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Effect of PM 2.5 and PM 10 on Birth Outcomes in Hillsborough County, Florida.

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An abstract of

A thesis submitted to the Faculty of the

Rollins School of Public Health Emory University

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ABSTRACT

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There is a lot of concern on pollution and the constant rise in the rate of pollution in many cities in the United States. Particulate matter has been studied as an air pollutant with possible effects on birth outcomes, but results have varied widely on its effects. The study is based on birth records in Hillsborough County, Florida. The objective was to assess the association between air particulate pollutants and feto-infant morbidity outcomes; low birth weight, very low birth weight, preterm birth, very preterm birth, and small for gestational age. A second objective was to asess the effect of the pollutants on feto-infant morbidity outcomes is modified by racio-ethnic differences**.** For this study, birth data for 103, 961 newborns were successfully linked to air pollution data from Environmental Protection Agency (EPA) from 2000-2007. Logistic regression models were used to determine the association between pollutants and the birth outcomes and Chi square test for demographic data analysis. We observed increased risk for overall feto-infant morbidity outcome in women exposed to any of the particulate pollutants. 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 (OR=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 (OR=3.32, 95% CI=2.56-4.30). We concluded that 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, thus providing a target for intervention.

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INTRODUCTION

Air pollution and Particulate Matter

Air pollution has been noted and documented as far back as the 13th century with the use of coal. From the 20th century however, the compounds that contribute to air pollution have changed significantly to include motor vehicle and industrial sources as the major players.("History of air pollution," 2000). Environmental contaminants include air pollutants such as particulate matter (PM), nitrogen dioxide (NO2), sulfur dioxide (SO2), carbon monoxide (CO), and the secondary pollutant ozone (O3). These substances have been regulated in many parts of the world, and environmental levels are closely monitored. In the United States, the main sources of these pollutants are traffic and industrial emissions, and their measurements are routine using fixed monitoring stations in all states around the country(resources, 2010).

Particulate matter, also known as particle pollution or PM, is a complex mixture of extremely small particles and liquid droplets suspended in the air (E.P.A, May 2008). Sources of particulate matter either occur naturally, originating from volcanoes, dust storms, forest and grassland fires, living vegetation, and sea spray or by human activities, such as the burning of fossil fuels in vehicles, power plants and various industrial processes. The two types of particulate matter that are of concern are: PM 10, which is less than or equal to 10 micrometers in diameter, and PM 2.5, which is fine particles 2.5 micrometers in diameter and smaller(E.P.A, May 2008).There is also coarse PM which is particles greater than 2.5 micrometers but less than 10 micrometers. The health effects occur because PM10 is readily

inhalable and because of their size, they are not filtered and penetrate deeply into the body and cause cardiovascular and respiratory disorders. PM 2.5 penetrates deeper into the lungs

and has been shown to have a causal relationship with cardiovascular disease.(Brook et al., 2010)

Hillsborough County

Hillsborough County is located in the U.S. state of Florida. The 2000 population was 998,948 based on the census count. In 2007, the Hillsborough County Planning Commission conducted a population estimate that put the county's population at 1,204,770.("National Association of Counties," 2010)

In 2000, there were 391,357 households, and 255,164 families residing in the county. The population density was 951 people per square mile (367/km²). There were 425,962 housing units at an average density of 405 per square mile (156/km²). The racial makeup of the county was 75.17% White, 14.96% Black or African American, 0.39% Native American, 2.20% Asian, 0.07% Pacific Islander, 4.66% from other races, and a 2.56% from two or more races. 17.99% of the population was Hispanic or Latino of any race. The county is the thirty-second most populous county in the nation(U.S. Census 2004).

Of the 391,357 households, 31.40% had children under the age of 18 living with them, 47.70% were married couples living together, 13.20% had a female householder with no husband present, and 34.80% were non-families. 26.90% of all households were made up of individuals and 8.10% had someone living alone who was 65 years of age or older. The average household size was 2.51 and the average family size was 3.07 (U.S. Census 2004)

The age distribution was 25.30% under the age of 18, 9.30% from 18 to 24, 31.70% from 25 to 44, 21.70% from 45 to 64, and 12.00% were 65 years of age or older. The median age was 35 years. For every 100 females there were 95.80 males. For every 100 females age 18 and over, there were 92.70 males (U.S. Census 2004).

The median income for a household in the county was $40,663, and the median income for a family was $48,223. Males had a median income of $34,111 versus $26,962 for females. The per capita income for the county was $21,812. About 9.10% of families and 12.50% of the population were below the poverty line (U.S. Census 2004)

Air Pollution and Birth Outcomes

In recent years, there has been a growing interest in research which focuses on the effect and potential impact of prenatal exposure to air pollution on birth outcomes and this is due to the mounting evidence of the vulnerability of subgroups such as children. Studies have examined the association between exposure to air pollution and several birth outcomes that include birth weight (Ballester et al., 2010; Gouveia, Bremner, & Novaes, 2004), birth defects (Hansen, Barnett, Jalaludin, & Morgan, 2009), (Ren, Melly, & Schwartz, 2010) post neonatal infant mortality and morbidity (Suwanwaiphatthana, Ruangdej, & Turner-Henson, 2010; Woodruff, Grillo, & Schoendorf, 1997). These studies have produced results that are inconsistent, but many researchers believe that ambient air pollution during pregnancy is associated with adverse birth outcomes(Hansen, et al., 2009).

Air pollution exposure can be defined as a process of hazardous substances in air entering an organism through exposure inhalation. Other means by which individuals come into contact with environmental contaminants include inhalation, ingestion, and/or skin (dermal)

absorption, which often depends on the pollutant exposed to (Zou, Wilson, Zhan, & Zeng, 2009). Uncertainty associated with exposure assessment is considered to be a critical issue in most pollution studies. Ideally, individual-level exposures or personal exposure monitoring would be desirable but, researchers have to rely on other techniques, such as using distances from residence to the monitors on a regional scale, to assign pollution level to individuals.(Sagiv et al., 2005).

The mechanism of injury has been proposed by several authors, though the exact pathogenesis of how fetal growth is restricted is not known. One study hypothesized that the air pollutants alter cell activity or reduce the amount of oxygen and nutrients the fetus receives in utero.(Rich et al., 2009). Another study showed that umbilical cord blood levels of carboxyhaemoglobin were correlated with ambient levels of carbon monoxide (CO) bringing evidence of fetal exposure to outdoor levels of air pollution(Pereira et al., 1998). Other potential biological mechanisms have been described through which air pollution could influence pregnancy outcomes such as the induction of inflammation (placental, pulmonary, or systemic) and oxidative stress (Kannan, Misra, Dvonch, & Krishnakumar, 2007) endothelial dysfunction, increased blood viscosity, thrombosis(Mills et al., 2005; O'Neill et al., 2005), eventually resulting in suboptimal placentation (Dejmek, Selevan, Benes, Solansky, & Sram, 1999) and increased maternal susceptibility to infections (Slama et al., 2008).

Trimester-specific associations between pollutants and birth outcomes have also been described. In Sao Paolo, researchers found impaired growth in the first trimester as a result of high exposure to CO. (Gouveia, et al., 2004) where as 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.(Wang, Ding, Ryan, & Xu, 1997), this finding was replicated in another study in Atlanta(Darrow, Klein, Strickland, Mulholland, & Tolbert, 2010). The trimester-specific findings from these studies however, remain inconsistent because they show different windows of increased risk of reduced birth weight and IUGR for each pollutant. The explanations for the variations in results still remain unclear (Parker et al., 2011; Salam et al., 2005).

Present Study

This study focused on PM2.5, coarse fraction and PM 10 particulate matter and its effects on birth outcomes using ambient air quality monitoring data from Hillsborough County linked to hospital discharge data and vital statistic birth data for Hillsborough County that contain information on birth outcomes. Our overall objective was to assess the effects of PM 2.5 and PM 10 on birth outcomes in Hillsborough County, Florida.

METHODS

Research Hypothesis

We conducted the study with the following hypotheses:

- PM2.5, coarse fraction and PM10 impair fetal growth and developmental processes throughout the period of pregnancy leading to feto-infant morbidity: low birth weight (<2,500 grams), very low birth weight (<1,500 grams), preterm birth (<37 weeks), very preterm birth (< 33 weeks), and small for gestational age (SGA).
- The impact of the three pollutants on feto-infant morbidity outcomes is modified by racio-ethnic differences.

Study population

The study population includes all births in Hillsborough County, Florida between 2000 and 2007 obtained from birth certificate data from birth registry records. The de-identified birth data for Hillsborough County were linked to the Hospital Inpatient Discharge Data containing ICD9 procedure and diagnosis codes information and charges for mothers and their infants obtained from the Florida Agency for Healthcare Administration which was denoted as Linked HC.

We had a total of 104,108 pregnancies in the study of which 105 (0.1%) were excluded for being non-viable pregnancies i.e. (gestational age outside the range of 20-44 weeks). Thus a final dataset of 103,961 singleton births were used in the study.

The birth certificates contained all demographic information. The variables included racial/ethnic status (white non-Hispanic, black non-Hispanic, Hispanic and other); maternal age (< 35, ≥ 35 years old at time of delivery); marital status (single [which includes women whose marital status was classified as unknown, widowed or divorced], married); education (less than high school, high school or greater); parity (nulliparous, multiparous); obese (body mass index (BMI) ≥ 30 or non-obese (BMI<30)), cigarette smoking during pregnancy (yes or no), and adequacy of prenatal care (adequate or inadequate). BMI computation and comparison was only possible for births after February 2004 since prior to that period the information for height and pre-pregnancy weight was not collected on the birth certificate. 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 (Alexander GR et al. 1996). 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. Pre-pregnancy BMI was calculated from height and pre-pregnancy weight, as recorded on the birth certificate.

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), preeclampsia (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 (Kahn et al. 2009; Lydon-Rochelle et al. 2005)

The main outcomes of interest included low birth weight (<2,500 grams), very low birth weight (<1,500 grams), preterm birth (<37 weeks), very preterm birth (< 33 weeks), and small for gestational age (SGA). We defined SGA as less than the 10th percentile of birth weight for a given gestational age using population-based national reference curves (Alexander & Kogan, 1998). Sex and race-specific reference curves were used in this study. Gestational age was computed in weeks as the interval between 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 (Taffel, Johnson, & Heuser, 1982).

PM 2.5, Coarse fraction and PM 10 Measurements

There are 14 stationary monitors located in Hillsborough County, Florida. These are placed and regulated by the Environmental Protection Agency (E.P.A.) the monitors record 24-

hour average concentrations once every six days for PM 2.5, coarse fraction and PM10 (measured in $\mu g/m^3$). Recordings from 2000 to 2007 were obtained and the 24 hr daily average concentration was computed. The attainments of the network of monitoring stations and the data handling capabilities of EPA have been considered to be of high quality. The pollution data also included latitudes and longitudes which were converted to zip codes using data from the Hillsborough Environmental Pollution Commission (EPC). The Euclidian distances between the centroid of the mother's residential zip code during pregnancy (from the linked HC dataset) and the centroid of each zip codes of the monitoring stations were computed. 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). The monitoring site with the minimum distance to a residential zip code was assigned to the residential zip code as the monitor with the greatest exposure for the mothers within that zip code (Hansen, et al., 2009). . If there were missing data from the closest 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 (Hansen, et al., 2009).

Exposure Estimation

Maternal exposure to air pollution during pregnancy was estimated using date of birth of the infant and the length of each gestational age. This results in weekly PM 2.5, coarse fraction and PM 10 exposure data for each day of every pregnancy because 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 (Lee et al., 2003). Mean exposures to

each air pollutant for the duration of each pregnancy were then assigned to each birth. [N. Gouveia et al, 2004].

Statistical Analysis

Demographic data from the birth certificates were linked to the air pollution data using residential zip codes of the mother and monitor zip codes. Women were divided into two groups as follows: the exposed group if the mother was exposed to any of the three particulates during pregnancy and the unexposed group if the mother was not exposed to any of the particulates during pregnancy. We defined exposure to any of the particulates if the value of the particulate falls above the median. We also performed subgroup analysis by considering interactions between each of the particulates (bivariate and three way interaction).

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 feto-infant morbidity outcomes (low birth weight, very low birth weight, preterm, very preterm and small for gestational age) among the exposed group was compared with 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. 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

Of the overall sample of 103,961 singleton births from data years 2000 to 2007, 24,090 (23.2%) were categorized as exposed while the unexposed group consisted of 79,871 (76.8%) births. The crude proportions of demographic information between exposed and unexposed women in Table 1. Women who were exposed to any of the particulates were less educated, were more likely to be non-smokers, whites, single, nulliparous, and less likely to have adequate prenatal care. In contrast, a high proportion of black women were in the exposed group (Table 1).

The median values (Median ± SD) for the three particulate pollutants in this population were 11.28 [±10.98] for PM2.5; 24.35 [±25.0] for PM10 and 13.1 (±13.3) for the coarse fraction. Among the common obstetric complications analyzed, only anemia, gestational hypertension and chronic hypertension showed a significant difference between the two groups. The prevalence of diabetes mellitus gestational hypertension, preeclampsia, eclampsia, placenta abruption, placenta previa and renal diseases was not different between the two groups of women (Table 1). The mean gestational age was higher in the unexposed group compared to the exposed group (mean ± standard deviation=38.5 ±1.8 weeks versus 38.4± 1.9 weeks, respectively; $p < 0.01$). Also, the infants of women who were not exposed to any of the particulate matter were 18 grams heavier on average compared to women who were exposed to any of the particulate matter (mean ± standard deviation=3,312± 541 grams versus 3,294± 557 grams, respectively; $p < 0.01$).

Overall, 24,661 (23.7%) infants had any morbidity outcome with 6,670 (6.4%) of them having low birth weight, 999 (1.0%) were very low birth weight, 9.459 were preterm, 1,133

(1.1%) were very preterm and 8,733 (8.4%) were SGA. The breakdown of these crude frequencies of feto-infant morbidity outcomes between the two groups is given in Figure 2. The adjusted odds ratios for the association between women who were exposed to any particulate versus those who were not exposed are summarized in Table 2. We observed about 8% increased odds for any morbidity outcome in women exposed to any particulate (either coarse fraction, PM2.5 or PM10) considered in this study. Also, the exposed group of 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 (OR=1.27, 95% CI=1.08-1.49). The risk for very preterm and SGA birth did not reach statistical significance. A similar finding was observed in women exposed to PM10. Women exposed to the coarse fraction had increased odds for all the morbidity outcomes. Additionally, a woman who was exposed to PM2.5 had greater odds for low birth weight, very low birth weight and SGA.

We also performed sub group analysis to investigate the interaction across the three particulate pollutants. The interaction of all three particulates appeared to be the most severe with an increased odds for all the morbidities considered (low birth weight, very low birth weight, preterm, very preterm and SGA infants). The greatest risk was observed for very low birth with a 23% increased odds (OR=1.23, 95% CI=1.06-1.41). Furthermore, the interaction between PM10 and coarse fraction had increased odds for very low birth weight and preterm birth. All the other pair wise interactions did not elevate the risks for feto-infant morbidities (Table 3).

In order to evaluate the differences in the effects of particulate matter on feto-infant morbidities by race and ethnicity, we further categorized infants by race and exposure status.

Eight subgroups were thus constructed and analyzed in 2 ways. All exposed women were compared to white unexposed women, taken as the referent group: whites, not exposed (12.6%); whites exposed (39.0%); blacks, not exposed (4.6%); blacks, exposed (15.7%); Hispanics, not exposed (3.9%); Hispanics, exposed (12.9%); others, not exposed (2.1%) and others, exposed (9.2%). This is one method used in which the lowest risk group is used as the referent for all comparisons. It is sometimes used to avoid spurious within group effects that tend to blunt the magnitude of the variation across racial/ethnic groups (Salihu et al., 2007). Crude proportions of feto-infant morbidity among these subgroups are presented in Figure 2.

The other method we used to evaluate the differences in the effects of particulate matter on feto-infant morbidities by race and ethnicity is to compare the adjusted odds ratios of the exposed with the unexposed within each race, as shown in Table 5.

Both subgroup analysis of the association between particulate matter, race and feto-infant morbidity outcomes showed that black women who were exposed to any particulate matter had the greatest odds for all the morbidity outcomes when compared with white unexposed women or to their unexposed counterparts. Table 4 showed the odds among black women exposed to any particulate was most pronounced for very low birth weight, with an odds ratio of OR=3.32 (95% CI=2.56-4.30) closely followed by very preterm birth OR = 2.99 (95% CI=2.34-3.81). Similarly, in Table 5 very low birth weight was OR=1.52 and very preterm OR=1.27.

In Table 4, Black mothers, who were not exposed had greater odds for all morbidity outcomes (low birth weight, very low birth weight, preterm, very preterm and SGA)

compared to white women who were exposed (confidence intervals for all the morbidity outcomes did not intersect).

DISCUSSION

We set out this study à priori with two specific hypotheses regarding the association between particulate pollutants and feto-infant morbidity outcomes. The first hypothesis posits that PM2.5, PM10 and the coarse fraction (PM10-PM2.5) 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, preterm and very preterm). 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 pollution (Gouveia, et al., 2004; Ballester, et al., 2010; Hansen, et al., 2009; Ren, et al., 2010; Woodruff, et al., 1997; Suwanwaiphatthana, et al., 2010; Lee, et al., 2003). In agreement with our study, Wieslow et al found that pregnant women that were exposed to fine particles [PM2.5] had a significant reduction in birth weight than those who were not exposed (Jedrychowski, et al., 2004). In California, researchers found that mothers who lived in areas with the highest levels of PM2.5 during their pregnancy delivered slightly smaller infants than their counterparts who lived in areas with lower levels of PM2.5 exposure (Ritz).

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. This effect was also noted in another study by Bell (Bell, Ebisu, & Belanger, 2007) The adjusted odds ratios for black exposed women was most pronounced for very low birth weight When compared to the risk estimates for black-white disparity in non-exposed 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 (Woodruff, et al., 2003) and are suggestive that addressing environmental factors that elevate pollution exposures, especially in poor black neighborhoods merit consideration and infusion of resources in current efforts to narrow disparities in infant health and survival.

Strengths

One of the strengths of our study is our ample study size. We had data for births from 2000-2007 and air pollution records for the same period. We were also able to examine various feto- infant morbidities unlike other studies that focused only on low birth weight. We narrowed our research to only particulate matter so as to determine the most important feto-infant morbidities as they are affected by air pollutants. We were able to point out racio-ethnic disparities in the birth outcomes which may be due to the differences in exposure

based on residential neighborhoods, genetic differences or socio economic status. Other factors like SES and educational status have been studied by Woodruff et al and (Parker, et al., 2011; Woodruff, Parker, Kyle, & Schoendorf, 2003) and whether genetic differences also play a part in these disparities is still unknown. Interestingly, a recent study used a common approach in research design to examine the relationship between low birth weight and PM10 across several locations and variations in the PM10-low birth weight relationships still remained(Parker, et al., 2011)

In our analysis, we controlled for many potential confounders year of birth, education, maternal age, tobacco use, parity, marital status, adequacy of prenatal care, gender of the infant, anemia and maternal morbidities; gestational diabetes, diabetes mellitus, gestational hypertension, chronic hypertension, preeclampsia, eclampsia, placenta abruption, placenta previa, and renal diseases. We however cannot rule out residual confounding from pre pregnancy factors like maternal weight and nutrition.

Limitations

Exposure analysis posed a big challenge for us. 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). This however is not peculiar to our study; other studies (Ballester, et al., 2010; Hansen, et al., 2009; Lee, et al., 2003; Mannes et al., 2005; Salam, et al., 2005; van den Hooven et al., 2009; Woodruff, et al., 1997) all reported measurement error based on this. Using an ambient network of pollution monitors to assess individual exposure introduces measurement error because of the distance between the monitor and the subject, and the

individual modifiers of exposure such as air conditioning(Hansen, et al., 2009). There is a possibility of exposure misclassification because county wide monitors are used to estimate exposures at individual level. There are many factors that could play a role in individual exposures, distance from roads, traffic, place of work and period of time in current address. An ideal method would be to put pollution monitors in a cohort of pregnant mothers, but this would be very expensive. 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 research.

Studies have shown that 12-33% of women move address during pregnancy (Canfield, Ramadhani, Langlois, & Waller, 2006; Fell, Dodds, & King, 2004; Hansen, et al., 2009; Khoury et al., 1988; Shaw & Malcoe, 1992), (Shaw & Malcoe, 1992)) which could have lead to an underestimatation of the association between the three pollutants and feto-infant morbidity outcomes examined in this study. Our model also assumes the mothers spend most of their time at the same zip code, hence being exposed to the levels of the residential zip code monitors.

The study did not use Geographic Information System to identify the geographic coordinates of personal residential addresses and then link the addresses to the census tract dataset to obtain socioeconomic status information, this method would have helped to link SES data so as to more adequately asses its effect on racio-ethnic differences example using maternal education or other individual based indicators (Parker, et al., 2011), unfortunately, this was not done.

Information on various potential confounders such as drug and alcohol use, diet ((i.e., Pre pregnancy weight-for-height, gestational weight gain, and intake of nutrients), and occupational exposures, was unavailable. However, many of these factors are constant over time and will not be confounded with the week-to-week changes in ambient air pollution levels. Differences between these factors at an area level would have been partly controlled by using area-level SES.(Hansen, et al., 2009).

Also although maternal nutrition may be a determinant of birth weight in the developing world,(Kramer, 1987) did not observe any significant role of maternal nutrition in LBW in developed countries. Also, earlier literature has shown that maternal race/ethnicity (Cohen et al., 2001) maternal education (Kramer, Seguin, Lydon, & Goulet, 2000) and maternal age (Haiek & Lederman, 1989) are associated with maternal nutrition. Because we adjusted for maternal education and maternal age, we may have indirectly adjusted for the nutritional differences during pregnancy. Therefore, maternal nutrition is unlikely to confound the associations of air pollutants with birth weight in this study.

Furthermore, the simultaneous inclusion of exposures from all 3 trimesters or with the inclusion of both a particular trimester exposure and the 9-month exposure, made conclusions about the relative importance of exposure during a particular trimester in our study difficult. A major challenge in the field of air pollution and pregnancy outcomes has been identifying gestational windows of vulnerability(Darrow, et al., 2010) but various approaches are available that may help to identify vulnerable gestational windows.

CONCLUSION

In summary, we found an association between exposure to abnormal levels of air particulate pollutants (PM2.5, PM10, and PM10-PM2.5) and specific feto-infant morbidity outcomes. Analysis by race/ethnicity revealed that the white-black disparity in these outcomes was widened in the presence of these exposures.

Compared to the inhalation of cigarette smoke during active or passive smoking, the gases and particles in ambient air pollution are relatively diluted, resulting in relatively small risk increases for reproductive and children's health outcomes. For the most part, the increased risks for pregnancy outcomes in more polluted versus less polluted areas range from 10 to 30 percent for preterm birth and low birth weight, and between 5 and 20 percent for infant mortality. Yet when we consider the number of infants, children, and pregnant women affected by air pollution, and that these outcomes are not rare, even small relative increases in risk translate into large numbers making the total impact due to air pollution a major problem. Certain vulnerable population segments -- the poorest and most exposed -- may experience much higher risks(Ritz, 2008).

Recommendations for Future Research

Future research should be done to build up on our findings while improving on exposure analysis especially individually assessed exposure levels based on personal monitors and daily activities such as commute, residence time etc.

Determinants of the differences seen in vulnerability by various groups should also be well researched. Factors such as genetics, SES and age have been suggested but more research is required to test these hypotheses.

The results of our study may have implications not only for the health and development of children but also for adult health. Epidemiologic studies in children indicate that prenatal hazards that restrict fetal growth may be associated with small but measurable delays in motor and social development through childhood and reduced cognitive development (Hediger, Overpeck, Ruan, & Troendle, 2002), further research is also needed to establish biological mechanisms and possible trajectories.

Recommendations for Policy Makers

Currently, air pollution impacts on pregnant women and infants are not taken into consideration in setting environmental standards on a state or federal level. Since these populations are perhaps the most vulnerable to air pollution, taking these impacts into consideration would help strengthen air quality controls which will in the long run be beneficial to all population groups.

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TABLES

Table 1 Proportions (%) of selected maternal demographic characteristics between the two groups of women exposed to PM 2.5 and PM10 in Hillsborough county (2000-2007)*

Characteristics	Un exposed group N=79,855	Exposed group N=27,106	p-value
Advanced for age (≥ 35 years old)	13.56	13.69	0.62
Education (≥ 12 years)	80.31	80.02	<0.01
Smokers	6.76	6.54	0.02
Adequate prenatal care	66.19	66.67	0.16
Race			
White	54.35	50.77	<0.01
Black	19.86	20.47	
Hispanic	16.75	16.83	
Other	9.03	11.93	
Married	60.39	59.09	<0.01
Nulliparity	66.1	66.37	0.45
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

Table 2: Adjusted estimates for the association between exposure to particulate pollutants and feto-infant morbidity outcomes

	Exposed group	PM2.5	PM10	Coarse fraction
Low birth weight n=6,679	1.18 (1.11-1.25)	1.07 (1.01-1.12)	1.13 (1.07-1.19)	1.09 (1.03-1.15)
Very low birth weight n=999	1.27 (1.08-1.49)	1.14 (1.01-1.29)	1.17 (1.02-1.34)	1.22 (1.07-1.39)
Preterm n=9,459	1.12 (1.06-1.18)	1.03 (0.98-1.07)	1.11 (1.06-1.16)	1.05 (1.01-1.09)
Very preterm n=1,133	1.14 (0.98-1.31)	1.05 (0.93-1.18)	1.10 (0.96-1.25)	1.13 (1.01-1.27)
SGA n=8,733	1.04 (0.99-1.10)	1.06 (1.01-1.11)	1.02 (0.97-1.07)	1.07 (1.02-1.12)
Any morbidity n=24,661	1.08 (1.04-1.13)	1.05 (1.01-1.08)	1.07 (1.03-1.11)	1.06 (1.02-1.10)

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, preeclampsia, eclampsia, placenta abruption, placenta previa, and renal diseases

Table 3: Adjusted estimates for feto-infant morbidity outcomes in the presence of particulate pollutant interactions

	PM10 and PM2.5	PM10 and coarse fraction	PM2.5 and coarse fraction	Three-way interaction
Low birth weight	0.95 (0.81-1.11)	1.13 (1.04-1.21)	0.98 (0.68-1.43)	1.10 (1.04-1.16)
Very low birth weight	0.86 (0.57-1.31)	1.17 (0.96-1.42)	1.66 (0.78-3.52)	1.23 (1.06-1.41)
Preterm	0.88 (0.77-1.00)	1.10 (1.03-1.18)	0.71 (0.50-1.01)	1.08 (1.03-1.13)
Very preterm	0.91 (0.62-1.34)	1.06 (0.89-1.27)	1.62 (0.80-3.29)	1.13 (0.99-1.29)
SGA	1.01 (0.88-1.15)	1.06 (0.99-1.13)	1.28 (0.95-1.73)	1.06 (1.01-1.12)
Any morbidity	0.94 (0.85-1.04)	1.09 (1.03-1.14)	1.00 (0.78-1.27)	1.08 (1.04-1.12)

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, preeclampsia, eclampsia, placenta abruption, placenta previa, and renal diseases

Table 4: Adjusted odds ratio for the association between any exposure to particulate matter and feto-infant morbidity outcomes categorized by race/ethnicity (using white unexposed as referent group)

	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)

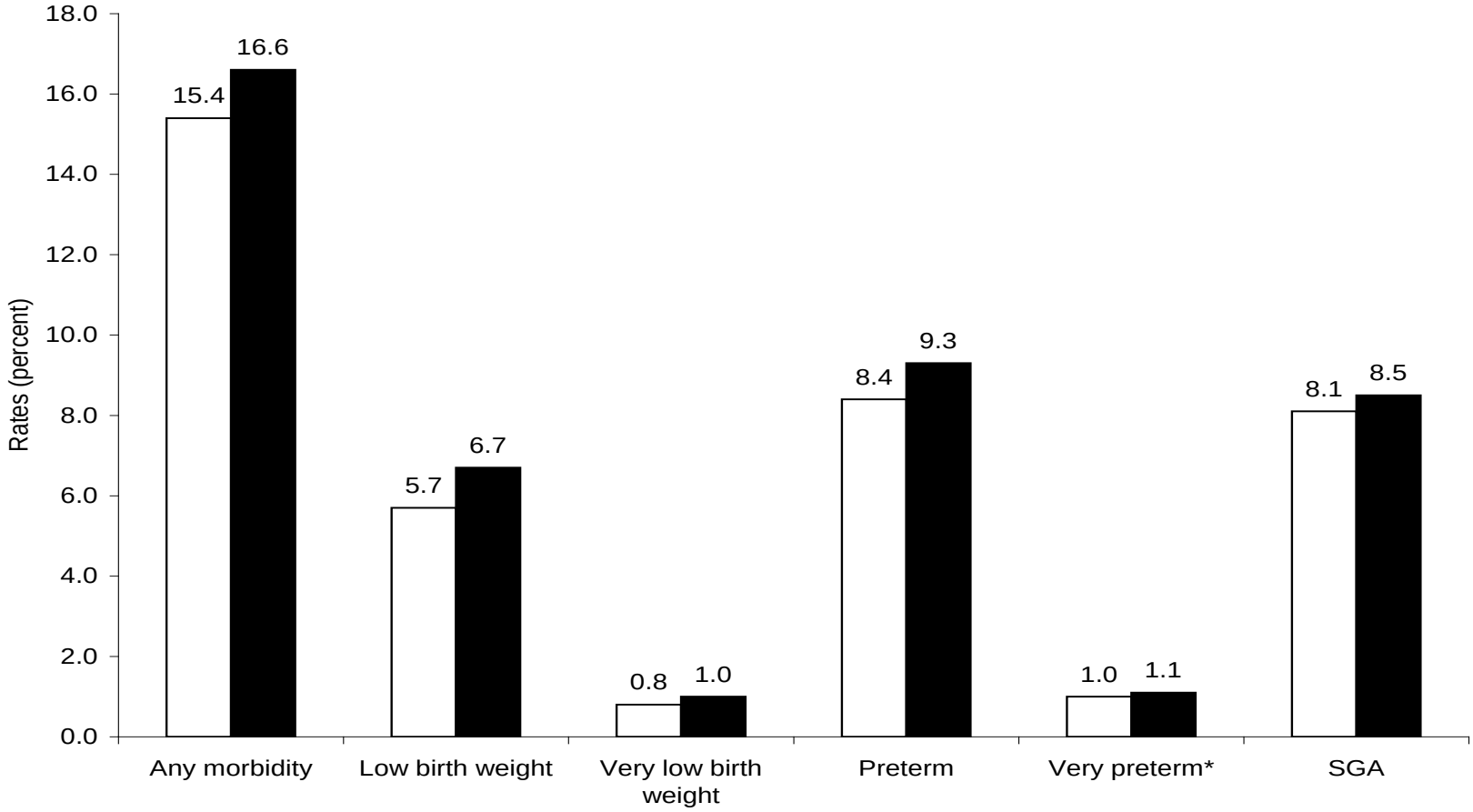
Adjusted odds ratios were generated after controlling for year of birth, education, maternal age, tobacco use, parity, marital status adequacy of prenatal care, sex of the infant, anemia, gestational diabetes, diabetes mellitus, gestational hypertension, chronic hypertension, preeclampsia, eclampsia,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

Table 5: Adjusted odds ratio for the association between any exposure to particulate matter and feto-infant morbidity outcomes categorized by race/ethnicity (using unexposed as referent groups for each race)

	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	1.00	1.24	1.00	1.21	1.00	1.08
Very low birth weight n=999	1.00	1.15	1.00	1.52	1.00	1.44	1.00	0.84
Preterm n=9,459	1.00	1.11	1.00	1.23	1.00	1.09	1.00	0.95
Very preterm n=1,133	1.00	1.14	1.00	1.27	1.00	1.14	1.00	0.77
SGA n=8,733	1.00	1.01	1.00	1.09	1.00	0.99	1.00	1.07
Any morbidity n=16,961	1.00	1.08	1.00	1.16	1.00	1.03	1.00	1.00

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, preeclampsia, eclampsia, placenta abruption, placenta previa, and renal diseases

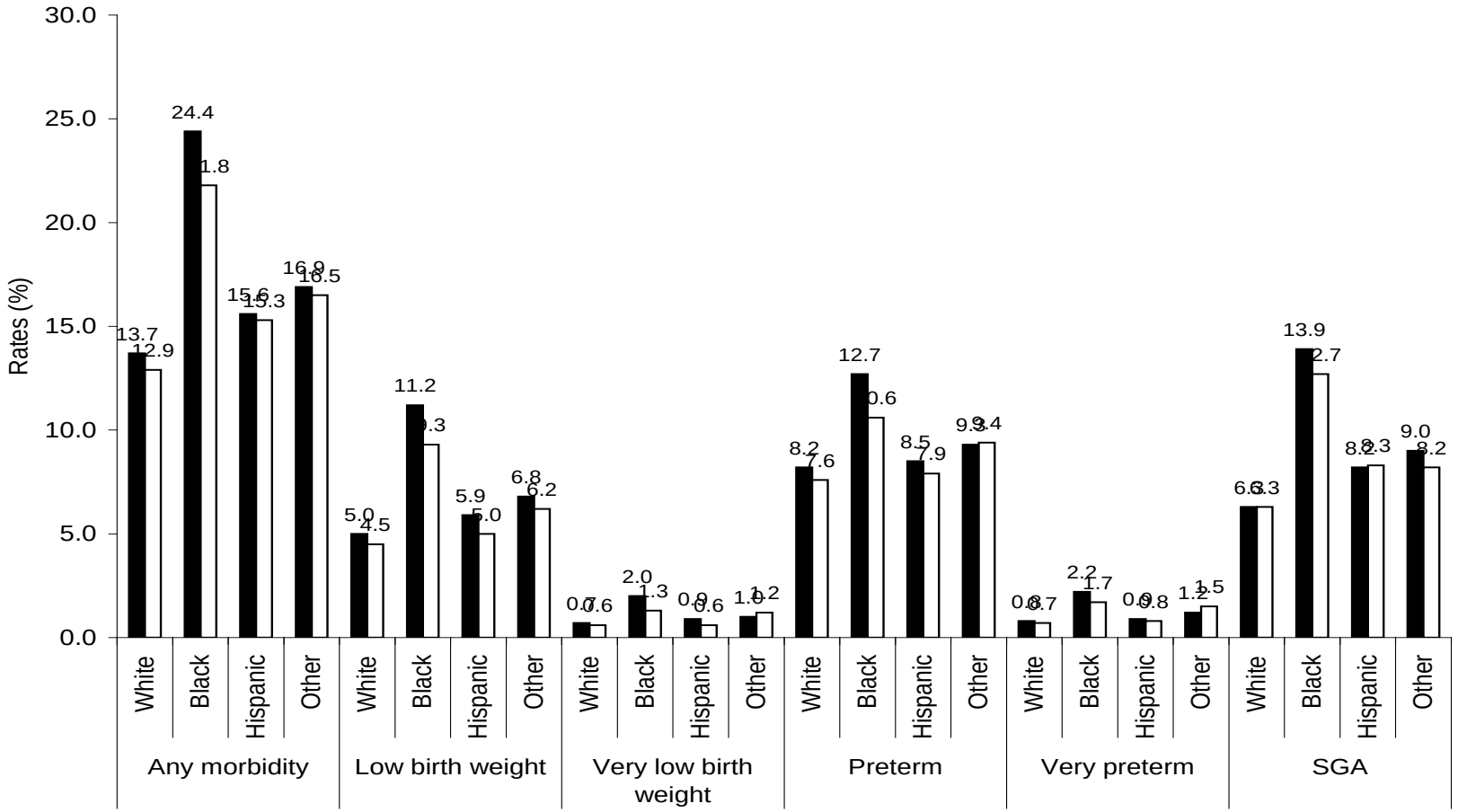
Figure 1: Crude proportions of feto-infant morbidity outcomes comparing women who were exposed and those who were not exposed to any particulate matter



□ =women who were not exposed to any particulate matte□ =women who were exposed to any particulate matter

*p-value =0.04; p-value for all other comparison <0.01

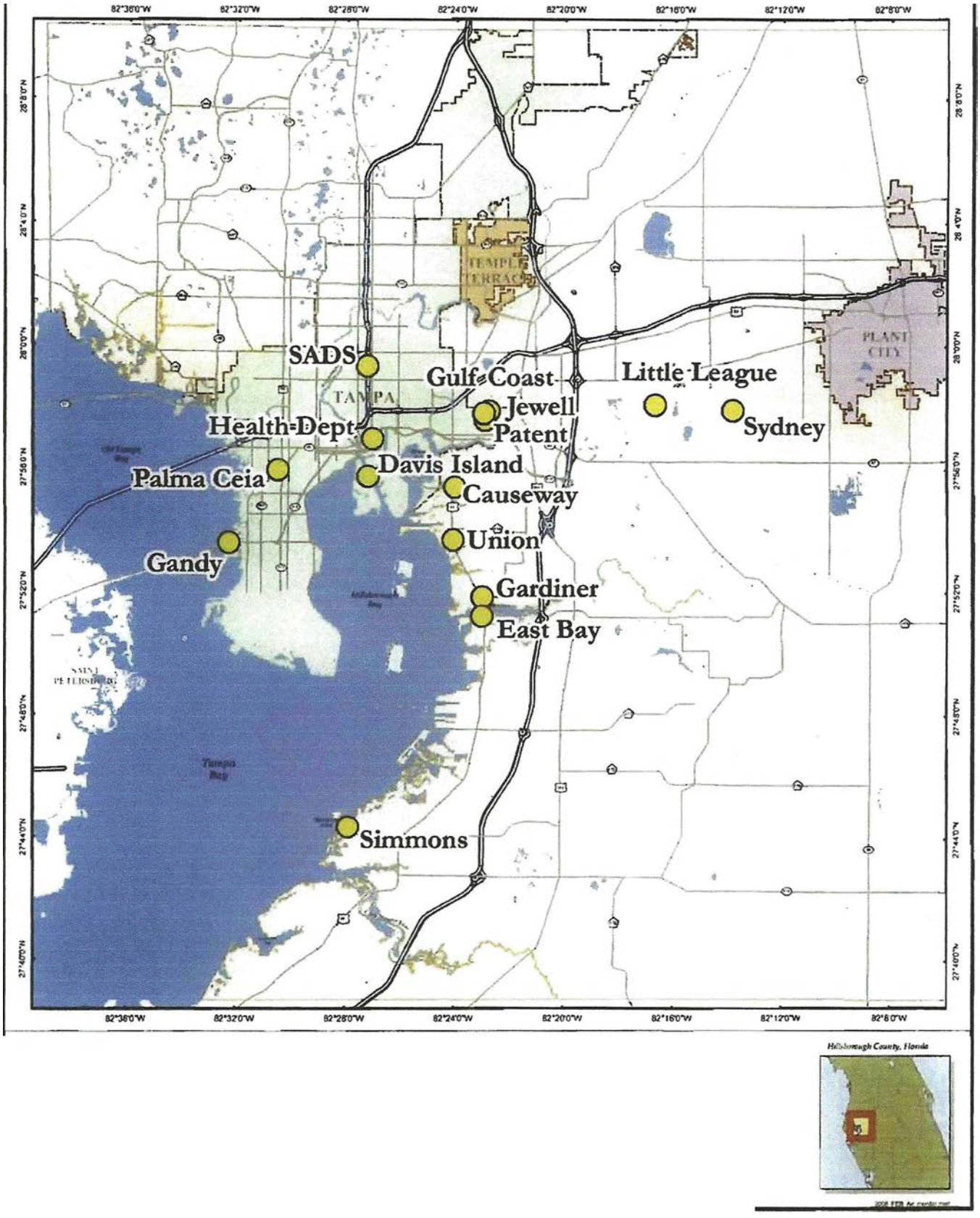
Figure 2: Crude proportions of feto-infant morbidity outcome comparing women who were exposed those unexposed to any particulate matter categorized by race/ethnicity



□=women who were not exposed to any particulate matter; ■=women who were exposed to any particulate matter

APPENDICES

ENVIRONMENTAL PROTECTION COMMISSION 2007 AIR MONITORING SITES



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Hills borough County, Florida Air Monitoring Stations

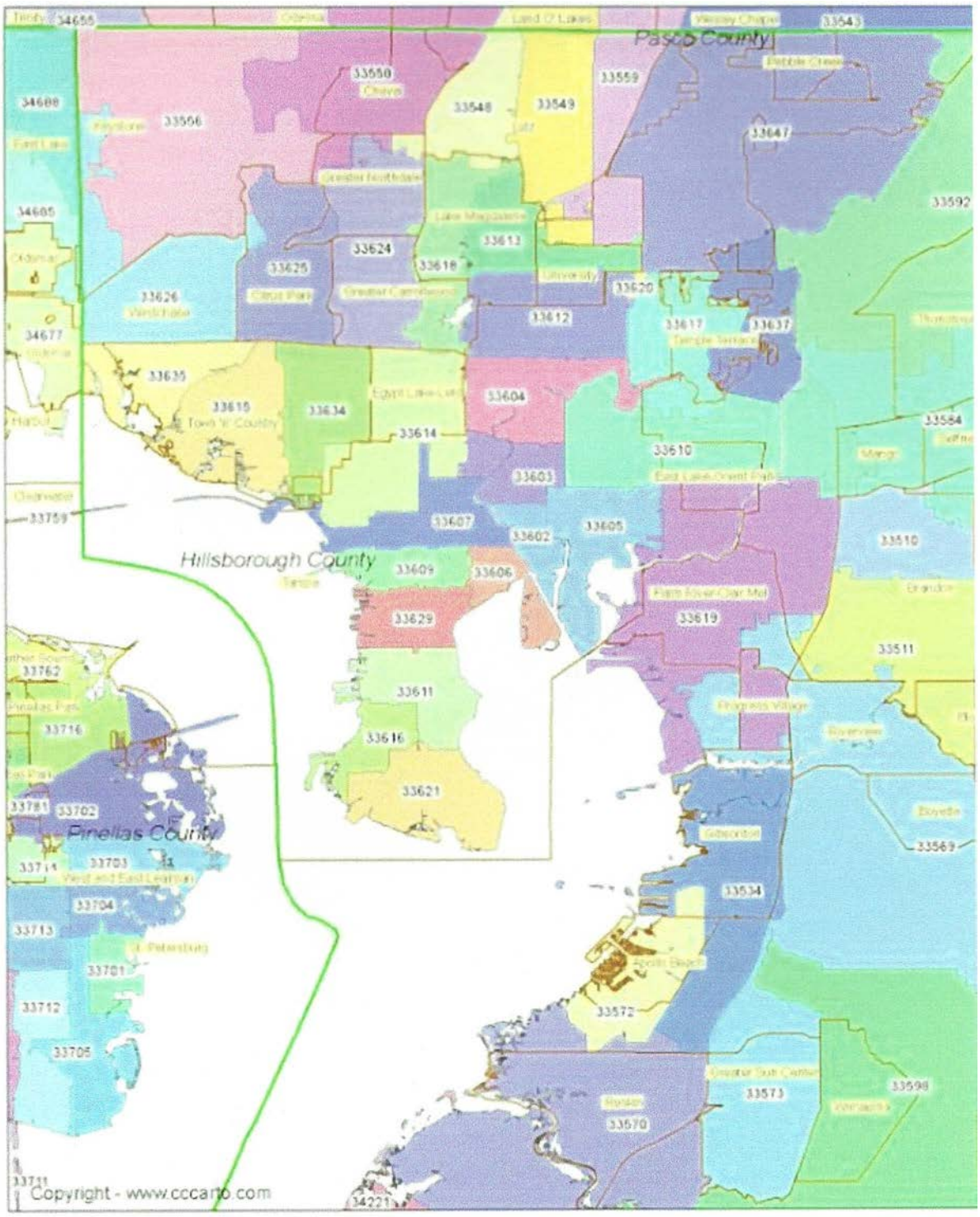
AIR MONITORING NETWORK DESCRIPTION.

EPC #	STATION NAME & ADDRESS	PARAMETERS	NAMS SLAMS SPM
1	Health Dept. 110S E. Kennedy Blvd.	PMI0*	SLAMS
15	Palma Ceia 3910 Morrison Ave.	PM2.5*/ CONTINUOUS PM2.S	SLAMS/ SPM
63	Davis Island 15S Columbia Dr.	03,S02,CONTINUOUS PMI0/WIND D&S	SLAMS/ SPM
69	Seminole Adult Day School, 4701 Central Ave.	CO/CONTINUOUS PMI0	SLAMS/SPM
93	Union 514 U.S. Highway 41 CWU	PMI0*	SLAMS
113	Simmons Park 2401 19th Ave., NW	03,S02/N02,WIND D&S	SLAMS/SPM
117	Gardinier U.S. Highway 41/Riverview Dr.	CONTINUOUS PMI0	SPM
124	Causeway 5012 Causeway Blvd.	S02,CONTINUOUS PM10	SLAMS
129	Brandon Little League 2929 Kingsway Rd.	PMI0	SPM
137	Gandy	03,N02/WIND D&S/ CONTINUOUS PM2.S/PM10/	SLAMS/SPM/

	S121 Gandy Blvd.	TOXICS (VOC'S, CARBONYLS, & METALS)	SPM/SPM
139	Gulf Coast Lead, CSX Modual Yard 1700 N. 66th St.	Lead*/TSP*	SLAMS/SPM
144	Patent 6811 E. 14th Ave.	Lead/TSP	SPM/SPM
146	Jewell 64th St. /Jewel Ave.	Lead/TSP	SPM/SPM
E BAY	East Bay, Eastbay Shopping Center U.S. Highway 41	S02/WIND D&S	SLAMS/SPM
SYD	Sydney 1167 N Dover Rd	03,N02,WIND D&S,CONTINUOUS PM2.5/PM2.S/PM10*/ PM2.S SPECIATION! TOXICS (VOC'S, CARBONYLS, & METALS)	SPM/SLAMS/ SLAMS
* Collocated sites			

Tampa Zip Codes and Map

Florida Zip Code Database for Tampa, Plant City, Temple Terrace Zip Codes.



http://www.cccarto.com/fl/hillsborough_zipcodes/

EMORY

UNIVERSIT'{

Nafisa Ghaji

Emory University School of Public Health

RE: Determination: No IRB Review Required PI: Nafisa Ghaji

Thank you for requesting a determination from our office about the above-referenced project.

Based on our review of the materials you provided, we have determined that it does not require IRB because it does not meet the definition(s) of "research" involving "human subjects" or the definition of "clinical investigation" as set forth in Emory policies and procedures and federal rules, if applicable.

Specifically, in this project, you will be utilizing a de-identified data set obtained from the University of South Florida. You had originally worked on this study as part of a summer practicum; however, you will have no link to any identifiers. You will be utilizing the analysis of this data as part of your thesis.

This determination could be affected by substantive changes in the study design, subject populations, or identifiability of data. If the project changes in any substantive way, please contact our office for clarification.

Emily Sanders, BS

Education and QA Analyst Assistant

This letter has been digitally signed

Emory University

1599 Clifton Road, 5th Floor - Atlanta, Georgia 30322

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UNIVERSITY OF SOUTH FLORIDA

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Dr. Hamisu Salihu

Lawton and Rhea Chiles Center for Healthy Mothers and Babies 3111 E. Fletcher Avenue

Tampa, FL 33612

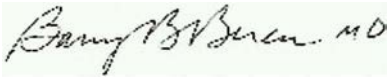
Activity Title: Examining environmental and non-environmental factors in connection with birth outcomes in the State of Florida

I have reviewed the information you provided regarding the above referenced project and have determined the activities do not meet the USF definition of human subjects research activities; therefore, IRB approval is not required. If, in the future, you change this activity such that it becomes human subjects research activities, prior IRB approval is required. If you wish to obtain a determination about whether the activity, with the proposed changes, will be human research activities, please contact the IRB Office for further guidance.

All research activities, regardless of the level of IRB oversight, must be conducted in a manner that is consistent with the ethical principles of your profession and the ethical guidelines for the protection of human subjects. As principal investigator, it is your responsibility to ensure subjects' rights and welfare are protected during the execution of this project

Also, please note that there may be requirements under the HIP AA Privacy Rule that apply to the information/data you will use in your activities. For further information about any existing HIPAA requirements for this project, please contact Vinita Witanachchi, J.D., HIPAA Program Coordinator, at 813-974-5478.

We appreciate your dedication to the ethical conduct of human subject research at the University of South Florida and your continued commitment to human research protections. If you have any questions regarding this matter, please call 813-974-5638.



Barry Bercu, MD, Chairperson USF Institutional Review Board