Effects of Transplacental Exposure to Environmental Pollutants on Birth Outcomes in a Multiethnic Population

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Inner-city, minority populations are high-risk groups for adverse birth outcomes and also are more likely to be exposed to environmental contaminants, including environmental tobacco smoke (ETS), polycyclic aromatic hydrocarbons (PAHs), and pesticides. In a sample of 263 nonsmoking African-American and Dominican women, we evaluated the effects on birth outcomes of prenatal exposure to airborne PAHs monitored during pregnancy by personal air sampling, along with ETS estimated by plasma cotinine, and an organophosphate pesticide (OP) estimated by plasma chlorpyrifos (CPF). Plasma CPF was used as a covariate because it was the most often detected in plasma and was highly correlated with other pesticides frequently detected in plasma. Among African Americans, high prenatal exposure to PAHs was associated with lower birth weight (p = 0.003) and smaller head circumference (p = 0.01) after adjusting for potential confounders. CPF was associated with decreased birth weight and birth length overall (p = 0.01 and p = 0.003, respectively) and with lower birth weight among African Americans (p = 0.04) and reduced birth length in Dominicans (p < 0.001), and was therefore included as a covariate in the model with PAH. After controlling for CPF, relationships between PAHs and birth outcomes were essentially unchanged. In this analysis, PAHs and CPF appear to be significant independent determinants of birth outcomes. Further analyses of pesticides will be carried out. Possible explanations of the failure to find a significant effect of PAHs in the Hispanic subsample are discussed. This study provides evidence that environmental pollutants at levels currently encountered in New York City adversely affect fetal development. *Key words:* **birth outcomes, development, environmental, ETS, PAH, pesticides, pollutants, prenatal.** *Environ Health Perspect* **111:201–205 (2003). [Online 31 October 2002]**

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The impact of environmental toxicants on children's health is increasingly being recognized as significant (Faustman 2000; GBPSR 2000; Landrigan et al. 1999; Perera et al. 1999; U.S. EPA 1996). Human and experimental studies indicate that the fetus and infant are more sensitive than adults to many environmental toxicants, including environmental tobacco smoke (ETS), polycyclic aromatic hydrocarbons (PAHs), and pesticides (Calabrese 1986; Mott et al. 1994; National Academy of Sciences 1993; WHO 1986; Whyatt and Perera 1995). Urban, minority populations represent high-risk groups for adverse birth outcomes (New York City Department of Health 1998; Perera et al. 2002). These same populations are likely to be more heavily exposed to ambient air pollution, ETS, and pesticides (Chen and Petitti 1995; Heritage 1992; Metzer et al. 1995; Olden and Poje 1995; Pirkle et al. 1996; Wagenknecht et al. 1993; Wernette and Nieves 1992). However, only limited information is available on the extent and impact of prenatal exposure to these environmental contaminants on fetal growth and development. Etiologic studies have largely been ecologic, lacking individual-level data on exposure.

In the present study we evaluated the effects of prenatal exposure to common urban pollutants: airborne PAHs monitored during pregnancy by personal air sampling of the mother; along with ETS, estimated by plasma concentrations of cotinine; and the organophosphate (OP) pesticide (CPF), estimated by plasma CPF concentrations. In addition to being genotoxic and carcinogenic, PAHs such as benzo[*a*]pyrene (BaP) are endocrine disruptors (Bostrom et al. 2002; Bui et al. 1986; Davis et al. 1993). Prior laboratory and two human studies in Central Europe indicate that transplacental exposure to PAHs at relatively high concentrations (annual average airborne concentrations of 7–17 ng/m^3 BaP in the human studies) is associated with adverse birth outcomes (Barbieri et al. 1986; Bui et al. 1986; Legraverend et al. 1984; Perera et al. 1998; Dejmek et al. 2000). ETS is a complex mixture of over 4,000 chemicals, including PAHs and carbon monoxide (Leikauf et al. 1995). Prenatal exposure to tobacco smoke has been associated with deficits in birth weight, birth length, and cognitive functioning at age 3 (Janerich et al. 1990; Martinez et al. 1994; Schuster-Kolbe and Ludwig 1994; Sexton et al. 1990). OP pesticides can act as developmental toxicants. For example, CPF exerts neurodevelopmental and/or behavioral effects in experimental studies when administered during gestation or postnatally (reviewed in (Eskenazi et al. 1999; GBPSR 2000; Landrigan et al. 1999; Whyatt et al. 2002). Effects on human fetal development have not yet been evaluated. As previously reported, we have obtained multiple measures of pesticides on our parent cohort of pregnant women and newborns (Whyatt et al. 2002). In the present analysis, CPF was selected as a covariate because it was the most commonly detected pesticide in plasma samples and because CPF levels in cord plasma were highly correlated with those of other frequently detected pesticides in plasma, including the organophosphate diazinon and carbamate bendiocarb (r = 0.8 and r = 0.5, respectively; p < 0.001, Spearman's rank). CPF and diazinon have been used widely on fruits and vegetables and in treatment of homes, although all residential uses are being phased out (Adgate et al. 2001; U.S. EPA 2002).

Here we tested the hypothesis that prenatal exposure to environmental pollutants is negatively associated with birth weight, length, and head circumference, after controlling for the effects of known physical, biologic, and toxic determinants of fetal growth. As reported previously, the study cohort has substantial exposure to multiple contaminants during pregnancy (Perera et al. 2002; Whyatt et al. 2001, 2002). Specifically, analysis of PAHs in

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air samples from the first 250 subjects showed that all samples had detectable levels of one or more carcinogenic PAHs, ranging over 4 orders of magnitude (Perera et al. 2002). Almost half of the mothers and infants initially enrolled had cotinine levels indicative of ETS exposure (≥ 0.05–25 ng/mL). Maternal and newborn plasma cotinine levels were significantly higher for mothers who reported smoking by others in the household than for mothers who reported no smoking in the home ($p < 0.001$). In addition, 85% of the subjects reported that some form of pest control was used during pregnancy, and 35% reported that their homes were sprayed by an exterminator during pregnancy (Whyatt et al. 2002). One-third of pest control users also reported use of can sprays or pest bombs during pregnancy (Whyatt et al. 2002). Pesticide concentrations measured in a subset of the prenatal air monitoring filters showed that 100% of the women had detectable exposure to multiple pesticides, including CPF at levels ranging from 0.7 to 193 ng/m^3 (Whyatt et al. 2002). CPF was significantly higher in African Americans than Dominicans in the full cohort after controlling for neighborhood of residence (Whyatt et al. 2002).

Methods

Study subjects. Study subjects are Dominican and African-American women residing in Washington Heights, Central Harlem, and the South Bronx, New York, who delivered at New York Presbyterian Medical Center (NYPMC), Harlem Hospital (HH), or their satellite clinics (Perera et al. 2002; Whyatt et al. 2002) (see Table 1 for demographic and exposure characteristics of the population). Ethnicity was self-identified. Nonsmoking women, ages 18–35, who registered at the obstetrics and gynecology clinics at NYPMC and HH by the 20th week of pregnancy, were free of diabetes, hypertension, or known HIV, and resided in the area for at least one year were eligible. Subjects included in the present analysis are those with valid prenatal personal monitoring data on PAHs, cord or maternal blood samples, complete questionnaire data, and birth outcome data. Subjects with missing information on any of these data points were excluded from the analysis. Seven subjects with plasma cotinine concentrations > 25 were excluded to rule out active smoking. Analyses were also run restricting to cotinine ≤ 15, which has also been used as a cutoff for ETS. There were no significant differences in sociodemographic characteristics or levels of exposure between subjects with missing data and those included in the present sample.

Personal interview. A 45-minute questionnaire was administered by a trained bilingual interviewer during the last trimester of pregnancy. The questionnaire included demographic information, lifetime residential history (country of birth, location, and duration of residence), travel outside the current area of residence during the past year, history of active and passive smoking [household members who smoke and estimated cigarettes smoked per day by smoker(s)], alcohol use during each trimester of pregnancy, and consumption of PAH-containing meat (frequency of consumption of fried, broiled, and barbecued meat). Socioeconomic information related to income and education was also collected. The questionnaire was based on that used in a prior study of women and newborns and adapted for the New York City population (Perera et al. 1998).

Prenatal personal PAHs assessment. During the third trimester of pregnancy, women were asked to wear a small backpack containing a personal monitor during the daytime hours for 2 consecutive days and to place the monitor near the bed at night. The personal air sampling pumps operated continuously over this period, collecting vapors and particles of ≤ 2.5 μg in diameter on a precleaned quartz microfiber filter and a precleaned polyurethane foam (PUF) cartridge backup. The samples were analyzed at Southwest Research Institute (SwRI) for eight carcinogenic PAHs: benz[*a*]anthracene, chrysene, benzo[*b*]fluroanthene, benzo[*k*]fluroanthene, B[a]P, indeno[1,2,3-*cd*]pyrene, disbenz[*a,h*]anthracene and benzo[*g,h,i*]perylene as described (Camann et al. 1995; Geno et al. 1993; Majumdar et al. 1993). To determine whether subjects complied with requests to carry backpacks equipped with environmental monitoring devices, motion detectors were installed in the backpacks of randomly selected women. For the average woman, nearly 95% of the total number of motion detections occurred during waking hours, consistent with the verbal reports of our subjects that they were complying with our request to wear the backpacks during daytime hours over the environmental monitoring period.

For quality control, each personal monitoring result was coded as to accuracy in flow rate, time, and completeness of documentation. A code of 0–1 indicated high quality, 2 indicated intermediate quality, and 3 indicated unacceptable quality. We restricted analysis to 263 subjects with code 0,1 samples and other relevant data (excluding 19 subjects with code 2 and 3 samples).

Biologic sample collection and analysis. Maternal blood (30–35 mL) was collected within 1 day postpartum, and umbilical cord blood (30–60 mL) was collected at delivery. Samples were transported to the laboratory immediately. The buffy coat, packed red blood cells, and plasma samples were separated and stored at –70°C. A portion of each sample was shipped to the Centers for Disease Control and Prevention (CDC) for analysis of cotinine and pesticides. Plasma cotinine was analyzed by the CDC using high-performance liquid chromatography atmospheric-pressure ionization tandem mass spectrometry as described (Bernert et al. 1997, 2000). Plasma levels of CPF were analyzed using isotope dilution gas chromatography–high resolution mass spectrometry as described (Barr et al. 2002).

Measures of fetal growth. Information was abstracted by the research workers from mothers' and infants' medical records following delivery including date of delivery, gestational age at birth, infant sex, birth weight, length, head circumference, infant malformations, Apgar scores, maternal height, prepregnancy weight, total weight gain, complications of pregnancy and delivery, and medications used during pregnancy.

Statistical analysis. To exclude active smokers, we restricted analyses to women and newborns with cotinine levels ≤ 25 ng/mL. Analyses were also done further restricting to ≤ 15 ng/mL (excluding 3 subjects). Because the eight PAH air concentration measures were significantly intercorrelated (r values ranging from 0.45–0.94; all p-values < 0.001 by Spearman's rank), a composite PAH variable was computed. This summed measure was dichotomized at the median (2.66 ng/m^3) to obtain a measure of high or low exposure. The dichotomous variable was used in the regression analysis because it is less vulnerable to error and uncertainty in the monitoring data. The results were similar when the continuous PAH variable was used. The maternal and cord plasma concentrations of both cotinine and CPF were significantly correlated ($r = 0.88$, $p < 0.001$ for cotinine; $r = 0.6$, $p < 0.001$ for CPF by Spearman's rank). Therefore, in cases where the umbilical cord levels were not available, the mothers' values were used. For CPF, we used the formula derived from the regression model: Ln newborn CPF = 0.46 + 0.61 × Ln maternal CPF. Cotinine and CPF and birth outcomes were log (Ln) transformed to provide a better fit to the data and/or to approximate the normal distribution and stabilize the variance. The relationships between the exposure variables and the birth outcomes were analyzed by multiple regression, adjusting for known or potential confounders, and including all tests of interactions between ethnicity and exposures. In addition to PAHs dichotomized as high/low and CPF as a continuous variable, the final regression model included covariates representing known or suspected risk factors that were associated with birth outcomes ($p \leq 0.1$). To measure the contribution of antenatal toxicant exposures to birth outcomes, we conducted multiple regression analyses. As shown in Table 2, regression analysis

proceeded in several steps. Model 1 evaluated the effects of PAHs, adjusting for potential confounders including body mass index (BMI), parity, gestational age, and infant sex. Cotinine, income, and alcohol consumption were not significant predictors of outcomes ($p > 0.1$) and were not included. Maternal age and parity were significantly intercorrelated ($r = 0.26$, $p < 0.001$) and inclusion of age instead of parity gave similar results. Because CPF was associated with decrements in birth weight and birth length after adjusting for the covariates in model 1, model 2 included CPF as a covariate (Table 2). In a separate model, because nonsmoking-related airborne PAHs were of primary interest, we adjusted for dietary PAHs as well as cotinine in addition to the covariates in model 1. The test for interaction between PAHs and ethnicity on birth outcomes was significant (see "Results"); therefore, stratified analyses were carried out by race/ethnic group. The percent reduction in each birth outcome associated with high prenatal PAHs was calculated using the formula [1–exp (β)] × 100%.

Results

Demographic and exposure characteristics for the subjects included in the present analysis are provided in Table 1 together with summary data on measured levels of PAHs, cotinine, and CPF. The subset did not differ from the overall cohort in terms of demographic variables. As noted above and in Table 1, the present subset included only nonsmoking women (cotinine ≤ 25 ng/mL). The results were unchanged when we repeated the analyses restricting to women with cotinine ≤ 15 ng/mL. Among subjects in the present analysis, 100% had detectable inhalation levels of one or more PAHs. Total PAH exposures averaged 3.7 ng/m^3 and varied substantially among the women, with a range of 0.36 ng/m^3 to 36.47 ng/m^3. Forty-two percent of mothers and 45% of newborns had cotinine levels > 0.05 and ≤ 25 ng/mL, indicative of ETS exposure. CPF was detected in 98% of the maternal and 94% of the cord samples, with means of 7.6 pg/g in cord blood and 7.1 in maternal blood. PAHs were correlated with cotinine ($r = 0.26$, $p < 0.001$). Self-reported ETS and plasma cotinine differed by ethnicity, with African Americans being significantly more likely to report ETS exposure ($p \leq 0.01$) and to have higher cotinine levels ($p \leq 0.01$).

The mean birth weight was 3382.6 g (SD = 499.2). Mean head circumference was 34.1 cm (SD = 1.6). Mean birth weight, birth length, and head circumference were lower and there was greater variability in these outcomes among African Americans than in Dominican infants. The differences between individual outcomes were not significant by *t*-test; however, by multivariate Hotelling's *t*-test, at least one of these outcomes (weight, length, head circumference) was significantly lower in African Americans ($p < 0.001$). Four percent of infants were preterm (< 37 weeks gestation). African-American infants had a significantly lower mean gestational age than Dominican infants (39 vs. 39.6 weeks, $p \leq 0.01$), despite the restriction of the sample to women who completed the air monitoring in the third trimester of pregnancy.

The test of interaction between PAHs and ethnicity was significant for birth weight ($p = 0.002$) and for head circumference ($p = 0.012$), such that PAHs had a significant adverse effect on birth weight and head circumference among African-American but not Dominican infants. Possible differential effects of toxicant exposure on different ethnic groups prompted us to conduct ethnic-specific regressions.

As shown in Table 2, among African Americans prenatal exposure to PAHs was associated with lower birth weight (β = –0.09, $p = 0.003$) and smaller head circumference (β = –0.02, $p = 0.01$) after adjusting for potential confounders (model 1). CPF was significantly associated with decreased birth weight overall (β = –0.04, $p = 0.01$) and among African Americans (β = –0.05, $p = 0.04$), but not among Dominicans (β = –0.03, $p = 0.11$). CPF was also associated with reduced birth length overall (β = –0.01, $p = 0.003$) and in Dominicans (β = –0.02, $p < 0.001$), but was not significantly associated with head circumference. After adjusting for

Table 1. Demographic and exposure characteristics of the population.[a]

	All ($n = 263$)	African American ($n = 116$)	Dominican ($n = 146$)
Maternal age (year)[b]	24.6 (5.2) ($n = 262$)	24.1 (5) ($n = 115$)	25 (5.3) ($n = 146$)
Maternal education (%)			
< High school	32.8	31	34.3
High school	45.3	46.6	44.3
> High school	21.9	22.4	24.4
Maternal ETS			
(% reporting smoker in the home)	42.8	51.8*	35.9
Maternal alcohol consumption			
(% drank alcohol during pregnancy)	24	12.4*	33.3
Maternal height (cm)[b]	162.6 (8.3) ($n = 263$)	164.5 (8.4) ($n = 116$)*	161 (8) ($n = 146$)
Maternal prepregnancy weight (kg)[b]	67 (16.5) ($n = 263$)	71.6 (19.7) ($n = 116$)*	63.3 (12.4) ($n = 146$)
Gestational age (weeks)[b]	39.3 (1.5) ($n = 263$)	39 (1.7) ($n = 116$)*	39.6 (1.2) ($n = 129$)
Newborn birth weight (g)	3382.6 (499.2) ($n = 262$)	3299 (548.7) ($n = 115$)[c]	3348.5 (449.4) ($n = 146$)
Newborn birth length (cm)[b]	50.9 (2.7) ($n = 259$)	50.8 (3.4) ($n = 115$)[c]	51.1 (2.2) ($n = 143$)
Newborn head circumference (cm)[b]	34.1 (1.6) ($n = 248$)	33.8 (1.9) ($n = 111$)[c]	34.3 (1.2) ($n = 136$)
Sex of newborn (% females)	51.5	48.3	54.1
Plasma cotinine (ng/mL)[b,d]	0.5 (2.4) ($n = 254$)	0.6 (2) ($n = 110$)*	0.5 (2.7) ($n = 143$)
Inhalation PAH (ng/m^3)[b]	3.7 (3.6) ($n = 263$)	3.5 (2.8) ($n = 116$)	3.9 (4.1) ($n = 146$)
Plasma chlorpyrifos (CPF) (pg/g)[b]	7.5 (7.5) ($n = 113$)	8 (6.3) ($n = 57$)	7.1 (8.5) ($n = 56$)

[a]Subjects with prenatal monitoring data on PAH, either cord or maternal blood sample, complete questionnaire data, and birth outcome data. There were no significant differences between the overall parent population and the present subset in terms of demographic, questionnaire-derived, and birth outcome variables shown in Table 1. [b]Mean (SD). Arithmetic means are presented for ease of comparison with other studies; however, the reported analyses are based on log-transformed data. [c]By Multivariate Hotelling's *t*-test, at least one of these outcomes (weight, length, head circumference) was significantly lower in African Americans ($p < 0.001$). [d]Subjects with cotinine > 25 ng/mL were excluded from analysis. Cotinine represents the level in cord blood or, if unavailable, the level in maternal blood, using the formula provided in the text. *$p \leq 0.01$ for African American vs. Dominican (Student's *t*-test for maternal height, prepregnancy weight, and gestational age; χ^2 for ETS exposure and alcohol; Mann-Whitney for cotinine).

Table 2. Associations between prenatal PAH exposure and birth outcomes,[a] by ethnic group, with and without CPF.

	Birth weight		Birth length		Head circumference	
	Model 1	Model 2[b]	Model 1	Model 2	Model 1	Model 2
All						
β	–0.02	–0.05	–0.003	–0.02	–0.006	–0.01
PAH *p*	0.27	0.08	0.57	0.08	0.24	0.12
No.	261	112	258	112	247	108
African American						
β	–0.09	–0.10	–0.01	–0.02	–0.02	–0.02
PAH *p*	0.003	0.02	0.30	0.24	0.01	0.06
No.	115	56	115	57	111	54
Dominican						
β	0.03	–0.009	0.003	–0.02	0.005	0.003
PAH *p*	0.16	0.81	0.65	0.11	0.33	0.80
No.	146	56	143	55	136	54

Model 1: covariates included BMI, parity, cotinine, sex of baby, and gestational age.
[a]Birth outcomes were log (LN) transformed. [b]Model 2 covariates included all variables in model 1 plus CPF.

CPF, relationships between PAHs and birth outcomes were essentially unchanged among African Americans (model 2): (β = –0.1, p = 0.02 for birth weight; and β = –0.02, p = 0.06 for head circumference). Table 3 summarizes the effects of all covariates in model 2. PAHs and CPF appear to be significant independent determinants of birth outcomes in that no significant interactions were observed between them; however, this analysis is limited by the number of subjects in the combined analyses. When we adjusted further in the analysis of personal air PAHs for other PAH sources (dietary PAHs and ETS as measured by cotinine), the associations between PAHs and birth outcomes remained significant (β = –0.08, p = 0.02 for birth weight; and β = –0.02, p = 0.04 for head circumference among African Americans).

Discussion

The association of PAHs with decreased birth weight and smaller head circumference among African Americans, before and after adjusting for CPF and other potential confounders, is of concern because several studies have reported that reduction in head circumference at birth or during the first year of life correlates with lower IQ as well as poorer cognitive functioning and school performance in childhood (Dam et al. 1998; Hack et al. 1991; Song et al. 1997). The present finding is consistent with our prior observation that the levels of PAH–DNA adducts in cord blood of Caucasian newborns in Poland were significantly associated with lower birth weight, reduced length, and head circumference (Perera et al. 1998). Fetal toxicity may be caused by antiestrogenic effects (Bui et al. 1986), binding to the human Ah receptor to induce P450 enzymes (Manchester et al. 1987), to DNA damage resulting in activation of apoptotic pathways (Meyn 1995; Nicol et al. 1995; Wood and Youle 1995), or to binding to receptors for placental growth factors resulting in decreased exchange of oxygen and nutrients (Dejmek et al. 2000).

In this study, among African Americans high PAHs were associated with a 9% reduction in birth weight and with a head circumference decrement of 2%. After adjusting for CPF, the corresponding reductions were 10% in birth weight and 2% in head circumference. No other studies have carried out personal monitoring of PAHs in pregnant women. However, we note that the mean PAH concentration observed in this study (3.7 ng/mL) is 46% lower than that measured in personal air samples from nonsmoking women who worked outdoors in a moderately polluted area of the Czech Republic (6.9 ± 4.4 ng/m^3, range 2.7–18.8 ng/m^3). However, the New York City range is wider and the upper bound is higher. PAHs are only one class of chemicals among many found in particulate matter from combustion sources. Nevertheless, our results are consistent with ecologic studies showing associations between ambient levels of air pollutants (including total suspended particulate matter) and low birth weight (Bobak 2000; Chen and Omaye 2001; Ha et al. 2001). A study in Northern Bohemia found that estimated exposure to carcinogenic PAHs in early gestation (based on ambient air monitoring data) was associated with reduced fetal growth (Dejmek et al. 2000).

The finding that effects of PAHs were significant only among African Americans may be attributed to unmeasured differences in exposure and/or susceptibility, to our limited sample size, or to the fact that birth outcomes (weight, head circumference, and gestational age) were generally less favorable and more variable in African Americans than in Dominicans. It is possible that with our limited sample size we were able to observe effects of prenatal exposure to pollutants only in African-American women because of the greater incidence of adverse outcomes and the greater variability in birth outcomes within this group compared with Dominicans. Although African-American mothers had significantly higher self-reported exposure to ETS and plasma cotinine levels, the personal air concentrations of PAHs and CPF did not differ significantly between ethnic groups (Table 1).

The data show an association between CPF and reduced birth weight and birth length. CPF is only one of a number of pesticides measured in the parent cohort study. Comprehensive analyses of the main effects of all measured pesticides on birth outcomes will be carried out. There are no prior comparable studies in humans, and the mechanism for possible effects on fetal growth is not known. However, exposure to CPF has been shown to induce brain cell loss and neurochemical and behavioral effects in the developing rat when given prenatally (Campbell et al. 1997; Chanda and Pope 1996). CPF has recently been regulated and residential uses are being phased out, but it has been the most heavily applied pesticide throughout New York State and in Manhattan in recent years (Landrigan et al. 1999; Thier et al. 1998; Whyatt et al. 2002).

In this study, cotinine was not a significant predictor of birth outcomes (p > 0.1). The absence of a significant effect of ETS (cotinine) may be due to the fact that the cohort is nonsmoking and cotinine levels are generally low.

This study has the advantage of being based on individual prenatal exposure data from personal monitoring and biomarkers, as well as extensive medical records and questionnaire data. However, it is limited by the modest sample of subjects for whom data from all relevant domains are currently available. Relationships observed in low-income, minority women might be different in women of other races or ethnic, cultural, or socioeconomic backgrounds. In our study cohort, adverse environmental conditions and adverse outcomes are common. However, we reported previously that DNA damage from PAHs was associated with worse birth outcomes among Caucasians in Central Europe who were exposed to air pollution from coal burning (Perera et al. 1998). Finally, due to budgetary constraints, we were able to obtain only a single prenatal air sample over a 48-hr period. Whether this single measurement is representative of average exposure either during

Table 3. Associations between all covariates in Model 2[a] and birth outcomes[b], by ethnic group.

	All			African American			Dominican		
	BW	BL	HC	BW	BL	HC	BW	BL	HC
PAH									
β	–0.05	–0.02	–0.01	–0.10	–0.02	–0.02	–0.009	–0.02	0.003
p-Value	0.08	0.08	0.12	0.02	0.24	0.06	0.81	0.11	0.80
CPF									
β	–0.04	–0.02	–0.005	–0.05	–0.01	–0.003	–0.02	–0.02	–0.005
p-Value	0.01	0.04	0.28	0.04	0.15	0.70	0.26	0.002	0.31
Parity									
β	–0.02	0.01	0.002	0.02	0.03	0.006	–0.06	0.001	–0.005
p-Value	0.66	0.25	0.89	0.74	0.21	0.75	0.23	0.93	0.70
BMI									
β	0.003	0.0003	0.0007	0.006	0.0007	0.001	0.003	0.0003	–0.0005
p-Value	0.14	0.69	0.26	0.06	0.56	0.17	0.47	0.76	0.65
Sex									
β	0.04	0.02	0.02	0.06	0.03	0.02	0.04	0.02	0.02
p-Value	0.14	0.02	0.02	0.18	0.13	0.10	0.27	0.04	0.07
Gestational age									
β	1.94	0.63	0.64	0.64	0.68	0.70	1.02	0.06	0.19
p-Value	< 0.001	< 0.001	< 0.001	0.003	< 0.001	< 0.001	0.20	0.78	0.36
No.	112	112	108	56	57	54	56	55	54

Abbreviations: BL, birth length; BW, birth weight; HC, head circumference.
[a]Model 2 covariates included all variables in model 1 plus CPF. [b]Birth outcomes were log (LN) transformed.

the third trimester or the entire pregnancy is unknown. Similarly, the biomarkers were measured at a single time point. In cases of chronic exposure, a single measure can be a representative internal dosimeter; however, this may not be true if exposures are sporadic and biomarkers have short half-lives. Studies are underway to determine the representativeness of a single 48-hr personal monitoring sample and a single biomarker measurement for various pollutants of interest.

Conclusion

This study provides evidence that environmental pollutants at levels currently encountered in New York City adversely affect fetal development.

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