

# Particulate Pollutants and Racial/Ethnic Disparity in Feto-Infant Morbidity Outcomes

Hamisu M. Salihu · Nafisa Ghaji · Alfred K. Mbah ·  
Amina P. Alio · Euna M. August · Ibrahimou Boubakari

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**Abstract** We sought to assess the association between air particulate pollutants and feto-infant morbidity outcomes across racial/ethnic subgroups. This is a retrospective cohort study from 2000 through 2007 based on three linked databases: (1) The Florida Hospital Discharge database; (2) The vital statistics records of singleton live births in Florida; (3) Air pollution and meteorological data from the Environmental Protection Agency. Using computerized mathematical modeling, we assigned exposure values of the air pollutants of interest ( $PM_{2.5}$ ,  $PM_{10}$  and the PM coarse fraction [ $PM_{10} - PM_{2.5}$ ]) to mothers over the period of pregnancy based on Euclidean minimum distance from the air

pollution monitoring sites. The primary outcomes of interest were: low birth weight, very low birth weight, preterm birth, very preterm birth, and small for gestational age (SGA). We used adjusted odds ratios to approximate relative risks. We observed increased risk for overall feto-infant morbidity outcome in women exposed to any of the three particulate pollutants (values above the median). Exposed women had increased odds for low birth weight, very low birth weight and preterm birth with the greatest risk being that for very low birth weight (AOR = 1.27, 95% CI = 1.08–1.49). Black women exposed to any particulate pollutant had the greatest odds for all the morbidity outcomes, most pronounced for very low birth weight (AOR = 3.32, 95% CI = 2.56–4.30). Environmental particulate pollutants are associated with adverse feto-infant outcomes among exposed women, especially blacks. Black–white disparity in adverse fetal outcomes is widened in the presence of these pollutants, which provide a target for intervention.

H. M. Salihu  
Department of Obstetrics and Gynecology, College of Medicine,  
University of South Florida, 2 Tampa General Circle, Tampa,  
FL 33606, USA

H. M. Salihu (✉) · A. K. Mbah  
Department of Epidemiology and Biostatistics, College of Public  
Health, University of South Florida, 13201 Bruce B. Downs  
Blvd., MDC 56, Tampa, FL 33612, USA  
e-mail: hsalihu@health.usf.edu

N. Ghaji  
Department of Occupational and Environmental Health, Emory  
University, Clairmont Rd #16B, Decatur, GA 30030, USA

A. P. Alio  
Department of Community and Preventive Medicine, University  
of Rochester, P.O. Box 278969, Rochester, NY 14627, USA

E. M. August  
Department of Community and Family Health, College of Public  
Health, University of South Florida, 13201 Bruce B. Downs  
Blvd., MDC 56, Tampa, FL 33612, USA

I. Boubakari  
Department of Public Health, Western Kentucky University,  
1906 College Heights Blvd., Bowling Green, KY 42101, USA

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## Introduction

In recent years, there has been a growing interest in research focusing on the effects or potential impact of prenatal exposure to air particulate pollutants primarily due to the mounting evidence of the vulnerability of subgroups such as children to insults from such exposures. Studies have examined the association between exposure to air pollution and a number of birth outcomes that include birth weight [1, 2], birth defects [3, 4], and infant mortality and morbidity [5, 6]. These studies have produced mixed results.

Environmental contaminants include air pollutants such as particulate matter (PM), nitrogen dioxide (NO<sub>2</sub>), sulfur dioxide (SO<sub>2</sub>), carbon monoxide (CO), and the secondary pollutant ozone (O<sub>3</sub>). The main source of these pollutants is traffic and industrial emissions and their measurements are routine using fixed monitoring stations in all states around the United States. Exposure assessment is a major challenge in most pollution studies. Ideally, exposure per pollutant per person would be desirable but this is practically difficult if not impossible at the population-level. Researchers have to rely on estimates using distances from residence to the monitors on a regional scale [7].

Particulate matter, also known as particle pollution or PM, is a complex mixture of extremely small particles and liquid droplets. The two types of particulate matter that are of health concern are: (1) PM<sub>10</sub>, which are smaller than 10 μm in diameter and (2) PM<sub>2.5</sub>, which are fine particles 2.5 μm in diameter and smaller. The health effects occur because PM<sub>10</sub>'s are readily inhalable and because of their size, they are not filtered and penetrate deeply into the body and cause cardiovascular and respiratory disorders. PM<sub>2.5</sub> penetrate deeper into the lungs and even though it is not clear how exactly they restrict fetal growth, some studies have shown that they alter cell activity or reduce the amount of oxygen and nutrients the fetus receives in utero [8].

Some studies have shown a period-specific association between air pollutants and adverse birth outcomes. In Sao Paolo, researchers found impaired growth in the first trimester as a result of high exposure to CO [1], while in Beijing, it was reported that areas with higher exposure levels to sulfur dioxide and total suspended particulates (TSPs) during the third trimester caused a greater risk for delivering a low birth weight infant [9]. However, it remains poorly understood whether differential effects of these pollutants exist across racial/ethnic subgroups thereby delineating sub-populations that are at increased risk. Accordingly, we undertook this study to assess the impact of exposure to PM<sub>10</sub>, PM<sub>2.5</sub> and the PM coarse fraction (PM<sub>10</sub> – PM<sub>2.5</sub>) on fetal morbidity outcomes with the unique feature of delineating differential effects on racial/ethnic subgroups. We conducted the study with the following hypotheses:

1. PM<sub>2.5</sub>, PM<sub>10</sub> and the PM coarse fraction (PM<sub>10</sub> – PM<sub>2.5</sub>) impair fetal growth and development processes in utero leading to feto-infant morbidity outcomes (low birth weight, very low birth weight, small for gestational age (SGA), preterm and very preterm).
2. The impact of particulate air pollutants on feto-infant morbidity outcomes varies by race/ethnicity.

## Methods

The study population includes all singleton live births in Hillsborough County, Florida covering the period from 2000 to 2007. The linked de-identified Hospital Inpatient Discharge (HID) and vital statistic birth data file for Hillsborough County was utilized for this study. The HID data were obtained from the Florida Agency for Health Care Administration (AHCA), and the vital records were acquired from the Florida Department of Health (FDOH). Additionally, air pollution and meteorological data from the US Environmental Protection Agency (Air Quality System, [www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm](http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm)), which included 6-day concentrations of PM<sub>2.5</sub> and PM<sub>10</sub>, were used for analysis. The air pollutants were monitored at 14 stationary monitoring stations located at different sites in Hillsborough County. From the available data, the 24-h daily maximum concentration was computed. The pollution data also encompassed latitudes and longitudes, which were converted to zip codes using relevant information from the Hillsborough Environmental Pollution Commission (EPC).

For maternal exposure assessment, the following steps were taken to assign air pollution exposure values to each mother. We computed the Euclidean distances between the centroid of the mother's residential zip code during pregnancy and the centroid of each zip code from the 14 monitoring stations. Since there were 14 monitoring sites and 97 residential zip codes in Hillsborough County, we had a matrix with entries of 97 × 14 distances (each residential zip code had 14 computed distances). Each residential zip code was assigned to one of the 14 monitoring sites based on closest proximity. If there were missing data from the closest monitoring site for a particular day of gestation, then the reading was taken from the next closest site without missing data. If the daily readings were missing across all sites, then the daily exposure estimate was left as missing [3]. No imputation or interpolation of missing data was carried out in this study.

Maternal exposure to air pollution during pregnancy was estimated using date of birth of the infant and the length of each gestational age. This resulted in weekly PM<sub>2.5</sub> or PM<sub>10</sub> exposure data for each pregnancy. Our interest was in maternal exposure occurring throughout pregnancy since studies have shown that air pollution exposure may interfere with placental development and subsequent oxygen and nutrient delivery to the fetus throughout pregnancy [10]. Mean exposures to each air pollutant for the length of each pregnancy were then computed and assigned to each birth [1]. We additionally considered the coarse fraction of PM, defined as PM<sub>10</sub> – PM<sub>2.5</sub> and ranged from 2.5 to 10 μm in diameter. The PM coarse fraction has been shown to be a risk factor for respiratory diseases, such as asthma [11]. In this study, the exposed group was comprised of

mothers exposed to one or more of the three particulates (PM<sub>2.5</sub>, PM<sub>10</sub>, or PM coarse fraction) during pregnancy), whereas the unexposed group was comprised of mothers not exposed to any of the particulates during pregnancy. A mother was classified as being exposed to a specific particulate if the value of the particulate pollutant fell above the median, and we defined a mother as unexposed if the values fell at or below the median. We also performed subgroup analyses to estimate interaction by categorizing mothers according to whether or not they were exposed to two types of particulate matter (two-way interaction) or whether or not they were exposed to all three of the particulates (three way interaction). Groups were constructed as follows: (1) two-way interaction = ([exposure to PM<sub>2.5</sub> and PM<sub>10</sub>] or [PM<sub>10</sub> and PM coarse fraction] or [PM<sub>2.5</sub> and PM coarse fraction]), and (2) three-way interaction = exposure to all three particulate matters (exposure to PM<sub>2.5</sub>, and PM<sub>10</sub>, and PM coarse fraction).

The main outcomes of interest were low birth weight (<2,500 g), very low birth weight (<1,500 g), preterm birth (<37 weeks), very preterm birth (<33 weeks), and SGA. We defined SGA as less than the 10th percentile of birth weight for a given gestational age using population-based national reference curves [12]. Sex and race-specific reference curves were used in this study. Gestational age was computed in weeks as the interval between the first day of the last menstrual period and the date of delivery of the fetus. When the menstrual estimate of gestational age was inconsistent with the birth weight (e.g., very low birth weight at term), a clinical estimate of gestational age on the vital records was used instead [13].

All demographic information was abstracted from birth certificate data. The variables included racial/ethnic status (white non-Hispanic, black non-Hispanic, Hispanic, or other); maternal age (<35 or ≥35 years old at the time of delivery); marital status (single [which included women whose marital status was classified as unknown, widowed or divorced] or married); education (<12 or ≥12 years); parity (nulliparous or multiparous); cigarette smoking during pregnancy (yes or no), and adequacy of prenatal care (adequate or inadequate). Adequacy of prenatal care was assessed using the revised graduated index algorithm, which has been found to be more accurate than several others, especially in describing the level of prenatal care utilization among groups that are high risk [14]. This index assesses the adequacy of care based on the trimester prenatal care began, number of visits, and the gestational age of the infant at birth.

Maternal pregnancy complications obtained from the hospital discharge data, based on ICD-9 principal and other diagnostic codes, included anemia (280–286), gestational diabetes (648.8), diabetes mellitus (250, 648.0), gestational hypertension (642.3) and chronic hypertension

(642.0, 401.0, 401.1, 401.9, 642.1, 642.2, 742.7), pre-eclampsia (642.4, 642.5, 642.7, 642.9), eclampsia (642.6), placental abruption (641.2), and placental previa (641.0, 641.1). Researchers have observed improved accuracy with discharge data compared to the birth certificate data in terms of maternal pregnancy complications [15, 16].

Baseline characteristics between women who were exposed and those who were not exposed were compared using Chi-square test for categorical variables and t-tests for continuous variables. The risk for fetoinfant morbidity outcomes (low birth weight, very low birth weight, preterm birth, very preterm birth, and SGA) among the exposed group were compared to that of the unexposed group using odds ratios and 95% confidence intervals after adjusting for baseline characteristics in multiple logistic models. We constructed the regression models and assessed goodness-of-fit of the regression models using the  $-2 \log$  likelihood ratio test. We estimated the significance of main effects by means of the Wald test. For subgroup analysis, we fitted separate regression models for two-way interaction (whether or not women were exposed to any two of the three particulates) and three-way interaction (whether or not they were exposed to all three particulate matter). The LOGISTIC procedure in SAS (SAS Institute, Inc., Cary, North Carolina, version 9.2) was used to conduct the analysis. All tests of hypothesis were two-tailed with a type 1 error rate fixed at 5%. This study was approved by the Institutional Review Board at the University of South Florida.

## Results

The median values (median ± SD) for the three particulate pollutants in this population were 11.28 μg/m<sup>3</sup> (±10.98) for PM<sub>2.5</sub>, 24.35 μg/m<sup>3</sup> (±25.0) for PM<sub>10</sub>, and 13.1 μg/m<sup>3</sup> (±13.3) for the PM coarse fraction (data not shown). Of the overall sample of 103,961 singleton births within the study population, 24,090 (23.2%) births were among women categorized as exposed while the unexposed group consisted of 79,871 (76.8%) births (Table 1).

The crude proportions of maternal demographic characteristics by exposure status to particulate matter are presented in Table 1. Women who were exposed to any of the particulates were less likely to smoke and were relatively less educated. Furthermore, they were also more likely to be of a racial/ethnic minority group (non-white) and to be unmarried. Among the common obstetric complications analyzed, only anemia and diabetes mellitus showed a significant difference between the two groups, with exposed women experiencing elevated proportions of anemia but lower proportions of diabetes mellitus.

**Table 1** Proportions (%) of selected maternal demographic characteristics and pregnancy complications between two groups of women exposed to PM<sub>2.5</sub> and PM<sub>10</sub> in Hillsborough County, Florida (2000–2007)

Characteristics N = 103,961	Unexposed group (%) <sup>a</sup> N = 79,871	Exposed group (%) <sup>a</sup> N = 24,090	P value <sup>b</sup>
Maternal age (years)			0.62
<35	86.44	86.31	
≥35	13.56	13.69	
Race/ethnicity			<0.01
White	54.35	50.77	
Black	19.86	20.47	
Hispanic	16.75	16.83	
Other	9.03	11.93	
Maternal education (years)			<0.01
<12	18.84	19.32	
≥12	80.31	80.02	
Missing/unknown	0.85	0.66	
Marital status			<0.01
Married	60.39	59.09	
Unmarried	39.60	40.89	
Missing/unknown	0.01	0.01	
Smoking during pregnancy			0.02
Yes	6.76	6.54	
No	92.08	92.08	
Missing/unknown	1.16	1.37	
Prenatal care			0.16
Adequate	66.19	66.67	
Inadequate	33.81	33.33	
Parity			0.45
0 (nulliparous)	66.10	66.37	
≥1 (multiparous)	33.90	33.63	
Pregnancy complications			
Anemia	8.51	10.41	<0.01
Gestational diabetes	5.67	5.73	0.72
Diabetes mellitus	0.81	0.68	0.03
Gestational hypertension	4.81	4.82	0.93
Chronic hypertension	1.4	1.45	0.58
Preeclampsia	3.9	3.64	0.06
Eclampsia	0.05	0.08	0.13
Abruption	0.8	0.86	0.34
Placenta previa	0.74	0.76	0.76
Renal disease	0.07	0.08	0.79

<sup>a</sup> The exposed group is comprised of mothers exposed to one or more of the observed particulates (PM<sub>2.5</sub>, PM<sub>10</sub>, or PM coarse fraction) during pregnancy, and the unexposed group is comprised of mothers not exposed to any of the particulates during pregnancy

<sup>b</sup> Significant values in bold font. P values <0.05 considered significant

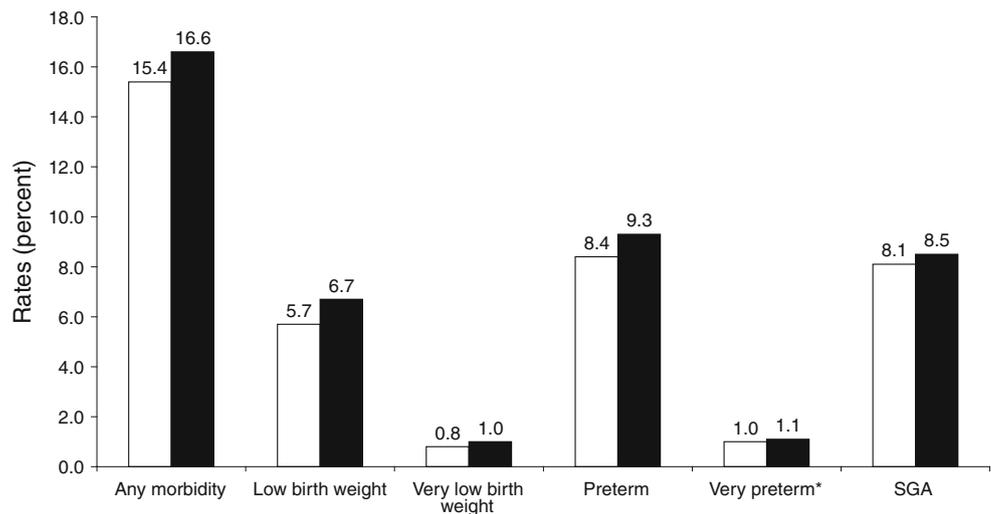
The mean gestational age of infants born to women exposed to particulate matter was lower compared to those of the unexposed group (mean ± SD = 38.4 ± 1.9 weeks vs. 38.5 ± 1.8 weeks, respectively;  $P < 0.01$ ; data not shown). Additionally, the infants of exposed women were, on average, 18 grams smaller compared to infants of unexposed women (mean ± SD = 3,294 ± 557 g vs. 3,312 ± 541 g, respectively;  $P < 0.01$ ; data not shown).

The crude frequencies of fetoinfant morbidity outcomes within the study population are presented in Fig. 1. Overall, 16,961 (16.3%) of infants had any morbidity outcome,

with 6,679 (6.4%) having low birth weight, 999 (1.0%) having very low birth weight, 9,459 being preterm (9.1%), 1,133 (1.1%) being very preterm, and 8,733 (8.4%) being SGA. When considering exposure status, all of the observed fetoinfant morbidity outcomes were more prevalent among women who were exposed to particulate pollutants.

The adjusted odds ratios for the association between women by exposure status to particulate matter are summarized in Table 2. We observed about 8% increased odds for any fetoinfant morbidity outcome in women exposed

**Fig. 1** Crude proportions of feto-infant morbidity outcomes comparing women who were exposed to any particulate matter and those who were not exposed to any particulate matter. \**P* value = 0.04; *P* value for all other comparison <0.01. *Open bar* women who were not exposed to any particulate matter. *Filled bar* women who were exposed to any particulate matter



**Table 2** Adjusted odds ratios (AOR) and 95% confidence intervals (CI) for the association between exposure to particulate pollutants and feto-infant morbidity outcomes

<i>N</i> = 103,961	Any particulate exposure AOR (95% CI)	PM <sub>2.5</sub> exposure only AOR (95% CI)	PM <sub>10</sub> exposure only AOR (95% CI)	PM coarse fraction exposure only AOR (95% CI)
Low birth weight ( <i>n</i> = 6,679)	<b>1.18 (1.11–1.25)</b>	<b>1.07 (1.01–1.12)</b>	<b>1.13 (1.07–1.19)</b>	<b>1.09 (1.03–1.15)</b>
Very low birth weight ( <i>n</i> = 999)	<b>1.27 (1.08–1.49)</b>	<b>1.14 (1.01–1.29)</b>	<b>1.17 (1.02–1.34)</b>	<b>1.22 (1.07–1.39)</b>
Preterm birth ( <i>n</i> = 9,459)	<b>1.12 (1.06–1.18)</b>	1.03 (0.98–1.07)	<b>1.11 (1.06–1.16)</b>	<b>1.05 (1.01–1.09)</b>
Very preterm birth ( <i>n</i> = 1,133)	1.14 (0.98–1.31)	1.05 (0.93–1.18)	1.10 (0.96–1.25)	<b>1.13 (1.01–1.27)</b>
Small for gestational age ( <i>n</i> = 8,733)	1.04 (0.99–1.10)	<b>1.06 (1.01–1.11)</b>	1.02 (0.97–1.07)	<b>1.07 (1.02–1.12)</b>
Any morbidity ( <i>n</i> = 16,961)	<b>1.08 (1.04–1.13)</b>	<b>1.05 (1.01–1.08)</b>	<b>1.07 (1.03–1.11)</b>	<b>1.06 (1.02–1.10)</b>

<sup>a</sup> Estimates were generated after controlling for year of birth, education, maternal age, tobacco use, parity, marital status, adequacy of prenatal care, sex of the infant, anemia, gestational diabetes, diabetes mellitus, gestational hypertension, chronic hypertension, preeclampsia, eclampsia, placenta abruption, placenta previa, and renal diseases. Significant values in bold font

to any particulate (either PM coarse fraction, PM<sub>2.5</sub> or PM<sub>10</sub>) considered in this study (AOR = 1.08, 95% CI = 1.04–1.13). The exposed group of women also had increased odds for low birth weight, very low birth weight and preterm birth. The risk for very preterm birth and small size for gestational age did not reach statistical significance among women exposed to any particulate pollutant. A similar finding was observed in women exposed to PM<sub>10</sub>. Women exposed to PM<sub>2.5</sub> had a higher likelihood of low birth weight, very low birth weight, and SGA. Women exposed to the PM coarse fraction had increased odds for all the observed feto-infant morbidity outcomes. The likelihood of low birth weight and very low birth weight was heightened across all exposure categories.

Furthermore, regardless of exposure level, the most elevated odds were observed for very low birth weight, ranging from a low of 14% with exposure to PM<sub>2.5</sub> (AOR = 1.14, 95% CI = 1.01–1.29) to a high of 27% with exposure to any particulate matter (AOR = 1.27, 95% CI = 1.08–1.49).

Sub-group analysis was performed to investigate the interaction across the three particulate pollutants (Table 3). The interaction of all three particulates appeared to be the most severe, with increased odds for low birth weight, very low birth weight, preterm birth, and small size for gestational age. The greatest risk was observed for very low birth with a 23% increased odds (AOR = 1.23, 95% CI = 1.06–1.41). Furthermore, the interaction between

**Table 3** Adjusted odds ratios (AOR) and 95% confidence intervals (CI) for fetoinfant morbidity outcomes in the presence of particulate pollutant interactions

<i>N</i> = 103,961	Unexposed	PM <sub>10</sub> and PM <sub>2.5</sub> AOR (95% CI) <sup>b</sup>	PM <sub>10</sub> and PM coarse fraction AOR (95% CI) <sup>b</sup>	PM <sub>2.5</sub> and PM coarse fraction AOR (95% CI) <sup>b</sup>	Three-way interaction AOR (95% CI) <sup>b</sup>
Low birth weight ( <i>n</i> = 6,679)	1.00	0.95 (0.81–1.11)	<b>1.13 (1.04–1.21)</b>	0.98 (0.68–1.43)	<b>1.10 (1.04–1.16)</b>
Very low birth weight ( <i>n</i> = 999)	1.00	0.86 (0.57–1.31)	1.17 (0.96–1.42)	1.66 (0.78–3.52)	<b>1.23 (1.06–1.41)</b>
Preterm birth ( <i>n</i> = 9,459)	1.00	0.88 (0.77–1.00)	<b>1.10 (1.03–1.18)</b>	0.71 (0.50–1.01)	<b>1.08 (1.03–1.13)</b>
Very preterm birth ( <i>n</i> = 1,133)	1.00	0.91 (0.62–1.34)	1.06 (0.89–1.27)	1.62 (0.80–3.29)	1.13 (0.99–1.29)
Small for gestational age ( <i>n</i> = 8,733)	1.00	1.01 (0.88–1.15)	1.06 (0.99–1.13)	1.28 (0.95–1.73)	<b>1.06 (1.01–1.12)</b>
Any morbidity ( <i>n</i> = 16,961)	1.00	0.94 (0.85–1.04)	<b>1.09 (1.03–1.14)</b>	1.00 (0.78–1.27)	<b>1.08 (1.04–1.12)</b>

Estimates were generated after controlling for year of birth, education, maternal age, tobacco use, parity, marital status, adequacy of prenatal care, sex of the infant, anemia, gestational diabetes, diabetes mellitus, gestational hypertension, chronic hypertension, preeclampsia, eclampsia, placenta abruption, placenta previa, and renal diseases. Significant values in bold font

PM<sub>10</sub> and the PM coarse fraction had increased odds for low birth weight, very low birth weight, and preterm birth. All the other pairwise interactions did not show elevated risks for fetoinfant morbidities.

In order to evaluate the differences in the effects of particulate matter on fetoinfant morbidities by race and ethnicity, we further categorized infants by race and exposure status (Table 4). Eight subgroups were thus constructed: whites, unexposed (referent group, 12.6%); whites exposed (39.0%); blacks, unexposed (4.6%); blacks, exposed (15.7%); Hispanics, unexposed (3.9%); Hispanics, exposed (12.9%); others, unexposed (2.1%) and others, exposed (9.2%). Subgroup analysis of the association between particulate matter and fetoinfant morbidity outcomes (low birth weight, very low birth weight, preterm birth, very preterm birth, and SGA) showed that black women who were exposed to any particulate matter had the greatest odds for all morbidity outcomes considered in this study. The most pronounced odds among black women exposed to any particulate were observed for very low birth weight, with a more than threefold increased likelihood (AOR = 3.32, 95% CI = 2.56–4.30). This was closely followed by very preterm birth (AOR = 2.99, 95% CI = 2.34–3.81). Black mothers who were not exposed also had elevated odds for all fetoinfant morbidity outcomes compared to unexposed white women. Hispanic women exposed to any particulate matter also had heightened risk estimates for fetoinfant morbidities with the greatest risk observed for very low birth weight (AOR = 1.44, 95% CI = 1.07–1.94).

Crude rates of fetoinfant morbidity by race/ethnicity and exposure status to particulate matter are presented in Fig. 2. For all of the observed morbidities, black women had the highest frequencies, regardless of exposure status. The most elevated rates among black women were observed for small size for gestational age (exposed, 13.9% and unexposed, 12.7%), followed by preterm birth (exposed, 12.7% and unexposed, 10.6%).

## Discussion

This study was initiated with two a priori hypotheses regarding the association between particulate pollutants and fetoinfant morbidity outcomes. The first hypothesis posits that PM<sub>2.5</sub>, PM<sub>10</sub> and the PM coarse fraction (PM<sub>10</sub> – PM<sub>2.5</sub>) impair fetal growth and development processes in utero, leading to fetoinfant morbidity outcomes (low birth weight, very low birth weight, preterm birth, very preterm birth, SGA). Our findings support this hypothesis for all the three particulates considered. When women were categorized into an exposed and unexposed group in which the exposed status was based on being positive on any of the three particulates, we found exposed mothers to have elevated risks for low birth weight, very low birth weight, and preterm birth, with the greatest risk observed for low birth weight. The results also demonstrated greater likelihood for adverse fetal outcomes due to exposure to specific particulates. Our results confirm many earlier reports of poor birth outcomes with exposure to air

**Table 4** Adjusted odds ratio for the association between any exposure to particulate matter and feto-infant morbidity outcomes categorized by race/ethnicity

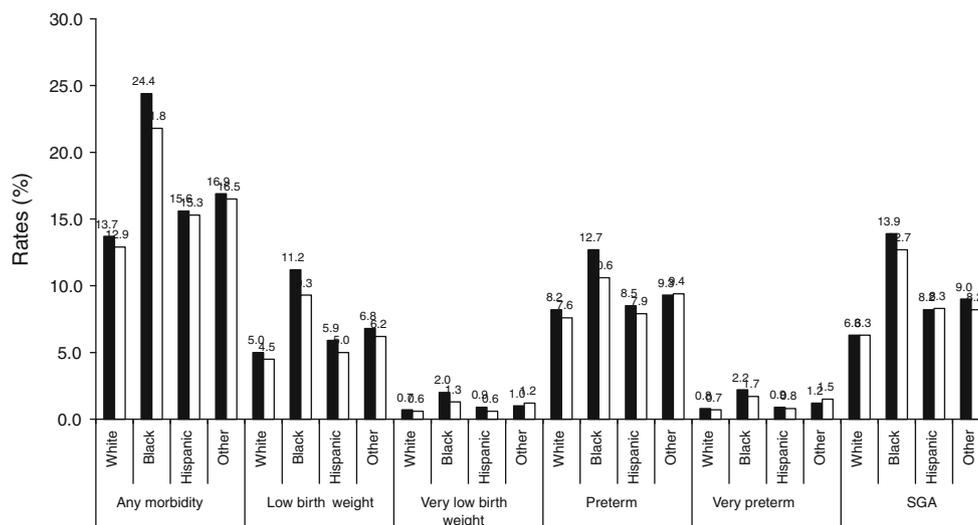
	White		Black		Hispanic		Other	
	Not exposed n = 13,094	Exposed n = 40,551	Not exposed n = 4,785	Exposed n = 16,348	Not exposed n = 4,036	Exposed n = 13,443	Not exposed n = 2,175	Exposed n = 9,529
Low birth weight n = 6,679	1.00	1.14 (1.04–1.26)	2.07 (1.82–2.36)	2.58 (2.33–2.85)	1.12 (0.95–1.32)	1.36 (1.21–1.52)	1.48 (1.22–1.80)	1.60 (1.42–1.79)
Very low birth weight n = 999	1.00	1.15 (0.89–1.48)	2.17 (1.54–3.05)	3.32 (2.56–4.30)	1.00 (0.63–1.60)	1.44 (1.07–1.94)	2.04 (1.29–3.21)	1.73 (1.27–2.35)
Preterm n = 9,459	1.00	1.11 (1.03–1.19)	1.42 (1.27–1.60)	1.76 (1.62–1.91)	1.05 (0.92–1.20)	1.15 (1.05–1.26)	1.32 (1.12–1.54)	1.26 (1.15–1.39)
Very preterm n = 1,133	1.00	1.14 (0.90–1.45)	2.34 (1.71–3.19)	2.99 (2.34–3.81)	1.19 (0.79–1.78)	1.36 (1.03–1.79)	2.25 (1.49–3.39)	1.74 (1.31–2.31)
SGA n = 8,733	1.00	1.01 (0.93–1.10)	2.06 (1.83–2.30)	2.26 (2.07–2.47)	1.34 (1.17–1.53)	1.33 (1.21–1.47)	1.42 (1.20–1.69)	1.53 (1.38–1.70)
Any morbidity n = 16,961	1.00	1.08 (1.02–1.14)	1.79 (1.64–1.96)	2.08 (1.95–2.22)	1.21 (1.09–1.34)	1.25 (1.16–1.34)	1.40 (1.24–1.59)	1.41 (1.31–1.52)

Adjusted odds ratios were generated after controlling for year of birth, education, maternal age, tobacco use, parity, marital status adequacy of prenatal care, sex of the infant, anemia, gestational diabetes, diabetes mellitus, gestational hypertension, chronic hypertension, pre-eclampsia, eclampsia, placenta abruption, placenta previa, and renal diseases

pollution [1–6, 10]. Similar to our study, Jedrychowski et al. [17] found that infants of pregnant women exposed to fine particles (PM<sub>2.5</sub>) had significantly lower birth weights compared to those of women who were not exposed.

Our second hypothesis postulates that the impact of particulate air pollutants on feto-infant morbidity outcomes varies by race/ethnicity. Our results showed that black women who were exposed to any particulate matter had the greatest odds for all the morbidity outcomes considered in this study. Of these morbidities, the risk for very low birth weight was most pronounced, with a more than threefold increased likelihood among exposed black women (AOR = 3.32, 95% CI = 2.56–4.30). When compared to the risk estimates for black–white disparity in unexposed women, we noted that exposure to air particulate pollutants widened the black–white disparity in feto-infant morbidity outcomes considerably. These findings are of immense importance since attempts to narrow the black–white gap in feto-infant morbidity and mortality have, so far, not been successful, frustrating the attainment of the expired Healthy People 2010 objective. One of the factors for this lack of progress in reducing or eliminating black–white disparity is our poor understanding of modifiable risk factors. The results of this study are in consonance with those of a previous study [18] and are suggestive that addressing environmental factors that elevate pollution exposures, especially in black neighborhoods, merit consideration and infusion of resources in current efforts to narrow disparities in infant health and survival. Unfortunately, we have limited data on socioeconomic and sociocultural factors within the study population that affect environmental factors. Hence, we cannot ascertain the interplay of these factors in the relationship across race/ethnicity, environmental pollutants, and feto-infant morbidities.

Certain limitations of this study are worth mentioning. Exposure analysis posed a big challenge. The exposed group was created based on a population-level estimation of the values of the particulate pollutants, rather than the actual amount/concentration of the particulates detected in the individual’s biologic sample (e.g., maternal blood). There is, therefore, a possibility of exposure misclassification because county-wide monitors were used to capture exposures at the individual level. This limitation is, however, not peculiar to our study, as other investigators [2, 3, 5, 10, 19–21] have also had to manage this measurement error. It is also noteworthy that dichotomizing exposure status based on the median ignores minimal exposure effects arising from values below the median, an approach that could have underestimated the magnitude of the association being reported in this paper. We were also unable to control for individual exposure variability induced by other factors including distance from roads, traffic, place of work, as well as period of time spent in the current address. For example, the observed racial/ethnic



**Fig. 2** Crude proportions of fetal-infant morbidity outcome comparing women who were exposed to any particulate matter and those unexposed to any particulate matter categorized by race/ethnicity.

Open bar women who were not exposed to any particulate matter. Filled bar women who were exposed to any particulate matter

differences in exposure to particulate matter may be due to variances in residential distance from sources of pollutants that could not be accounted for within the study design. Studies have also shown that 12–33% of women move from their initial address during pregnancy [3, 22–25], a potential source of non-differential misclassification [22], which could have led to an underestimate of the association between the three pollutants and fetal-infant morbidity outcomes examined in this study. Additional unmeasured confounding by other ambient pollutants is plausible and could have biased our results. The study could not identify the exact pollution source, information that will be pertinent for further analyses and intervention strategies. However, such extended exposure and risk assessment go beyond the scope and objectives of the study.

Despite the aforementioned shortcomings, certain strengths of the study merit mention, as well. One of the strengths is the availability of ample sample size. We had data for births from 2000 to 2007 and air pollution records for the same period. We were also able to examine various fetal-infant morbidity outcomes at birth, unlike other studies that focused only on low birth weight. We controlled for several potential confounders, although we cannot rule out residual confounding due to unmeasured variables, as well as the determination of exposure by residential zip code.

In summary, we found an association between exposure to abnormal levels of air particulate pollutants ( $PM_{2.5}$ ,  $PM_{10}$ ,  $PM_{10} - PM_{2.5}$ ) and specific fetal-infant morbidity outcomes. Analysis by race/ethnicity revealed that the black–white disparity in these outcomes was widened in the presence of these exposures.

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