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Spatial and Temporal Trends of Polycyclic Aromatic Hydrocarbons and Other Traffic-Related Airborne Pollutants in New York City

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Abstract

Traffic-related air pollutants have been associated with adverse health effects. We hypothesized that exposure to polycyclic aromatic hydrocarbons (PAHs), elemental carbon (EC, diesel indicator), particulate matter ($PM_{2.5}$), and a suite of metals declined from 1998 to 2006 in NYC due to policy interventions. PAH levels from personal monitoring of pregnant mothers participating in the Columbia's Center for Children's Environmental Health birth cohort study, and EC, $PM_{2.5}$, and metal data from five New York State Department of Environmental Conservation stationary monitors were compared across sites and over time (1998–2006). Univariate analysis showed a decrease in personal PAHs exposures from 1998 to 2006 ($p < 0.0001$). After controlling for environmental tobacco smoke, indoor heat, and cooking, year of personal monitoring remained a predictor of decline in Σ_8PAHs ($\beta = -0.269$, $p < 0.001$). Linear trend analysis also suggested that $PM_{2.5}$ declined ($p = 0.09$). Concentrations of EC and most metals measured by stationary site monitors, as measured by ANOVA, did not decline. Across stationary sites, levels of airborne EC and metals varied considerably. By contrast $PM_{2.5}$ levels were highly intercorrelated (values ranged from 0.725 to 0.922, $p < 0.01$). Further policy initiatives targeting traffic-related air pollutants may be needed for a greater impact on public health.

Introduction

Exposure to traffic-related airborne pollutants has been associated with an array of health effects, including decreases in pulmonary and cardiovascular function, asthma, low birth weight, impairment of neurocognitive development, and cancer (1–3). Large cities often carry disproportionate burdens of exposure to air pollution (4) and health consequences (5). Critical to public health strategies is the correct characterization of the spatial and temporal trends incurred by such pollutants, particularly in urban areas where traffic is a major contributor (6).

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The component parts of traffic-related emissions are complex but include byproducts of combustion of fuel and lubricating oils, abrasion products from both vehicles and roadways, and resuspension of road dust (6–8). Incomplete combustion produces both gaseous and condensed particulate emissions that include elemental carbon (EC), PAHs, and certain metals (9). While PAHs can be in either the gas-phase or associated with particulate matter, elemental carbon and metals are associated with particles. Diesel emissions are of particular concern as they produce 30–100 times more particles than gasoline vehicles with emission controls (10). In Europe and the United States, urban traffic contributes 46–90% of the total PAHs in ambient air (11,12). Traffic was also responsible for approximately 20% of the $PM_{2.5}$ in Toronto (13). Morning traffic peaks contributed 19% of the fine particulate material in Los Angeles (14). Brake abrasions, tire wear, traffic-related resuspension of roadway dust, and tailpipe emissions, as well as additives in oil and diesel fuel and aging catalytic converters are known emitters of metals (15,16). Abrasion produces significant amount of particles less than 2.5 μm in diameter even though most of the mass is in particles at larger diameters.

In New York City (NYC), sources of air pollution historically have included coal for space heating (peak impact during the 1920s; ended in the 1960s), oil combustion, and incineration of municipal solid waste (peak impact during the 1930s–1960s; in decline during the 1970s) (17,18). Petroleum-related pollution expanded after 1913 when the first assembly line for cars was put into operation. From the mid 20th Century onward, a large network of freeways was built, and by the end of the century, 31% of New Yorkers lived within 75 m (ms) of a major road and another 36% lived between 75 and 200 ms of major roads (19). Hence, traffic, local and from commuting areas such as New Jersey and Connecticut, became a major contributor of air pollution in NYC (and other urban areas) over the last several decades. Additional outdoor sources in NYC include local and upwind (e.g., Ohio Valley) power source emissions (20). In addition to these outdoor sources of air pollution, indoor sources (e.g., cooking, space heating, environmental tobacco smoke [ETS], use of candles and incense) contribute to personal exposure to air pollutants such as PAH, $PM_{2.5}$, carbon, and trace metals. Daily activities can increase true personal exposure levels beyond those detected by community-based stationary air monitors (21). In NYC, all these sources combined often drive indoor and personal exposure to air pollution (22).

Since the 1970s, Clean Air Act provisions have been important drivers of air quality trends. Recent initiatives included the conversion to ultralow-sulfur diesel fuel, aimed to be 97% cleaner, that occurred voluntarily in NYC over recent years with new bus and truck fleets, and officially as of July 1, 2006 (23). In April 2000, the Metropolitan Transportation Authority of New York announced a fleet-wide plan to reduce emissions from diesel buses, and increase the use of cleaner fuels (e.g., compressed natural gas and ultralow-sulfur diesel fuel) (7) (results posted in ref 23). A national mandate also was passed in October 2006 requiring that cleaner fuel be used on new truck and bus engines to cut their particulate emissions by 90% (www.epa.gov/air/caa/). Despite such legislation, few studies have tracked ambient levels of major traffic-related air pollutants over time. Studies focusing on PAHs are particularly scarce, despite growing evidence of their associated adverse health effects in children (24,25). We hypothesized that exposure to PAHs, measured by personal monitoring and $PM_{2.5}$, diesel, and trace metals, measured at community-based stationary sites, may have undergone measurable declines from 1998 to 2006 in NYC. Our strategy was to compare data collected during this period from five local stationary sites across NYC, as well as from personal monitoring of pregnant women conducted as part of a longitudinal birth cohort study in northern NYC.

Experimental Procedures

Personal Monitoring for PAHs

Personal exposure during pregnancy to a suite of PAHs was collected by the Columbia Center for Children's Environmental Health (CCCEH) from women recruited between 1998 and 2006, as previously described in refs 26,27. Monitoring of PAHs was part of a longitudinal, cohort-based study investigating the effects of prenatal and postnatal air pollution exposures on health effects in children. Included in the cohort were nonsmoking Dominican and African American women from Northern Manhattan and the South Bronx (N = 743). Participants carried mobile, personal monitors for 48 h of continuous monitoring during later pregnancy (mean: 34 weeks; range: 19–42 weeks). These backpacks were placed close to the breathing zone during wake time and placed beside the participant's bed at night (28). At enrollment, a questionnaire was administered that elicited information on behaviors and daily activities related to traffic and combustion byproduct and ETS exposures. A second questionnaire was administered during the 48 h personal PAH collection that queried ETS scenarios (home, work, and elsewhere), charcoal broiling, grilling, frying, and sautéing (frequency, duration), use of candles and incense, exposure to heating sources, time spent outdoors, and means of transportation (27, 29).

Monitors carried by participants were fitted with a quartz filter and polyurethane foam (PUF) cartridge that were analyzed by Southwest Research Institute for eight PAHs: benz(a) anthracene (BaA), benzo(b)fluoranthene (BbF), benzo(k)fluoranthene (BkF), benzo(ghi) perylene (BghiP), benzo(a)pyrene (BaP), chrysene/isochrysene (Chrys), dibenz (a,h) anthracene (DahA), indeno(1,2,3-cd)pyrene (IP), as described in ref 30. Analytical protocol and details of quality controls are described in the Supporting Information (SI).

Stationary Site Monitoring for Carbon, $PM_{2.5}$, and Metals

Data on carbon (elemental, total, and organic), $PM_{2.5}$, sulfur, and 10 elements (Ni, Ca, V, Pb, Fe, Cu, Mn, Cl, Zn, and Br), hereon referred to as "metals," were provided by the New York State Department of Environmental Conservation (DEC). Publicly available data sets from five DEC sites in Manhattan, the Bronx, and Queens were downloaded (http://www.dec.ny.gov/). These stations were the New York Botanical Gardens (NYBG), Morrisania II (MII), Canal Street (CANL), Intermediate School 52 (IS-52), and Queens College (PS-219). Site characteristics are described (SI Table S.1.). Sites collected particulate and speciated data at approximately 72 h continuous and variable intervals, in most cases beginning in 1999. DEC's instrumentation, analytical and quality control protocols (including collocation and duplication of samples) are posted in refs 29, 31–33. Analysis was conducted by the Research Triangle Institute (Research Triangle Park, NC), and protocols were standardized across the years queried. Three DEC stations (NYBG, IS-52, and MII) were located in the area where CCCEH conducted personal PAH monitoring (Bronx County, NY). Morrisania II collected only $PM_{2.5}$ data; the other four sites collected both particulate and speciated data. 63.9% of DEC's EC and metal samples received at least one of 109 different types of quality control flags. Indications included protocol infractions (e.g., shipping temperature of samples outside of specifications) (most common), equipment malfunction, and atypical incidents in the local environment (e.g., sandblasting, unusual traffic congestion).

Statistical Analysis

Ambient monitoring data from DEC's Web sites were downloaded. Because DEC's readings were not available for every day at each site, and data were not always available on the same day across all sites, these data were aggregated by week, month, year, and site. This allowed us to maintain compatibility across sites as each data record in the aggregated file contained average values in weeks and months. In order to assess the relationship between airborne

personal monitoring of individual CCCEH cohort subjects and DEC site-based readings, cohort data also were aggregated by week, month, and year, and then merged by month and year with aggregated DEC data.

Data from personal monitoring and from DEC's Web sites were analyzed using SPSS 15.0.1.1 for Windows (Chicago, release 3 July 2007). For quality control, $PM_{2.5}$ duplicate samples were verified as highly correlated at two sites tested (Canal Street: r=0.88, $p < 0.001$; IS-52 r=0.810, $p < 0.001$). The PAHs from personal air monitoring of CCCEH cohort is reported as airborne concentrations of the sum of the eight PAH compounds measured (Σ_8PAH). Ten PAH samples were excluded from statistical analyses because of technical failures resulting in a final PAH sample size $N = 733$.

The distribution of continuous variables was examined and data were natural-log transformed when necessary to reduce variance and fulfill the distribution requirements of the parametric tests when used. Initially, descriptive statistics were compared using the complete DEC data set. Descriptive statistics were repeated following exclusion of all data points that were flagged for quality control issues that would affect data collection (e.g., equipment malfunction). Analyses continued with only data points thus verified for a total sample size of $n = 1126$ for EC and metals and of $n = 6955$ for $PM_{2.5}$. To compare pollutant levels across sites, and to compare individual pollutants within each site, nonparametric Spearman correlations of weekly and monthly aggregate data were used.

To screen for temporal variations among Σ_8PAH, EC, $PM_{2.5}$, sulfur, and a suite of 10 metals, analyses of variance (ANOVA) were conducted. The effect of year of PAH monitoring was analyzed further using regression modeling controlling for sautéing and/or frying at home, amount of time of indoor heat use, home ETS exposure (all recorded during the 48 h monitoring), ethnicity, and indoor heating season (October 1st to May 31st, according to NYC regulations). *Year of monitoring* was added to the regression models as an interval variable and analyzed as the independent predictor of interest. In exploratory analyses, the effect of summer was evaluated through regression modeling by stratifying summer data (defined as July–August) in comparison to the rest of the year. Temporal changes of individual PAHs were also measured through ANOVA.

Results

CCCEH participants were Dominican (64.1%) and African American (35.7%). Ninety one percent of the participants received Medicaid, 41.4% received public assistance, and 29.3% had less than a 12th grade education at time of enrollment. Participants reported exposure to ETS (31.1%) and sautéing and or frying (50.2%) at some point during monitoring. The mean (range) of airborne PAH concentration (ng/m^3) in personal air monitor collected are summarized in SI Table S.2.

Spatial Distribution Across Stationary Sites

Total, elemental, and organic carbons were highly correlated across stationary monitoring sites (Spearman r value ranges: 0.417 to 0.974; e.g., IS-52 $r = 0.812$ for total vs elemental carbons [ECs]; $r = 0.970$ for total vs organic carbon; and $r = 0.674$ for elemental vs organic; $p < 0.001$ in all cases). In light of these correlations, and the literature suggesting that EC measurements can be used as surrogates for diesel particles which are of concern for this study (4), we focused on EC levels in subsequent analyses. EC levels varied across the stationary sites, with intersite correlations ranging from $r = 0.276$ (P = NS) to $r = 0.548$ ($p < 0.001$). As shown by SI Table S.3, EC levels correlated with metal levels that previous studies (15) have associated with traffic tailpipe emissions, brake and tire wear, and roadway dust resuspension including Zn, Pb, Fe, Ca, Ni, Cu, and Br.

Correlations between EC levels from stationary site monitoring and Σ_8PAH levels from personal monitoring were not significant for three (IS-52, CANL, PS-219) of the four sites compared, including one (IS-52) located within CCCEH's enrollment area. However, EC from NYBG, also located in the CCCEH area, was moderately but significantly correlated with Σ_8PAH ($r = 0.290$, $p = 0.027$). Nickel and zinc were consistently correlated with Σ_8PAH. Sulfur levels correlated negatively with Σ_8PAH at IS-52 ($r = -0.297$, $p = 0.033$) (SI Table S.3).

$PM_{2.5}$ levels were highly correlated across the five sites with correlation coefficients ranging from 0.725 to 0.922 ($p < 0.01$). Among the particle components analyzed, sulfur was the most highly correlated with $PM_{2.5}$, but correlations between $PM_{2.5}$ and sulfur varied from 0.327 to 0.543 ($p = 0.019$ and $p < 0.001$ respectively) across the four sites (NYBG, IS-52, PS-219, and CANL). Correlations between $PM_{2.5}$ and EC differed across the sites (Spearman *r*'s from 0.044, p = NS, to 0.417, $p = 0.004$). Notably, significant correlations were not found between $PM_{2.5}$ levels measured by local stationary site monitors and PAHs (neither with Σ_8PAH or the individual PAHs) measured by personal monitoring (SI Table S.3).

Temporal Distributions

Σ_8PAH levels assessed by personal monitoring showed a significant decrease over the period of time analyzed (from 1998 to 2006) when both monthly and yearly means were examined ($p < 0.0001$ in both cases) (Figure 1). The declines were apparent when the individual PAHs were assessed as well (SI Figure S.1). Statistically significant declines in Σ_8PAH levels were detected during both the summer and winter months ($p < 0.0001$ in both cases). In exploratory regression analyses, summer (July–August) was a significant predictor of Σ_8PAH decline ($\beta = -0.244$, $p < 0.001$) in addition to *year of monitoring*.

Multivariate regression modeling was conducted to assess the contribution of the covariates including ethnicity, indoor cooking, ETS exposure and indoor heating during PAH monitoring (Table 1). Despite the overall decrease in reported ETS exposure among study participants (46% in 1998 to 20% in 2006, assessed at enrollment) year of monitoring remained an independent predictor for the decline in Σ_8PAH ($\beta = -0.269$, $p < 0.001$). ETS was not a significant predictor of Σ_8PAH levels ($\beta = 0.048$, $p = 0.233$). Self-reported cigarette exposure during monitoring was low and infrequent (SI Table S.4), supporting the apparent nonsignificant contribution of ETS exposure to the decline in Σ_8PAH. Similarly, despite the contribution of indoor heating to PAH levels (ascertained by queries on indoor heating use and by use of the dichotomous variable of *heating season*), year of monitoring remained a significant predictor of Σ_8PAH, yielding an absolute number that exceeded the beta for "*heat on*" (Table 1). Given the infrequent use of candles (18.5%), incense (8.8%), broiling (14%), and grilling (1.6%) in the cohort, such potential covariates were excluded from the models.

In addition, mean levels of $PM_{2.5}$ decreased from 15.44 ug/m^3 in 1999 to 12.63 ug/m^3 in 2006, the latter reaching a level below the national attainment standard of 15 ug/m^3 (http://www.epa.gov/air/particlepollution/). While such trend was not significant when ANOVA analyses of yearly $PM_{2.5}$, means were conducted (p = NS), analyses of linearity showed a significant downward trend in $PM_{2.5}$ concentrations from 1999 to 2006 ($p = 0.009$). Significant temporal changes were not apparent for EC and sulfur levels measured at the stationary sites between 2000 and 2006, the time period of data analysis (SI Figure S.2). Among the metals analyzed, only lead and calcium showed statistically significant changes at two stations. At IS-52, lead decreased from 0.007 to 0.005 $\mu g/m^3$ ($p < 0.001$), and calcium increased from 0.047 to 0.105 $\mu g/m^3$ ($p < 0.001$). At NYBG, lead decreased from 0.007 to 0.005 $\mu g/m^3$ ($p < 0.001$), and calcium increased from 0.048 to 0.113 between 2000 and 2005 ($p < 0.001$).

Discussion

We hypothesized that exposure to PAHs, EC, $PM_{2.5}$, and a suite of metals declined from 1998 to 2006 in NYC, as a result of policy interventions. These pollutants are associated with traffic-related emissions as well as an array of adverse health effects. Notably, this is the first study to assess temporal trends in personal PAH exposure and to report a significant downward trend in the levels of Σ_8PAH. We also report a significant downward trend in $PM_{2.5}$ concentrations measured by stationary sites. In contrast, EC and metals measured by local stationary monitors did not show similar declines. With the exception of $PM_{2.5}$, levels of pollutants varied considerably across NYC sites, consistent with the unequal spread of traffic emissions within the study area.

Personal Σ_8PAH levels were very similar for the pregnant women who reported being exposed to ETS as compared to those who reported not being exposed to ETS. Hence, reported ETS, in this cohort of nonsmoking participants, appears independent of the decline in Σ_8PAH from 1998 to 2006 (Figure 2a and b), strongly suggesting that other PAH sources were more important to the personal exposure of these women. Though some studies have reported that ETS questionnaires yield reliable data (34), others have advised against their use (35). However, as reported in previous studies, our group has validated the ETS self-reports analyzed in this paper by establishing significant correlations with cotinine levels in urine samples from participating mothers and children ($r = 0.4$, $p < 0.001$ in both cases) (36). The chi-square association between self-reported ETS and cotinine in participating mothers has been found to be $\chi^2 = 48.57$ ($p < 0$ 0.001) (37).

The finding that ETS appears independent of the decline in Σ_8PAH seems to be in conflict with literature showing the importance of cigarette smoking to PAH exposure (38). However, there are multiple explanation for this apparent inconsistency. First, PAH emissions from cigarettes are dominated by sidestream cigarette smoke where lower molecular weight PAHs are more prevalent than the high molecular weight PAHs measured in this study (38). Although the number of participants reporting ETS decreased over time, the overall change in the number of cigarettes smoked in the womens' presence during monitoring did not appear to decrease (data not shown) and was low (geometric mean of 3.06 cigarettes per day, including indoor and outdoor exposure) (SI Table S.4). As these were nonsmoking participants in their third trimester of pregnancy, one may presume that it is unlikely that these isolated smoking events occurred in confined airspaces such as a car. Hence, the portion of the ETS exposure that occurred in outdoor locations may have an even smaller impact.

In addition to being independent of ETS status, the decreases in Σ_8PAH levels remained independent of other covariates such as ethnicity, cooking behaviors, as well as indoor heating which was assessed both by questionnaire data, and by dichotomizing across the presence or absence of the regulated indoor heating season. However, as heating season appeared to be a significant contributor to Σ_8PAH levels, the decline may be partly explained by changes in the use or configuration of domestic heating systems that were not measured by the study. As exploratory analyses also showed that summer was also a predictor of the decline in the Σ_8PAH, other unmeasured seasonal effects seemed to have contributed as well.

It is reasonable to conclude that policy initiatives directed at reducing traffic-related air pollution and promotion of clean air may have indeed impacted air pollution levels in New York City. This is supported by the fact that improvements were more apparent for PAHs than for $PM_{2.5}$ and that sulfur, an important component of $PM_{2.5}$ that is emitted in large quantities by electric utilities in upwind regions, did not decline in NYC. Decreases in other components of particulate mass that were not measured by this study (e.g., nitrates, which derive from motor-vehicle related NO_xemissions) may account for the moderate decline in $PM_{2.5}$ levels.

Lead levels decreased to almost 3 orders of magnitude below the threshold currently established by National Ambient Air Quality Standards (NAAQS): 1.5 $\mu g/m^3$. The minor increases of calcium (not a regulated pollutant) are negligible by current NAAQS criteria (www.epa.gov/air/) and could reflect increases in city construction.

The inability to detect temporal declines in EC levels may reflect, in part, methodological issues at stationary sites, including possible contamination in the samples between 2001 and 2003, as well as changes in set flow-rates across time periods and sites (Dirk Felton, personal communication). The spatial variability in EC and metal levels in NYC suggests that these pollutants are strongly influenced by local sources of exposure, consistent with the large spatial variability in traffic density. In contrast to the local variability of carbon and metals, $PM_{2.5}$ levels measured at different sites throughout the city were strongly correlated. However, judging by highly differing correlations between $PM_{2.5}$ and the other pollutants at each site, the composition of fine particulate mass at each site also seems variable. The exception was sulfur, which was consistently correlated to $PM_{2.5}$ corroborating that sulfur is also an important component of the fine particulate mass in NYC.

The results suggest an overall absence of a correlation between PAHs by personal monitoring and EC measured by stationary sites, likely reflecting the contribution of different local sources of air pollution in the study area. Previous studies have shown that PAHs and EC levels had similar patterns of temporal variation on decadal time scales: for example, analyses of a sediment core collected from an urban lake in Seattle, WA, showed that graphitic black carbon and PAHs had similar patterns of variation between the 1950s and 1970s (39). In comparison, different profiles for both pollutants obtained from sediment cores from Central Park Lake (New York, NY) between the 1870s and 1990s using similar analyses, suggest that they were emitted by sources that varied temporally (17). Other than variable sources, the absence of a correlation between these two pollutants in the study area could be attributed to discrepancies between sampling methods (i.e., stationary rooftop sites vs personal sampling at breathing zone). Notably, stationary site monitors at rooftop level are regulated to avoid oversampling of local sources such as roadways. By contrast, personal monitoring more readily reflects local and microenvironment sources (40) including indoors sources and those related to personal activities such as cooking.

It is also possible that absence of correlation between PAH and EC is explained by EC monitoring being limited to a few sites, only three of which were located in the general area where personal monitoring was conducted. The methodological issues related to EC sampling at the stationary sites may also explain the absence of a correlation. Additional preliminary data from the CCCEH cohort using residential monitoring of the same suite of PAHs measured in the same laboratory conducted during a similar time period seem to support these arguments. PAHs and EC levels during two-week indoor residential monitoring sessions were highly correlated (Spearman $r = 0.518$, $p < 0.0001$, $n = 148$). The placement of the NYBG stationary monitor within the CCCEH study area may explain the single positive correlation between PAH measured by CCCEH and EC measured at NYBG.

We acknowledge limitations in our approach. Variations in testing procedures (e.g., collection time), exclusion of air exchanges as a variable in the multivariate modeling, possible photodegradation of BaP, and the limited repertoire of individual PAHs measured restrict more substantiated source apportionment from this data set (41–43). Examination of vehicle transportation statistics could clarify further the issue of source apportionment. Other studies have used PAH ratios for source apportionment analysis (41–45). However, because significant differences exist between the collection methods used to determine ratio apportionment and our methods of PAH collection, we were precluded from analyzing these ratios. Reductions in Σ_8PAH over time also could have occurred as a result of unmeasured cohort characteristics

between those recruited during earlier years versus later years (e.g., ETS exposure not ascertained by questionnaire), or other confounders not entered in the model. Stationary sites were not located in the immediate area of the women's residences and may not detect as readily local traffic-related sources. Additional stationary and continuous background monitoring would be needed to help distinguish residential exposures from those related to meteorological or seasonal effects (30). PAH air monitors were not collocated along the community-based stationary monitors, limiting the spatial inferences drawn from correlations between personal and stationary sources.

In sum, a decline in Σ_8PAH, as measured by personal monitoring of pregnant women living in Northern Manhattan from 1998 to 2006, is evident. Further source apportionment analyses may clarify the differential decline of PAHs versus other airborne toxicants. Decline in $PM_{2.5}$ is also suggested by our analyses. The reduction in PAHs and $PM_{2.5}$ may reflect partially successful policy initiatives that have targeted emissions from traffic-related and regional sources. However, in the CCCEH cohort, prenatal exposure to these ambient concentrations of PAHs has been associated with reduced head circumference in neonates (46) developmental deficits at age three (3), and parental report of difficulty breathing and probable asthma at ages 2 years in children (24). Comparable levels of urban diesel and $PM_{2.5}$ emissions have been associated with decreased lung function in children and adolescents (47). Combined, these suggest that further interventions and vigilance are needed. These may include strategies to reduce air pollution from indoor sources. The improvements in personal PAH levels and PM detected here only reinforce the need to continue to implement policy initiatives targeting traffic-related air pollutants for a greater impact on public health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

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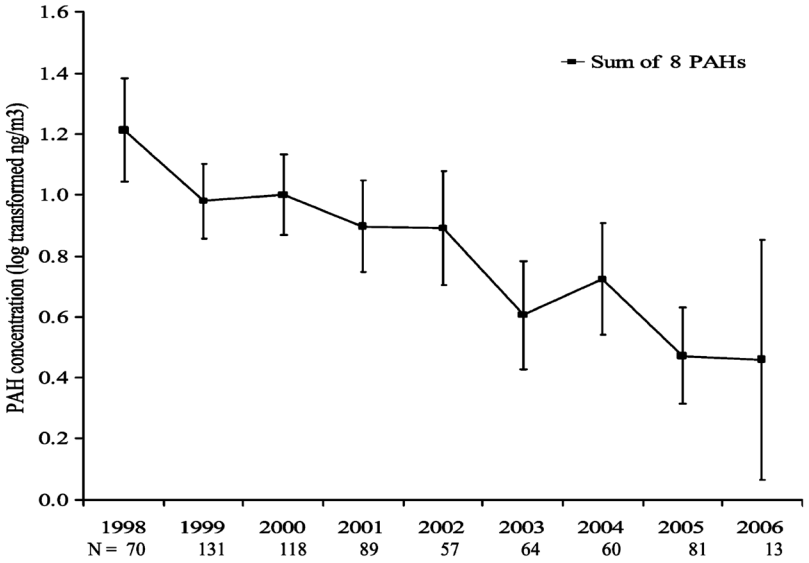

FIGURE 1.

Personal exposure to the sum of 8 PAHs (Σ_8PAH) from 1998 to 2006 Data (n = 733) are displayed as natural log-transformed averaged each year. One error bar = 95% CI; Σ_8PAH $p < 0.0001$, ANOVA.

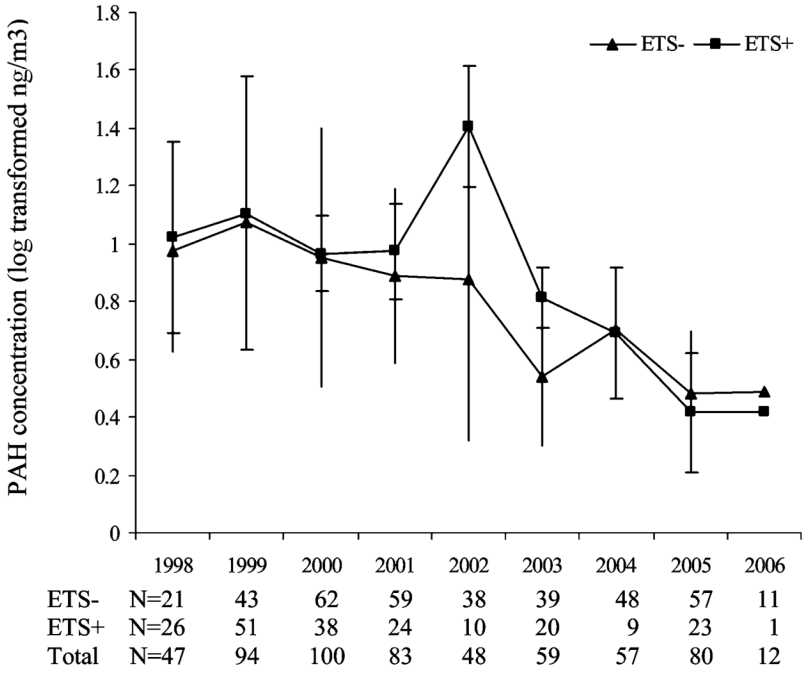

FIGURE 2.

Personal exposure to Σ_8PAH, stratified by ETS exposure Data (n = 683) are displayed as natural log-transformed averaged each year. N = 50 excluded because of missing ETS questionnaire data. ETS includes exposure at home or elsewhere. One error bar = 95% CI. Personal exposure to Σ_8PAH, stratified by report of ETS during personal monitoring. ETS appears to have no effect (p = NS), Tests of Between-Subjects Effects. Year of monitoring had a significant main effect (p = 0.001) (+) ETS exposure reported either at home, work or other location. (−) No ETS exposure reported either at home, work or other location.

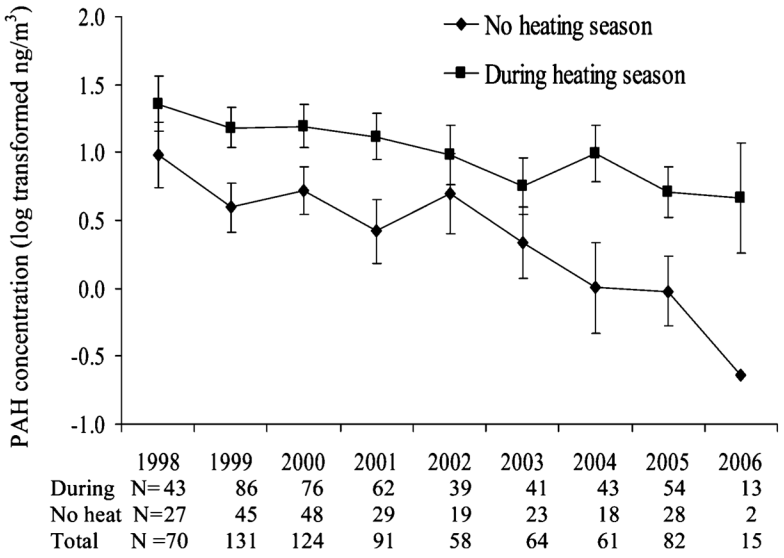

FIGURE 3.

Personal exposure to Σ_8PAH stratified by heat season Data (n = 683) are displayed as natural log-transformed averaged each year. N = 50 excluded because of missing data on heat season. One error bar = 95% CI. Heating season is defined as Oct. 1st to May 31st, according to NYC regulations. Personal exposure to Σ_8PAH, stratified by heat season ($p < 0.0001$ for both) during monitoring, ANOVA.

TABLE 1

Year of Monitoring Predicts Σ_8PAH

	Σ_8PAH [a]	
	β	**P**
Ethnicity	0.004	0.919
frying/sautéing	−0.039	0.319
heat on[b]	0.116	0.025
ETS exposure[c]	0.048	0.233
heating season[d]	0.277	0.000
year of monitoring	−0.269	0.000

[a] Data were natural log-transformed to reduce variance and fulfill the distribution requirements of shown tests.

[b] "Heat on" refers to heating system in use during monitoring.

[c] Exposure during PAH monitoring.

[d] "Heating season" refers to the time period between October 1st to May 31st according to NYC regulations; coding was 1 for heating season and 0 for rest of the year.