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## Maternal Exposure to Fine Particulate Pollution During Narrow Gestational Periods and Newborn Health in Harris County, Texas

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### Abstract

It remains unclear when the fetus is most susceptible to the effects of particulate air pollution. We conducted a population-based study in a large urban area to evaluate associations between preterm birth (PTB) and fetal growth and exposures to fine particles (PM<sub>2.5</sub>) during narrow periods of gestation. We identified 177,816 births during 2005–2007 among mothers who resided in Harris County, Texas at the time of delivery. We created three mutually exclusive categories of mildly (33–36 completed weeks of gestation), moderately (29–32 weeks of gestation), and severely (20–28 weeks of gestation) PTB, and among full term infants, we identified those who were born small for their gestational age. Using routine air monitoring data, we generated county-level daily time series of estimated ambient air levels of PM<sub>2.5</sub> and then computed exposure metrics during every 4 weeks of a mother's pregnancy. We evaluated associations in each 4-week period using multiple logistic regression. A 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> exposure in the first 4 weeks of pregnancy significantly increased the odds of mildly, moderately and severely PTB by 16, 71 and 73 %, respectively. Associations were stronger when infants with birth defects were excluded. Our findings indicate an association between PM<sub>2.5</sub> and PTB, with stronger associations for

moderately and severely PTB infants. Efforts should continue to implement stricter air quality standards and improve ambient air quality.

## Keywords

Air pollution; Particulate matter; PM<sub>2.5</sub>; Preterm birth; Small for gestational age

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## Introduction

The air quality in the Houston area (Harris County, TX) is influenced by multiple sources of air pollutants due to extensive road traffic, proximity to one of the largest petrochemical industrial complexes in the United States (US) and port activities in the Houston Ship Channel. A risk assessment identified fine particles (PM<sub>2.5</sub>) posing “definite” health risks for Houstonians [1] and ambient air levels of PM<sub>2.5</sub> suggest that the Houston area may be near or exceed the new national ambient air quality standard for this pollutant (<http://www.epa.gov/pm/actions.html#jun12>). Although Harris County is the fifth largest metropolis in the US with documented problems of poor air quality [1] and known health, social and economic disparities [2], relatively few studies have examined health risks in relation to air pollution in the area [3–5] and only one focused on adverse birth outcomes [6].

The results of previous air pollution studies for preterm birth (PTB) and fetal growth restriction have been equivocal depending upon which gestational periods and air pollutants were studied [7–10]. Moreover, particulate matter varies in size (course, PM<sub>10</sub>; fine, PM<sub>2.5</sub>; and ultra-fine, PM<sub>0.1</sub>) and chemical composition depending on the source of the particles. Therefore the chemical constituents of particulate matter to which pregnant women are exposed vary across different geographic areas potentially resulting in different associations between particles and adverse birth outcomes [11]. Most studies have evaluated trimester-specific exposures and questions remain whether shorter time periods during pregnancy might better reflect the critical window of susceptibility to air pollution for these outcomes.

With approximately one of every ten newborns born before 37 completed weeks of gestation, PTB is a critical public health problem in the US. Preterm infants experience higher risks for a host of conditions and are three times as likely to die in infancy compared to infants born full term [12]. Infants born at the earliest gestations are at the highest risk for complications and infant death. Despite sizeable efforts to identify risk factors, the etiology of PTB remains poorly understood. Moreover, as many cases of mildly PTB are iatrogenic [13], their etiology differs from that of moderately and severely PTB. Additionally, full term infants may be born small for their gestational age (SGA), which is associated with an increased risk of fetal death, fetal distress and infant mortality [14].

We address a gap in the literature by conducting a population-based study to evaluate whether three mutually exclusive categories of PTB and term SGA are associated with PM<sub>2.5</sub> during narrow 4-week periods of gestation. Data were drawn from the Houston area, Harris County, Texas, with its large, diverse population and documented poor air quality. We evaluated associations in each 4-week period while adjusting for pollutant levels in the other

relevant 4-week exposure windows. We also excluded infants with birth defects who are at increased risk for PTB and term SGA that may involve mechanisms other than those related to air pollutant exposure.

## Materials and Methods

### Study Population

Our study population included all live births occurring between January 1st of 2005 and December 31st of 2007 among women who resided in Harris County, Texas at the time of delivery. We obtained electronic records of births already geocoded for maternal residence at delivery from the Texas Department of State Health Services (TX DSHS) (n = 222,735). Birth record data were prepared for analyses using SAS version 9.2 (SAS Institute Inc., Cary, NC) and Arc GIS 10 (ESRI, Redlands, CA). Institutional Review Board approvals were obtained from The University of Texas Health Science Center at Houston and the TX DSHS.

### Classification of Preterm Birth and SGA

Two estimates of gestational age at birth were computed from birth record data, one based on the first day of the last menstrual period (LMP) and the other based on the assessment of the physician who delivered the infant. We used the estimate based on the LMP [15] unless this variable was missing (1.8 %) or when the difference between the two estimates of gestational age was large (2–23 weeks) (9.7 %). If LMP was used to estimate gestational age, then the date of LMP was assigned as the first day of pregnancy; if the physician's estimate was used to estimate gestational age, then the first day of pregnancy was computed by subtracting the estimate of the gestational age at birth from the infant's date of birth.

Preterm births were classified into the following mutually exclusive categories: mildly PTB (33–36 completed weeks of gestation), moderately PTB (29–32 completed weeks of gestation) and severely PTB (20–28 completed weeks of gestation). Infants that were full term, i.e., had gestational ages of 37 weeks or more, were classified as term SGA if their birth weight was below the tenth percentile, using separate birth weight percentiles for male and female infants at 37, 38, 39, 40, 41, 42, 43, 44 and 45 weeks gestation [16].

### Air Pollution Exposure Assessment

Gravimetric monitoring data for PM<sub>2.5</sub> were obtained from the Air Quality System Data Mart of the US Environmental Protection Agency (EPA) for Harris County, Texas during the period of January 1, 2004 through December 31, 2007 (<http://www.epa.gov/ttn/airs/aqsdatamart/index.htm>). Speciated particulate matter data were not available. Six PM<sub>2.5</sub> monitors were operating in the county (three monitors in 2006 and 2007), each reporting 24-hour average concentrations in units of µg/m<sup>3</sup> at a sampling frequency that varied by monitoring location (i.e., every day, every third day or every sixth day). We were interested in accounting for spatial and temporal variability in ambient PM<sub>2.5</sub> levels and accessed additional data from monitoring locations in counties contiguous to Harris County from the US EPA Data Mart. However, when we attempted to apply kriging, a geostatistical technique for optimal linear spatial prediction at unmonitored sites [17], we were unable to generate

predictions due to a limited number of monitoring sites. Hence, we generated the county's daily mean value by averaging 24-hour concentrations ( $n = 1,383$ ) across all monitoring locations (mean  $\pm$  SD,  $14.62 \pm 6.04$ ; minimum, 2.65; fifth percentile, 6.64; tenth percentile, 7.90; 25th percentile, 10.40; 50th percentile, 13.60; 75th percentile, 17.90; and 90th percentile, 22.68; maximum, 46.2). Using the time series of daily air pollutant levels and information about the mother's first day of pregnancy as described above, we computed the mean exposure to  $PM_{2.5}$  for every 4 weeks (28 days) of a mother's pregnancy. R programming language (version  $\times 64$  2.13.0) was used to compute the time series of daily air pollutant levels and the 4-week exposure metrics for each mother during her pregnancy.

### Birth Certificate Data

Variables include the following: infant sex, maternal age, race/ethnicity, educational status, smoking and method of type of payment for delivery. Additionally, new to Texas birth certificates in 2005 and later, data were also available on mother's report of her weight prior to pregnancy and height, which were used to compute maternal body mass index (BMI) (weight in kilograms divided by height in meters squared) and categorized as follows per US Centers for Disease Control and Prevention (CDC) guidelines for females [18]: being underweight ( $<18.5 \text{ kg/m}^2$ ), normal weight ( $18.5\text{--}24.9 \text{ kg/m}^2$ ), overweight ( $25.0\text{--}29.9 \text{ kg/m}^2$ ) or obese ( $\geq 30 \text{ kg/m}^2$ ). For women aged 19 years and younger, BMI categories were based on percentile ranges, as per CDC guidelines for teenagers. We created a three-level variable for mother's level of support received from the infant's father: mother married; mother not married but reported information on infant's father (age, education or address); and mother not married and did not report information about the infant's father.

### Statistical Analysis

To assess the potential for multicollinearity between our  $PM_{2.5}$  exposure metrics (computed for every 4 weeks of pregnancy), we performed correlation analyses for all exposure metrics and obtained correlation coefficients that ranged from  $-0.45$  to  $0.56$ . Logistic regression analyses were then used to examine the associations between PTB and term SGA as the outcomes and the air pollution metrics (modeled as a continuous variable) as the predictors. Separate analyses were conducted for each outcome where the comparison group for the PTB outcomes was non-PTB and the comparison group for term SGA was all term, non-SGA. To maintain comparisons between the index and referent groups for the gestational ages of the infant and account for variable lengths of pregnancy, we applied the following restrictions: models assessing mildly PTB included each of the eight 4-week periods prior to 33 weeks gestation, models assessing moderately PTB included each of the seven 4-week periods prior to 29 weeks gestation and models assessing severely PTB included each of the five 4-week periods prior to 21 weeks gestation. Because our analyses of term SGA was restricted to full term infants, logistic regression models included all of the nine 4-week periods for this outcome. Thus, we calculated odds ratios (ORs) associated with each of the relevant 4-week time periods during pregnancy and each OR was adjusted for covariates including levels of  $PM_{2.5}$  present during all other relevant 4-week time periods.

Additionally, the analysis controlled for the following variables (obtained from information reported on the birth records) that changed the estimate for one or more of the  $PM_{2.5}$  metrics

by more than 10 % in one of our models: birth season (summer, June–August; fall, September–November; winter, December–February; and spring, March–May); mother’s smoking status (no smoking, smoking prior to pregnancy, and smoking throughout pregnancy); race (non-Hispanic white, Hispanic-foreign born; Hispanic-US born, black, and other); level of education (<12 years, High School Graduate or General Equivalency Diploma (GED), 13–15 years, and 16+ years); age; BMI prior to pregnancy; support received from infant’s father; receipt of Women, Infants, and Children (WIC) services; trimester prenatal care initiated; parity; and type of payment for delivery (private insurance, Medicaid, self-pay, other/missing).

### Sensitivity Analyses

Because studies have reported associations between levels of air pollution and birth defects [19, 20] and between birth defects and PTB [21], we also linked birth records to the records of the Texas Birth Defects Registry, covering the period 2005–2007. This linkage allowed us to perform analyses excluding all infants with birth defects recorded in the registry.

### Goodness of Fit

Using a significance level of 0.05, we applied the Hosmer and Lemeshow [22] Goodness of Fit test to the fully adjusted models and detected no evidence of a lack of fit for five out of the eight models that we ran. For the remaining three models (term SGA with and without birth defects and severely PTB without birth defects), we ran the goodness of fit test on the models that included the air pollution exposure metrics only; in these cases, we failed to reject the null hypothesis that the model fits. Hence, the lack of fit in the fully adjusted models for term SGA and severely PTB (without birth defects) appears to have arisen because of the added covariates rather than due to a lack of fit for the PM<sub>2.5</sub> exposure metrics. Further, when comparing the two models (exposure metrics only vs. model with other covariates) we found a 11 % (1–4 weeks gestation) and 18 % (17–20 weeks) difference in results between the two models for severely PTB without birth defects suggesting evidence of joint confounding whereas we found little difference in the results for term SGA. For ease of interpretation, we have chosen to report on the fully adjusted models for all outcomes (mildly PTB, moderately PTB, severely PTB and term SGA), with and without birth defects.

All logistic regression analyses were conducted using SAS version 9.2 (SAS Institute Inc., Cary, NC).

### Results

Among the 222,735 computerized records for births occurring in Harris County, we omitted 34,191 deliveries of mothers who delivered but did not live in Harris County and 4,564 deliveries of mothers with missing addresses. We further excluded deliveries with missing birth weight (n = 13) or gestational age (n = 41). Multiple births (n = 5,665), infants with computed gestational ages <20 or >52 weeks (n = 445), or implausible combinations of gestational age and birth weight (n = 466) were also excluded. After these exclusions, there remained 177,816 singleton births.

Table 1 presents the proportion of PTB and term SGA in our study population stratified by maternal and infant characteristics. PTBs accounted for 10.4 % of all singleton births occurring to mothers residing in Harris County during the study period with 15,220 mildly PTB, 1,906 moderately PTB, and 1,363 severely PTB. SGA births accounted for 10.8 % of all full term singleton births (n = 17,220).

Table 2 shows fully adjusted ORs for the associations between PM<sub>2.5</sub> and mildly PTB, moderately PTB, severely PTB and term SGA because we detected some differences in the magnitude of the OR in these models when compared with the results from the regression analyses that included the air pollutant metrics only (results not shown). A 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> levels during the first 4 weeks of pregnancy was significantly associated with mildly PTB [OR 1.16, 95 % confidence interval (95 % CI) 1.05, 1.28], moderately PTB (OR 1.71, 95 % CI 1.30, 2.24) and severely PTB (OR 1.73, 95 % CI 1.25, 2.39). Elevated ORs were also associated with a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> levels during the third 4-week gestational period (9–12 weeks) for both mildly (OR 1.26, 95 % CI 1.13, 1.41 and moderately (OR 1.71, 95 % CI 1.25, 2.33) PTB. Among severely PTB, while an increase in the OR was observed during the same gestational period, the OR was less precise (OR 1.21, 95 % CI 0.84, 1.74).

Among all three groups of PTB, ambient air levels of PM<sub>2.5</sub> occurring during the last relevant 4-week period of pregnancy were associated with increased odds of PTB. Mildly PTB was significantly associated with a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> occurring during the 4-week period between 29 and 32 weeks of gestation (OR 1.30, 95 % CI 1.17, 1.45); moderately PTB was significantly associated with a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> during the 4-week period between 25 and 28 weeks of gestation (OR 1.80, 95 % CI 1.34, 2.39); and severely PTB was significantly associated with a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> during the 4-week period between 17 and 20 weeks of gestation (OR 1.65, 95 % CI 1.14, 2.37). No other significant associations between levels of PM<sub>2.5</sub> during specific gestational time periods and mildly, moderately or severely PTB were found. Among term SGA, the only significant association with levels of PM<sub>2.5</sub> was during the 4-week period between 21 and 24 weeks gestation: OR 1.13, 95 % CI 1.01, 1.25 (Table 2).

Because infants with birth defects experience increased risks for PTB and fetal growth restriction [21] all infants with one or more birth defects were excluded from the analyses irrespective of whether they were PTB, term SGA or full term (see Table 3). After excluding infants with one or more birth defects, mildly PTB was significantly associated with a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> occurring during the 4-week period between 29 and 32 weeks of gestation (OR 1.30, 95 % CI 1.17, 1.46). For moderately PTB, we observed ORs of 1.91 [95 % CI 1.44, 2.54] and 1.93 [95 % CI 1.43, 2.60] for a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> exposure during the first month of gestation and the 4-week period between 25 and 28 weeks of pregnancy, respectively. For severely PTB, a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> exposure was associated with the first month of gestation (ORs of 1.89, 95 % CI 1.33, 2.68) and the 4-week period between 17 and 20 weeks of gestation (1.69, 95 % CI 1.15, 2.50). No significant associations were found among term SGA and ambient levels of PM<sub>2.5</sub> when all infants with birth defects were excluded (Table 3).

## Discussion

Temporal variability in air pollutant levels over a 4-year period in the study area contributed to differential exposures to ambient air levels of PM<sub>2.5</sub>, allowing for an evaluation of PM<sub>2.5</sub> levels during narrow gestational periods. We detected associations between PTB and maternal exposure to ambient levels of PM<sub>2.5</sub> during the first month of pregnancy and during the last 4-week period for which pregnancies were at risk of each of three levels of PTB with stronger associations observed for infants born moderately (29–32 completed weeks of gestation) or severely (20–28 completed weeks of gestation) preterm. We also detected stronger associations when infants with one or more congenital anomalies were excluded, based on records from an active, statewide population-based birth defects registry. We are unaware of other studies that have made such linkages and yet this element of our study design is likely important as the excess risks of PTB and fetal growth restriction among infants with birth defects [21] may be due to other biological mechanisms.

Assessing exposures for each month of gestational age while controlling for PM<sub>2.5</sub> concentrations in the other months of pregnancy allowed us to more accurately assess potentially susceptible exposure windows. The majority of air pollution studies that have investigated associations with adverse birth outcomes have relied upon exposure metrics computed for each trimester of pregnancy, although some have also focused on the first month of gestation [23–28] or periods at the end of pregnancy (e.g., the last 2, 4 or 6 weeks of gestation) [23, 26–30]. However, even fewer studies evaluated associations with air pollution for every month of pregnancy and those that did focused on birth outcomes other than PTB [31–34].

Because multiple etiologic pathways characterize PTB, we conducted separate analyses of mildly, moderately and severely preterm infants. In the first 4 weeks of pregnancy excluding infants with birth defects, we found a 16 % increase in risk for a 10-unit increase in PM<sub>2.5</sub> for mildly PTB as compared to an increase of 91 or 89 % for infants born moderately and severely preterm. Most previous studies typically evaluate PTB as a single category (i.e., <37 completed weeks of gestation). However, Wu et al. [35] observed higher ORs for infants born before 30 weeks of gestation compared with the ORs for all PTBs. Compared to all PTBs, a Canadian study also reported larger associations between PM<sub>2.5</sub> exposures and very preterm (<30 weeks) births [27].

Evidence from this study suggests that the most vulnerable period for PTB occurs early and late in pregnancy, suggesting multiple pathways for the effect of PM<sub>2.5</sub> on PTB. Fetal requirements for oxygen and nutrients place increasing physiological demands upon the mother during pregnancy, and the greatest absolute demands occur between 20 and 40 weeks of gestation when the fetus grows from an average weight of 400–3,500 grams [16]. It has been proposed [36] that particulate matter causes inflammation of the maternal lungs and the placenta, which may result in a suboptimal supply of oxygen and nutrients to the fetus, particularly in the last half of pregnancy when the demand is greatest. While effects were observed at the end of pregnancy, we found stronger effects during the first 4 weeks of pregnancy indicating that an optimal blood supply during implantation and early embryonic growth is important for a healthy pregnancy. This finding is consistent with the observation

of Smith et al. [37] that infants born moderately to severely preterm or SGA were more likely to be measured as small for gestational age by a first trimester ultrasound exam. Thus, although the greatest amount of absolute growth occurs during the last 20 weeks of pregnancy, optimal conditions at the time of implantation or early embryonic life also appear to be important.

Among term SGA, the only significant association that we detected with a  $10 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$  levels was during the 4-week period from 21 to 24 weeks gestation. When infants with birth defects were excluded the odds ratio diminished slightly. Previous studies of the associations between SGA and  $\text{PM}_{2.5}$  have reported equivocal findings. For a unit increase in  $\text{PM}_{2.5}$  levels ( $\mu\text{g}/\text{m}^3$ ) during the second trimester of pregnancy, Mannes et al. [38] reported a three percent increase in the odds of SGA for infants born in Sydney, Australia (adjusted odds ratio = 1.03 [95 % CI 1.01–1.05]). No effects were observed during the first or third trimester or during the month before birth. However, Liu et al. [39] reported positive associations between SGA and  $\text{PM}_{2.5}$  in all three trimesters whereas Rich et al. [40] observed positive associations in the first and third trimesters, but not in the second trimester of pregnancy.

Our study had several strengths. The catchment area included all live singleton births in Harris County and, thus, the potential for selection bias was diminished as compared, for example, to studies that restrict analyses to women on the basis of their residential proximity to the closest monitor. In a review of air pollution and adverse birth outcomes, smoking and BMI are missing in many studies [8]. Texas birth records now report on maternal smoking both before and during pregnancy and on pre-pregnancy BMI. Both are important determinants of PTB and term SGA [13, 37, 41, 42] and were included in the final models that are reported in this study. Nonetheless, we recognize that smoking is under reported on birth certificates [43] and, therefore, residual confounding due to smoking may still be present. We also conducted additional post hoc analyses stratified by mother's smoking status and did not find, as expected, significant differences for nonsmokers when compared to our current findings (95 % of the women reported no smoking during pregnancy). Due to small sample sizes, unstable estimates were generated for mothers who quit smoking prior to pregnancy and mothers who smoked throughout their pregnancy.

We could have constructed a time series of maximum daily averages (as measured at a single monitor) rather than mean averages across all monitoring locations but this approach has a serious limitation in terms of representing "the county experience" and we chose not to use this alternative time series in our study. Because our exposure metrics were temporally-based, residential mobility during pregnancy would not have introduced error in our study unless a mother lived outside of Harris County during pregnancy and moved to a residence within the county at the time of delivery. In a previous study, we found that changes in maternal residence during pregnancy (comparing addresses at conception and delivery during pregnancy among Texas mothers participating in the National Birth Defects Prevention Study) were generally within short distances (median distance moved was within 4 miles) [44].



A study aimed at characterizing spatial and temporal variability in  $PM_{2.5}$  levels in the greater Houston area reported spatially homogenous levels when the averaging time extended over seasons or longer time periods [45]. However, our exposure periods were shorter in duration (4 weeks) and some misclassification of exposure is likely to have occurred. Similar to Warren et al. [6] who were unable to apply their spatial model for evaluating air pollutant effects when the geographic region for analyses diminished in size (from state-level to Harris County), our attempts to generate spatially resolved kriged estimates were also unsuccessful because of too few monitoring locations in the study area. Interestingly, a recent meta-analysis examined associations between maternal exposure to particulate air pollution and term and low birth weight reported that studies applying a temporal rather than a spatial–temporal contrast in their exposure assessment tended to report stronger associations [46] and we do not know if patterns would be similar if analyses were extended to studies of particulate matter and PTB.

Another limitation of our study is that we could not evaluate alcohol use, passive smoking or nutritional status as potential confounding variables. However, results from a nested case–control study [47] suggest that effects of air pollution on PTB were not affected by lack of control for these risk factors. Finally, while we did not address the association between air pollutants and late fetal death, very few studies of air pollution and late fetal death have been published in part because fetal death certificates undercount stillbirths [48]. Researchers that have access to unbiased samples of pregnancies affected by late fetal death may wish to consider undertaking studies in this relatively unexplored area.

## Conclusions

This study assessed associations between maternal exposure to ambient levels of  $PM_{2.5}$  and PTB and term SGA in Houston, Harris County, Texas, assessing narrower windows of susceptibility than were typically examined in previous investigations. Our analyses corroborate reports in other parts of the US and elsewhere with findings that maternal exposure to ambient levels of  $PM_{2.5}$  is associated with PTB. Our strongest associations were detected during the first and last 4 weeks of pregnancy and were also higher among infants born moderately or severely premature. To our knowledge our study is the first of its kind to evaluate more narrow windows of exposure to air pollution in a large population-based study and adds to the growing literature on the association between particulate matter and adverse birth outcomes. Further, to mitigate the consequences of periods of diminished air quality in the region that range from local air quality alerts to enforcement of stricter national ambient air quality standards for  $PM_{2.5}$ , interventions are warranted.

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## Abbreviations

AQS

Air Quality System

<b>BMI</b>	Body mass index
<b>CDC</b>	Centers for Disease Control
<b>EPA</b>	US Environmental Protection Agency
<b>LMP</b>	Last menstrual period
<b>PM<sub>2.5</sub></b>	Particulate matter <2.5 $\mu$ m in aerodynamic diameter
<b>PTB</b>	Preterm birth
<b>SGA</b>	Small for gestational age
<b>US</b>	United States
<b>WIC Women</b>	infants, and children

## References

1. Institute for Health Policy. A closer look at air pollution in Houston; identifying priority health risks: Report of the Mayor's task force on the health effects of air pollution. Houston, TX: The University of Texas, Health Science Center at Houston and the City of Houston; 2006.
2. Institute for Health Policy. Health of Houston survey HHS 201 a first look. Houston, TX: The University of Texas, School of Public Health; 2011.
3. Buffler PA, Cooper SP, Stinnett S, et al. Air pollution and lung cancer mortality in Harris County, Texas, 1979–1981. *American Journal of Epidemiology*. 1988; 128(4):683–699. [PubMed: 3421235]
4. Ensor KB, Raun LH, Persse D. A case-crossover analysis of out-of-hospital cardiac arrest and air pollution. *Circulation*. 2013; doi: 10.1161/CIRCULATIONAHA.113.000027
5. Whitworth KW, Symanski E, Coker AL. Childhood lymphohematopoietic cancer incidence and hazardous air pollutants in southeast Texas, 1995–2004. *Environmental Health Perspectives*. 2008; 116(11):1576–1580. DOI: 10.1289/ehp.11593 [PubMed: 19057714]
6. Warren J, Fuentes M, Herring A, et al. Spatial-temporal modeling of the association between air pollution exposure and preterm birth: Identifying critical windows of exposure. *Biometric*. 2012; 68(4):1157–1167. DOI: 10.1111/j.1541-0420.2012.01774.x
7. Maisonet M, Correa A, Misra D, et al. A review of the literature on the effects of ambient air pollution on fetal growth. *Environmental Research*. 2004; 95(1):106–115. [PubMed: 15068936]
8. Shah PS, Balkhair T. Air pollution and birth outcomes: A systematic review. *Environment International*. 2011; 37(2):498–516. DOI: 10.1016/j.envint.2010.10.009 [PubMed: 21112090]
9. Sram RJ, Binkova B, Dejmek J, et al. Ambient air pollution and pregnancy outcomes: A review of the literature. *Environmental Health Perspectives*. 2005; 113(4):375–382. [PubMed: 15811825]
10. Stieb DM, Chen L, Eshoul M, et al. Ambient air pollution, birth weight and preterm birth: A systematic review and meta-analysis. *Environmental Research*. 2012; 117:100–111. DOI: 10.1016/j.envres.2012.05.007 [PubMed: 22726801]
11. Parker JD, Woodruff TJ. Influences of study design and location on the relationship between particulate matter air pollution and birthweight. *Paediatric and Perinatal Epidemiology*. 2008; 22(3):214–227. DOI: 10.1111/j.1365-3016.2008.00931.x [PubMed: 18426516]
12. Damus K. Prevention of preterm birth: A renewed national priority. *Current Opinion Obstetrics and Gynecology*. 2008; 20(6):590–596. DOI: 10.1097/GCO.0b013e3283186964
13. Savitz, DA.; Pastore, LM. Causes of prematurity effectiveness and implementation. Cambridge: Cambridge University Press; 1999.
14. Kramer MS, Olivier M, McLean FH, et al. Impact of intrauterine growth retardation and body proportionality on fetal and neonatal outcome. *Pediatrics*. 1990; 86(5):707–713. [PubMed: 2235224]

15. David RJ. The quality and completeness of birthweight and gestational age data in computerized birth files. *American Journal of Public Health*. 1980; 70(9):964–973. [PubMed: 7406096]
16. Oken E, Kleinman KP, Rich-Edwards J, et al. A nearly continuous measure of birth weight for gestational age using a United States national reference. *BMC pediatrics*. 2003; 3:6.doi: 10.1186/1471-2431-3-6 [PubMed: 12848901]
17. Waller, LA.; Gotway, CA. *Applied spatial statistics for public health data*. Hoboken, NJ: Wiley; 2004.
18. US Centers for Disease Control and Prevention. About BMI for adults. 2011. [http://www.cdc.gov/healthyweight/assessing/bmi/adult\\_bmi/index.html](http://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html)
19. Gilboa SM, Mendola P, Olshan AF, et al. Relation between ambient air quality and selected birth defects, seven county study, Texas, 1997–2000. *American Journal of Epidemiology*. 2005; 162(3): 238–252. DOI: 10.1093/aje/kwi189 [PubMed: 15987727]
20. Lupo PJ, Symanski E, Waller DK, et al. Maternal exposure to ambient levels of benzene and neural tube defects among offspring: Texas, 1999–2004. *Environmental Health Perspectives*. 2011; 119(3):397–402. DOI: 10.1289/ehp.1002212 [PubMed: 20923742]
21. Honein MA, Kirby RS, Meyer RE, et al. The association between major birth defects and preterm birth. *Maternal and Child Health Journal*. 2009; 13(2):164–175. DOI: 10.1007/s10995-008-0348-y [PubMed: 18484173]
22. Hosmer, DW.; Lemeshow, S. *Applied logistic regression*. New York: Wiley; 2000.
23. Wilhelm M, Ritz B. Local variations in CO and particulate air pollution and adverse birth outcomes in Los Angeles County, California, USA. *Environmental Health Perspectives*. 2005; 113(9):1212–1221. [PubMed: 16140630]
24. Hansen C, Neller A, Williams G, et al. Maternal exposure to low levels of ambient air pollution and preterm birth in Brisbane, Australia. *BJOG*. 2006; 113(8):935–941. DOI: 10.1111/j.1471-0528.2006.01010.x [PubMed: 16907939]
25. Ritz B, Yu F, Chapa G, et al. Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. *Epidemiology*. 2000; 11(5):502–511. [PubMed: 10955401]
26. Huynh M, Woodruff TJ, Parker JD, et al. Relationships between air pollution and preterm birth in California. *Paediatric and Perinatal Epidemiology*. 2006; 20(6):454–461. [PubMed: 17052280]
27. Brauer M, Lencar C, Tamburic L, et al. A cohort study of traffic-related air pollution impacts on birth outcomes. *Environmental Health Perspectives*. 2008; 116(5):680–686. [PubMed: 18470315]
28. Le HQ, Batterman SA, Wirth JJ, et al. Air pollutant exposure and preterm and term small-for-gestational-age births in Detroit, Michigan: Long-term trends and associations. *Environment International*. 2012; 44:7–17. DOI: 10.1016/j.envint.2012.01.003 [PubMed: 22314199]
29. Wu J, Wilhelm M, Chung J, et al. Comparing exposure assessment methods for traffic-related air pollution in an adverse pregnancy outcome study. *Environmental Research*. 2011; 111(5):685–692. DOI: 10.1016/j.envres.2011.03.008 [PubMed: 21453913]
30. Rudra CB, Williams MA, Sheppard L, et al. Ambient carbon monoxide and fine particulate matter in relation to preeclampsia and preterm delivery in western Washington State. *Environmental Health Perspectives*. 2011; 119(6):886–892. DOI: 10.1289/ehp.1002947 [PubMed: 21262595]
31. Dejmek J, Selevan SG, Benes I, et al. Fetal growth and maternal exposure to particulate matter during pregnancy. *Environmental Health Perspectives*. 1999; 107(6):475–480. [PubMed: 10339448]
32. Dejmek J, Solansky I, Benes I, et al. The impact of polycyclic aromatic hydrocarbons and fine particles on pregnancy outcome. *Environmental Health Perspectives*. 2000; 108(12):1159–1164. [PubMed: 11133396]
33. Darrow LA, Klein M, Strickland MJ, et al. Ambient air pollution and birth weight in full-term infants in Atlanta, 1994–2004. *Environmental Health Perspectives*. 2011; 119(5):731–737. DOI: 10.1289/ehp.1002785 [PubMed: 21156397]
34. Lee BE, Ha EH, Park HS, et al. Exposure to air pollution during different gestational phases contributes to risks of low birth weight. *Human Reproduction*. 2003; 18(3):638–643. [PubMed: 12615838]

35. Wu J, Ren C, Delfino RJ, et al. Association between local traffic-generated air pollution and preeclampsia and preterm delivery in the south coast air basin of California. *Environmental Health Perspectives*. 2009; 117(11):1773–1779. [PubMed: 20049131]
36. Kannan S, Misra DP, Dvonch JT, et al. Exposures to airborne particulate matter and adverse perinatal outcomes: A biologically plausible mechanistic framework for exploring potential effect modification by nutrition. *Environmental Health Perspectives*. 2006; 114(11):1636–1642. [PubMed: 17107846]
37. Smith GC, Shah I, Pell JP, et al. Maternal obesity in early pregnancy and risk of spontaneous and elective preterm deliveries: A retrospective cohort study. *American Journal of Public Health*. 2007; 97(1):157–162. DOI: 10.2105/AJPH.2005.074294 [PubMed: 17138924]
38. Mannes T, Jalaludin B, Morgan G, et al. Impact of ambient air pollution on birth weight in Sydney, Australia. *Occupational and Environmental Medicine*. 2005; 62(8):524–530. DOI: 10.1136/oem.2004.014282 [PubMed: 16046604]
39. Liu S, Krewski D, Shi Y, et al. Association between maternal exposure to ambient air pollutants during pregnancy and fetal growth restriction. *Journal of Exposure Science & Environmental Epidemiology*. 2007; 17(5):426–432. DOI: 10.1038/sj.jes.7500503 [PubMed: 16736056]
40. Rich DQ, Demissie K, Lu SE, et al. Ambient air pollutant concentrations during pregnancy and the risk of fetal growth restriction. *Journal of Epidemiology and Community Health*. 2009; 63(6):488–496. DOI: 10.1136/jech.2008.082792 [PubMed: 19359274]
41. Abrams B, Newman V. Small-for-gestational-age birth: Maternal predictors and comparison with risk factors of spontaneous preterm delivery in the same cohort. *American Journal of Obstetrics and Gynecology*. 1991; 164(3):785–790. [PubMed: 2003542]
42. Lieberman E, Gremy I, Lang JM, et al. Low birth-weight at term and the timing of fetal exposure to maternal smoking. *American Journal of Public Health*. 1994; 84(7):1127–1131. [PubMed: 8017537]
43. Dietz PM, Adams MM, Kendrick JS, et al. Completeness of ascertainment of prenatal smoking using birth certificates and confidential questionnaires: Variations by maternal attributes and infant birth weight. PRAMS working group. Pregnancy risk assessment monitoring system. *American Journal of Epidemiology*. 1998; 148(11):1048–1054. [PubMed: 9850126]
44. Lupo P, Symanski E, Chan W, et al. Differences in exposure assignment between conception and delivery: The impact of maternal mobility. *Paediatric and Perinatal Epidemiology*. 2010; 24:200–208. [PubMed: 20415777]
45. Russell M, Allen DT, Collins DR, et al. Daily, seasonal, and spatial trends in PM<sub>2.5</sub> mass and composition in Southeast Texas. *Aerosol Science and Technology*. 2004; 38(S1):14–26.
46. Dadvand P, Parker J, Bell ML, et al. Maternal exposure to particulate air pollution and term birth weight: A multi-country evaluation of effect and heterogeneity. *Environmental Health Perspectives*. 2013; 121(3):267–373. DOI: 10.1289/ehp.1205575 [PubMed: 23384584]
47. Ritz B, Wilhelm M, Hoggatt KJ, et al. Ambient air pollution and preterm birth in the environment and pregnancy outcomes study at the University of California, Los Angeles. *American Journal of Epidemiology*. 2007; 166(9):1045–1052. [PubMed: 17675655]
48. MacDorman MF, Kirmeyer S. Fetal and perinatal mortality, United States, 2005. *National Vital Statistics Reports: From the Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System*. 2009; 57(8):1–19.

**Table 1**  
Maternal and infant characteristics for all births, PTBs and SGA term births, Harris County, Texas, 2005–2007

Characteristic	All births	PTBs	n (%)	Term births	SGA term births	n (%)
Totals	177,816	18,489		159,327	17,220	
<i>Maternal race/ethnicity</i>						
Non-Hispanic white	40,572	3,997	(9.9)	36,575	2,883	(7.9)
African-American/Black	33,281	5,099	(15.3)	28,182	4,733	(16.8)
Hispanic white, US born	34,204	3,556	(10.4)	30,648	3,253	(10.6)
Hispanic white, foreign born	58,937	4,872	(8.3)	54,065	4,912	(9.1)
Other	10,822	965	(8.9)	9,857	1,439	(14.6)
<i>Maternal age (years)</i>						
16	4,101	489	(11.9)	3,612	583	(16.1)
17–19	18,383	2,032	(11.1)	16,351	2,410	(14.7)
20–24	46,407	4,786	(10.3)	41,621	5,135	(12.3)
25–29	48,512	4,653	(9.6)	43,859	4,406	(10.1)
30–34	38,164	3,856	(10.1)	34,308	3,001	(8.8)
35–39	18,174	2,111	(11.6)	16,063	1,315	(8.2)
40	4,073	562	(13.8)	3,511	369	(10.5)
Missing	2	0	(0.0)	2	1	(50.0)
<i>Parity</i>						
First birth	68,405	7,239	(10.6)	61,166	8,343	(13.6)
Second or subsequent birth	109,355	11,240	(10.3)	98,115	8,870	(9.0)
Missing	56	10	(17.9)	46	7	(15.2)
<i>Infant sex</i>						
Male	90,745	9,980	(11.0)	80,765	9,078	(11.2)
Female	87,071	8,509	(9.8)	78,562	8,142	(10.4)
<i>Maternal education (years)</i>						
<12	65,684	6,505	(9.9)	59,179	6,714	(11.4)
High school graduate or GED	41,530	4,524	(10.9)	37,006	4,486	(12.1)
13–15	37,667	4,328	(11.5)	33,339	3,493	(10.5)
16+	32,725	3,078	(9.4)	29,647	2,503	(8.4)

Characteristic	All births	PTBs	n (%)	Term births	SGA term births	n (%)
Missing	210	54	(25.7)	156	24	(15.4)
<i>Source of payment for delivery</i>						
Private insurance	62,906	6,608	(10.5)	56,298	4,901	(8.7)
Medicaid	64,225	7,226	(11.3)	56,999	7,563	(13.3)
Self-pay	38,424	3,422	(8.9)	35,002	3,707	(10.6)
Other	11,900	1,197	(10.1)	10,703	1,018	(9.5)
Unknown	361	36	(10.0)	325	31	(9.5)
<i>Mother on WIC during pregnancy</i>						
No	84,708	9,472	(11.2)	75,236	7,436	(9.9)
Yes	92,663	8,939	(9.7)	83,724	9,742	(11.6)
Missing	445	78	(17.5)	367	42	(11.4)
<i>Father support</i>						
Married	102,547	9,713	(9.5)	92,834	8,426	(9.1)
Unmarried, with father's support	47,311	4,984	(10.5)	42,327	5,139	(12.1)
Unmarried, no father's support	27,958	3,792	(13.6)	24,166	3,655	(15.1)
<i>Trimester prenatal care began</i>						
1st trimester	99,937	9,827	(9.8)	90,110	9,031	(10.0)
2nd trimester	48,719	4,382	(9.0)	44,337	4,984	(11.2)
3rd trimester or none	20,831	2,807	(13.5)	18,024	2,464	(13.7)
Missing	8,329	1,473	(17.7)	6,856	741	(10.8)
<i>Maternal smoking</i>						
No smoking	169,527	17,384	(10.3)	152,143	16,050	(10.6)
Stopped smoking trimester 1 or 2	3,503	380	(10.9)	3,123	361	(11.6)
Smoked throughout pregnancy	4,472	674	(15.1)	3,798	763	(20.1)
Missing	314	51	(16.2)	263	46	(17.5)
<i>Pre-pregnancy BMI (kg/m<sup>2</sup>)</i>						
Normal (18.5–24.9)	86,877	8,689	(10.0)	78,188	9,205	(11.8)
Underweight (<18.5)	7,287	834	(11.5)	6,453	1,205	(18.7)
Overweight (25.0–29.9)	49,550	4,964	(10.0)	44,586	4,189	(9.4)
Obese (≥ 30)	33,127	3,801	(11.5)	29,326	2,526	(8.6)
Missing	975	201	(20.6)	774	95	(12.3)

*PTB* preterm birth, *SGA* small for gestational age, *US* United States, *GED* certificate of high school equivalency, *WIC* Women, Infants, and Children, *BMJ* body mass index

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**Table 2**

Adjusted<sup>a</sup> ORs and 95 % CIs for the association between a 10-unit increase in maternal exposure to ambient levels of PM<sub>2.5</sub> (µg/m<sup>3</sup>) and moderately, mildly and severely PTBs and term SGA in Harris County, TX, 2005–2007, including infants with birth defects (n = 177,816)

Period of pregnancy (weeks)	Severely PTBs (n = 1,363) OR [95 % CI]	Moderately PTBs (n = 1,906) OR [95 % CI]	Mildly PTBs (n = 15,220) OR [95 % CI]	SGA (n = 17,220) OR [95 % CI]
1–4	1.73 [1.25, 2.39]	1.71 [1.30, 2.24]	1.16 [1.05, 1.28]	1.04 [0.95, 1.14]
5–8	0.85 [0.59, 1.24]	1.05 [0.77, 1.44]	1.11 [0.99, 1.24]	0.99 [0.89, 1.10]
9–12	1.21 [0.84, 1.74]	1.71 [1.25, 2.33]	1.26 [1.13, 1.41]	1.07 [0.96, 1.19]
13–16	0.75 [0.54, 1.07]	0.94 [0.70, 1.25]	0.95 [0.85, 1.05]	1.05 [0.95, 1.17]
17–20	1.65 [1.14, 2.37]	0.97 [0.71, 1.32]	1.14 [1.02, 1.27]	0.87 [0.78, 0.97]
21–24	–	0.90 [0.67, 1.22]	0.82 [0.73, 0.91]	1.13 [1.01, 1.25]
25–28	–	1.80 [1.34, 2.39]	1.10 [0.99, 1.22]	1.02 [0.92, 1.13]
29–32	–	–	1.30 [1.17, 1.45]	1.01 [0.91, 1.12]
33–36	–	–	–	0.93 [0.84, 1.03]

OR odds ratio, CI confidence intervals, SGA small for gestational age, WIC Women, Infants, and Children, BMI body mass index

<sup>a</sup>Adjusted for pollutant concentrations during the other 4-week periods, season, race, level of education, age, BMI status, support received from infant's father, receipt of WIC services; trimester prenatal care initiated; parity; and type of payment for delivery



**Table 3**

Adjusted<sup>a</sup> ORs and 95 % CIs for the association between a 10-unit increase in maternal exposure to ambient levels of PM<sub>2.5</sub> (µg/m<sup>3</sup>) and moderately, mildly and severely PTBs and term SGA in Harris County, TX, 2005–2007, excluding infants with birth defects (n = 171,923)

Period of pregnancy (weeks)	Severely PTBs (n = 1,188) OR [95 % CI]	Moderately PTBs (n = 1,701) OR [95 % CI]	Mildly PTBs (n = 14,397) OR [95 % CI]	SGA (n = 16,520) OR [95 % CI]
1–4	1.89 [1.33, 2.68]	1.91 [1.44, 2.54]	1.16 [1.05, 1.28]	1.04 [0.94, 1.14]
5–8	0.90 [0.60, 1.33]	1.09 [0.78, 1.52]	1.08 [0.96, 1.21]	0.98 [0.88, 1.10]
9–12	1.27 [0.87, 1.87]	1.64 [1.18, 2.28]	1.26 [1.12, 1.41]	1.09 [0.98, 1.22]
13–16	0.71 [0.49, 1.03]	0.96 [0.71, 1.32]	0.96 [0.86, 1.07]	1.05 [0.95, 1.17]
17–20	1.69 [1.15, 2.50]	0.92 [0.67, 1.27]	1.16 [1.03, 1.30]	0.87 [0.78, 0.98]
21–24	–	0.88 [0.64, 1.21]	0.83 [0.74, 0.93]	1.11 [0.98, 1.23]
25–28	–	1.93 [1.43, 2.60]	1.10 [0.99, 1.23]	1.01 [0.92, 1.12]
29–32	–	–	1.30 [1.17, 1.46]	1.01 [0.91, 1.12]
33–36	–	–	–	0.94 [0.84, 1.04]

OR odds ratio, CI confidence intervals, SGA small for gestational age, WIC Women, Infants, and Children, BMI body mass index

<sup>a</sup>Adjusted for pollutant concentrations during the other 4-week periods, season, race, level of education, age, BMI status, support received from infant's father, receipt of WIC services; trimester prenatal care initiated; parity; and type of payment for delivery