentile and maximum values of 1-OHPG were 0.51 and 8.9 pmol/mL, respectively. Thirty-four percent of the participants' residences and 29% of their workplaces did not have a road with measured traffic within 500 feet and were therefore assigned traffic density values of zero. The traffic density distributions appeared log normal with a wide range of values. The median traffic density was 10,986 VMT/d/mi^2 for residences and 38,717 VMT/d/mi^2 for workplaces. The maximum traffic density was 687,250 VMT/d/mi^2 for residences and 1,499,208 VMT/d/mi^2 for workplaces.

Table 2 provides the results from categorical comparisons of the survey-based exposure data with 1-OHPG levels. Subjects who reported smoking in the last 48 hours had median 1-OHPG concentrations (1.61 pmol/mL) that were 10 times higher than those of nonsmokers (0.16 pmol/mL) and the difference was statistically significant ($P = 0.01$). Because there were only five subjects who reported smoking, further comparisons between PAH exposures and 1-OHPG values were limited to nonsmokers ($n = 299$). Nonsmokers who ate grilled, broiled, or roasted meat during the 48-hour period had levels of 1-OHPG significantly higher ($P = 0.02$), with a median concentration (0.25 pmol/mL) more than four times higher, than those who did not eat meat prepared by these methods (0.06 pmol/mL). Subjects that traveled on roads by car, bus, or bike or on foot for ≥3 hours during the 48-hour period also had 1-OHPG concentrations (0.23 pmol/mL) significantly higher ($P = 0.03$) than those who traveled on roads for <3 hours (0.11 pmol/mL). Median 1-OHPG values did not differ by exposure to secondhand tobacco smoke, cooking by sautéing, frying, or grilling, or living on a street with heavy or moderate traffic or within four blocks of a highway. The results were similar using only exposure information from the 24 hours coinciding with urine collection (data not shown).

Table 1. Distributions of 1-OHPG levels (pmol/mL) in urine and traffic density (VMT/d/mi^2) within 500 feet of location

Value	1-OHPG (pmol/mL)	Residential traffic (VMT/d/mi^2)	Workplace traffic (VMT/d/mi^2)
n	304	304	221
Mean (SD)	0.58 (1.2)	43,675 (87,877)	87,948 (178,705)
Minimum	≤0.05*	0	0
% Minimum	45	34	29
25th Percentile	0.05	0	0
50th Percentile	0.18	10,986	38,717
75th Percentile	0.51	54,577	108,368
90th Percentile	1.4	112,057	187,549
Maximum	8.9	687,250	1,499,208

*The reporting limit for 1-OHPG was 0.05 pmol/mL.

Table 2. Categorical comparison of survey-based exposure estimates and biological monitoring results using Kruskal-Wallis one-way ANOVA by ranks

Exposure category*	Subjects (*n*)	Median 1-OHPG (pmol/mL)	*P*
Smoked	5	1.61	0.01
Did not smoke	299	0.16	
Among nonsmokers only ($n = 299$)			
Exposed to secondhand smoke	24	0.25	0.20
Not exposed to secondhand smoke	275	0.16	
Ate grilled meat	134	0.25	0.02
Did not eat grilled meat	165	0.06	
Cooked by grilling, etc.	129	0.21	0.67
Did not cook by grilling, etc.	170	0.13	
Wood burning in home	23	0.25	0.89
No wood burning	276	0.16	
Traveled on roads ≥3 hours	88	0.23	0.03
Traveled on roads <3 hours	211	0.11	
Heavy/moderate traffic on street	54	0.25	0.18
Light traffic on street	244	0.12	
Lived within four blocks of highway	97	0.10	0.33
Did not live within four blocks of highway	202	0.19	

*For the 24 hours preceding and 24 hours coinciding with urine collection.

Table 3 presents the results from categorical comparisons of GIS-based traffic exposure estimates and 1-OHPG levels. Among nonsmokers, 1-OHPG concentrations did not differ between urban and rural residents. Nor did 1-OHPG concentrations differ between categories of residential and workplace traffic density. Subjects in the highest traffic density exposure category (≥100,000 VMT/d/mi^2) had a median 1-OHPG concentration at the reporting limit (0.05 pmol/mL). The results were similar for residential traffic density when restricted to participants that did not report a work address and were likely to spend the most time at home. We obtained similar results with respect to urban or rural status and workplace traffic density levels when limited to respondents who did not report smoking, eating grilled, broiled, or roasted meat, or traveling ≥3 hours on roads (data not shown).

Table 4 shows the correlations between continuously measured exposure measures and 1-OHPG levels. The number of cigarettes smoked and number of meals eaten with grilled, broiled, or roasted meat during the 48-hour period were both positively correlated ($P < 0.05$) with 1-OHPG levels. Although these correlations were statistically significant, the correlation coefficients were not large ($r = 0.13$ and 0.16, respectively). The number of meals cooked by sautéing, frying, or grilling during the 48-hour period was not related to 1-OHPG levels. When we used continuous, instead of categorical, variables, the results were different for exposure to secondhand smoke and time spent traveling on roads. Hours of exposure to secondhand tobacco smoke was positively correlated with 1-OHPG levels ($P < 0.05$), although the median 1-OHPG levels were not different when compared as a categorical variable (Table 2). The number of hours spent traveling on roads over the last 48 hours was not significantly correlated with 1-OHPG concentrations ($P = 0.10$), but the median levels were different when compared categorically. The traffic densities (VMT/d/mi^2) within 500 feet of the subjects' residences or workplaces were also not correlated with 1-OHPG levels. The relationship between continuous measures of traffic density and 1-OHPG

Table 3. Categorical comparison of GIS-based exposure estimates and biological monitoring results using Kruskal-Wallis one-way ANOVA by ranks

Exposure category	Subjects (*n*)	Median 1-OHPG (pmol/mL)	*P*
Among nonsmokers only (n = 299 for residential and 217 for workplace)			
Urban residence	122	0.16	0.84
Rural residence	177	0.17	
Residential traffic density* ≥100,000	36	0.05	0.12
Residential traffic density* 1-99,999	160	0.21	
Residential traffic density† 0	103	0.11	
Workplace traffic density* ≥100,000	56	0.05	0.38
Workplace traffic density* 1-99,999	98	0.21	
Workplace traffic density† 0	63	0.28	

*Traffic density (VMT/d/mi^2) within 500 feet of location.
†No measured roads within 500 feet.

concentrations did not change substantially when we excluded smokers and those who consumed grilled meats.

Table 5 presents the correspondence between GIS-based residential traffic density values and self-reported data on traffic exposure. Subjects who reported living on streets with heavy to moderate traffic had mean traffic density values almost four times higher than those who reported living on streets with light traffic. Respondents that reported living within four blocks of a highway also had much higher mean traffic density values as did those who lived in urban areas compared with rural locations. These results were all highly statistically significant (P = 0.0001) and illustrate the large differences in traffic density between self-reported exposure categories.

Discussion

In this study, 1-OHPG levels in urine were related to self-reported exposure to tobacco smoke. We observed much higher 1-OHPG levels among the five smokers. It has been estimated that smoking a pack of cigarettes per day doubles the dose of carcinogenic PAHs a person receives (39). A previous study in Korea found that 1-OHPG concentrations in urine were significantly correlated with cigarette smoking (40). Two studies that measured 1-hydroxypyrene in urine also noted higher levels in smokers than in nonsmokers (41, 42). In the present study, among nonsmokers, the median 1-OHPG levels between those exposed and not exposed to secondhand smoke were not different, but the number of hours of exposure to secondhand smoke was significantly correlated with 1-OHPG concentrations.

1-OHPG levels were related to eating meat prepared using methods (such as grilling, broiling, or roasting) that lead to charring. Previous studies of urinary 1-OHPG concentrations and dietary PAH exposures also found higher levels of 1-OHPG postconsumption compared with preconsumption (43, 44). For adult nonsmokers, it has been estimated that diet accounts for 96% of the potential dose of carcinogenic PAHs (39). Dietary and nondietary (soil and dust) ingestion also accounted for the majority of measured PAH exposure in a recent study of nine young children (45). Barbequed and charbroiled meats have the highest measured PAH levels in food (46). However, due to greater consumption volumes, grains are thought to be the largest contributor of PAHs in the average western diet (47, 48). Our study was not designed to estimate PAH exposures from the intake of grains or foods other than meat during the relevant exposure period for the excretion of PAH metabolites in urine. Additionally, we only obtained information on the doneness of meats consumed by participants during the in-person interviews for the 24-hour period before urine collection. Our results, and those of others, show that the collection of information about the doneness of grilled foods should be considered an important component for future studies (28).

Although previous monitoring studies have identified vehicle traffic as a major contributor to PAH concentrations in outdoor air, we did not see a relationship between survey- or GIS-based measures of proximity to traffic and urinary 1-OHPG levels. A previous study of toll station workers in Taiwan found that 1-OHPG concentrations increased in a dose-response pattern with traffic counts (49). Higher 1-hydroxypyrene levels have also been observed among people occupationally exposed to traffic exhaust, including toll booth attendants, bus mechanics, and waste collection workers (50, 51). However, in this study, participants who spent the most time traveling on roads did have significantly higher 1-OHPG levels than those who spent less time traveling on roads. A previous study found that the concentrations of several measured pollutants, including benzene, carbon monoxide, and particulate matter, were considerably higher inside vehicles traveling along California roads than at ambient monitoring sites (52). In Baltimore, gasoline and diesel vehicles contributed an estimated 16% to 26% of the total PAHs found in urban air (53). Measured levels of PAHs in outdoor air were higher in close proximity to a bus terminal and along bus routes in Massachusetts (54). In Boston, ~46% of PAH mass measured in outdoor air was attributable to primary vehicle emissions near a busy road (55). A study in Denmark found that traffic contributed 60% to 90% of PAHs to outdoor air near a busy street was and 40% to city background air (56). In a study of PAH concentrations measured in indoor air, traffic was also the major outdoor emission source, but the effects of cooking and smoking were also significant (57). Long travel time on roads had a similar influence on 1-OHPG concentrations as exposure to secondhand smoke in this study. Exposure to secondhand smoke ranks third as a major preventable cause of death behind only smoking and alcohol (58). Further research on the health effects of long travel times on busy roads is warranted.

A study of Korean children found a correlation (r = 0.8) between 1-OHPG concentrations and an index of particulate matter pollution across six regions varying in levels of urban development (59). Two studies have observed significantly higher levels of 1-hydroxypyrene in the urine of residents from more urban areas (60, 61), but another study did not find such a trend among children (62). PAH concentrations measured in homes of nonsmokers were also attributed mainly to vehicle emissions in an urban area of Germany (63). We did not observe a difference in 1-OHPG levels between urban and rural residents in our study despite a fairly large difference in the traffic density levels between the two areas. The urban areas included in our study, however, are better described as

Table 4. Spearman rank correlation coefficients for continuous exposure measures and 1-OHPG levels (pmol/mL) in urine

48-h Exposure measure	Reported range	Subjects (*n*)	Spearman correlation coefficient	*P*
Meals eaten with grilled meat	0-4	300	0.16	0.01
No. cigarettes smoked	0-40	304	0.13	0.02
Hours of secondhand smoke exposure	0-16	304	0.12	0.04
Hours of travel on roads	0-8	304	0.09	0.11
Meals cooked by grilling, etc.	0-3	301	0.04	0.47
Residential traffic density*	0-687,250	304	−0.04	0.52
Workplace traffic density*	0-1,499,208	221	−0.09	0.17

*Traffic density (VMT/d/mi^2) within 500 feet of a location.

Table 5. Comparison of survey responses and traffic density values at residence using Kruskal-Wallis one-way ANOVA by ranks

Exposure category of residence	Data source	Subjects (n)	Mean traffic density* (VMT/d/mi^2)	P
Heavy/moderate traffic	Survey	55	78,237	0.0001
Light traffic		248	19,858	
Highway within four blocks	Survey	99	64,790	0.0001
No highway		205	14,569	
Urban residence	GIS	124	61,620	0.0001
Rural residence		180	9,779	

*Within 500 feet of residence.

suburbs, with a population density lower than that often associated with more urban areas.

There are several limitations to our study. 1-OHPG urine concentrations represent exposure during the previous 6 to 35 hours (31). For this study, urine samples were collected over a 24-hour period and our exposure information from the survey covered the 24-hour urine collection period and the preceding 24 hours. Therefore, we might have missed some relevant exposure information, and for some of the information collected, we might not have allowed sufficient time for excretion of this PAH metabolite. This study was cross-sectional with results based on only one measurement and did not allow for the evaluation of intraindividual variation. There are other urinary metabolites that may be potentially useful for determining exposure to traffic exhaust, but these were not measured in this study. A significant correlation was observed between urinary 2-naphthol and ambient particulate matter, suggesting that this may be a good biomarker for inhalation exposure to PAHs (64). Urinary hydroxymetabolites of phenanthrene have been proposed as specific markers of occupational exposure to diesel exhaust but may not be sensitive enough for ambient exposure levels (50).

We did not have information on some potentially important sources of outdoor PAH emissions, including neighborhood wood burning, off-road diesel equipment, and industrial combustion sources (53). A recent study found that roadside PAH concentrations were significantly associated with large diesel vehicle counts but not with total vehicle traffic counts (38). The traffic count data we used in this study did not differentiate between gasoline- and diesel-powered vehicles. Our traffic density measure also did not consider factors that influence the dispersion of pollutants, including wind speed and direction. Due to limitations in the accuracy of the road networks used in this study (65), we were unable to evaluate the relationship between traffic density within a smaller radius (e.g., 150 feet) and 1-OHPG levels. To better estimate PAH inhalation exposures, more detailed emission data are needed in future studies.

Individual metabolic variation, which our analysis did not account for, is likely to be a more important issue. In a previous study, 1-OHPG levels varied 8-fold among individuals exposed to the same amount of PAHs by ingestion (44). Based on metabolic rate variations, the differences in PAH air concentrations in our study were probably too small to detect. Results from a breast cancer study showed that PAH DNA adduct levels were not related to evaluated exposure measures, suggesting that adduct formation may be more closely related to individual metabolism than to dose (15). Glutathione *S*-transferase (GSTM1) genotype has been shown to influence the association between PAH exposure and 1-OHPG levels in urine (25, 40). Several genetic polymorphisms, including a cytochrome *P*450 variant (CYP1A1), glutathione *S*-transferases (GSTT1 and GSTM1), *N*-acetyltransferase (NAT2), and an epoxide hydrolase (mEH3), have also been related to variation in 1-hydroxypyrene levels in urine (42, 66-69).

In this study, self-reported exposure to cigarette smoke, consumption of grilled, broiled, or roasted meat, and time spent traveling on roads were significant predictors of urinary 1-OHPG levels, suggesting that information for these factors collected from individual surveys is valuable in estimating PAH exposures. The results from our comparison of GIS-based traffic density and self-reported traffic levels also show the utility of self-reported data for characterizing proximity to vehicle traffic. The GIS- and survey-based methods we used for estimating proximity to traffic did not predict urinary 1-OHPG concentrations, however. Higher workplace traffic density levels and the association of 1-OHPG levels with self-reported time spent traveling on roads suggest that future studies need to consider these factors in addition to residential traffic density in future GIS-based studies.

Based on our results, 1-OHPG seems to be an effective biomarker for integrating PAH exposures from both inhalation and ingestion. Over 45% of all subjects had 1-OHPG urine concentrations below the detection limit and levels among most nonsmokers that did not eat grilled, broiled, or roasted meat were near the detection limit, indicating that PAH exposures from ambient air were low in this study. Information on key genetic polymorphisms should also be incorporated into future studies using 1-OHPG as a biomarker of exposure to PAHs.

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