The effects of exposure to particulate matter and neighbourhood deprivation on gestational hypertension

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Summary

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Gestational hypertension, pre-eclampsia and eclampsia are conditions that affect the health of both mothers and infants during and after pregnancy. Recent research indicates the importance of considering environmental, social and individual contributors to poor pregnancy outcomes. Our research examined particulate matter (PM) concentrations as one measure of environmental exposure and neighbourhood quality as one measure of the social environment. We used these measures, as well as maternal characteristics, to predict the risk of gestational hypertension (including pre-eclampsia and eclampsia). North Carolina Detailed Birth Record data for 2000–2003 were obtained and geocoded for all singleton births. Levels of PM_{10} and $PM_{2.5}$ were determined using air quality data from the US Environmental Protection Agency. Information on a woman's residential neighbourhood was determined from 2000 Census data. Modified Poisson regression models clustered by tract were used to examine the associations between PM levels, neighbourhood deprivation and maternal characteristics with gestational hypertension. Analysis was restricted to women residing within 20 km of a PM monitor. Both PM_{10} and $PM_{2.5}$ were associated with gestational hypertension; the risk ratios for an interquartile range (IQR) increase in exposure were 1.07 [95% confidence interval (CI) 1.04, 1.11] for PM_{10} (IQR: 3.92 μg/m³) and 1.11 [95% CI 1.08, 1.15] for $PM_{2.5}$ (IQR: 2.24 μg/m³). Living in a neighbourhood with increased levels of deprivation was also associated with gestational hypertension. Any smoking during pregnancy, younger age and higher level of education were inversely associated with risk of gestational hypertension. Compared with non-Hispanic White women, non-Hispanic Black women were at higher risk of gestational hypertension, whereas Hispanic women were at lower risk. Increased levels of PM and neighbourhood deprivation, as well as certain individual characteristics, were associated with higher risk of gestational hypertension.

Keywords: *air pollution, social deprivation, gestational hypertension, prenatal environment, particulates.*

Introduction

Hypertensive disorders during pregnancy [gestational hypertension, pre-eclampsia and eclampsia, hereafter referred to as gestational hypertension (GH)] are detrimental to short-term and long-term health of both the mother and child. Pre-eclampsia/eclampsia is one of the leading causes of maternal and perinatal death.[1–3]

Infants born to mothers with GH are more likely to be small for gestational age, preterm and have low birthweight.[4,5] They have also been shown to have higher rates of admission to neonatal intensive care units[6] and increased risk of hospitalisation for infectious, nervous, respiratory, endocrine, metabolic and blood/blood-forming organ diseases.[7] Children of mothers with GH are more likely to have elevated

blood pressure during adolescence,[8,9] and an increased risk of stroke during adulthood.[5] Having GH during a pregnancy has also been associated with increased risk of cardiovascular disease, stroke and Type II diabetes later in life.[1,10–12]

In the US, the rates of GH have been rising in recent years.[13] In 2003–2004, the incidence rates were 30.6/1000 for gestational hypertension (without pre-eclampsia) and 29.4/1000 for pre-eclampsia.[13] Prior studies have shown that previous pregnancy[14–16] and maternal race/ethnicity[17] affect the rates of GH. In addition, a few studies have examined socio-economic status of the maternal residential neighbourhood, but the results have been inconsistent.[18–20]

Auchincloss *et al.* examined the association between mean levels of particulate matter (PM) with mean aerodynamic diameters $\leq 2.5\,\mu m$ ($PM_{2.5}$) over 30- and 60-day intervals and general blood pressure levels.[21] The study demonstrated a change in hypertension measures with increases in $PM_{2.5}$. It is plausible that if long-term $PM_{2.5}$ exposure levels increase the risk of higher blood pressure levels, women exposed during their pregnancy to high $PM_{2.5}$ exposure levels may be at increased risk of GH. The relationship between GH and air pollution, specifically PM, has been explored in a limited number of studies. A recent study reported a positive association between $PM_{2.5}$ and pre-eclampsia.[22] A prospective cohort study in the Netherlands also reported an increased risk of GH with higher PM_{10} concentrations.[23]

A recent article by Miranda *et al.* described pregnancy outcomes as the result of the forces of three sides of an integrated triangle, with environmental factors, social factors and host factors each forming one side of the triangle.[24] Our paper utilises that same conceptual framework. We examine the associations between an environmental factor (PM), a social factor (neighbourhood deprivation) and host factors (such as age and parity) with GH. We used data on women living in North Carolina and giving birth to a singleton infant between 2000 and 2003. The results of this study can be used to elucidate the social and environmental factors that may contribute to GH.

Methods

Birth data

North Carolina Detailed Birth Record data were obtained from the North Carolina State Center for Health Statistics. The data included all registered births occurring in North Carolina between 1 January 2000 and 31 December 2003 ($n = 467\,780$). Data from birth records were also used to determine whether a woman had GH, pre-eclampsia and/or eclampsia during her pregnancy. These were combined to form one variable representing GH, similar to previous research.[17] Information on maternal age, education, race/ethnicity, marital status, parity and smoking during pregnancy was also obtained from birth records.

In addition, the mother's residential address at birth was geocoded at the street level using ArcGIS 9.3 software (ESRI, Redlands, CA, USA) with a geocoding success rate of about 91% for women living in a county with a PM monitor (86% overall success rate). Women whose addresses could not be geocoded were excluded from further analysis ($n = 67\,367$). All aspects of the study were conducted in accordance with a human subjects research protocol approved by the Institutional Review Board.

Air data

Data on PM_{10} and $PM_{2.5}$ levels were downloaded from the US Environmental Protection Agency's Air Quality System (AQS) for 1999–2003.[25] Daily 24-h average concentrations were obtained every 1, 3 or 6 days from active monitors across North Carolina. During the study period there were 27–37 active PM_{10} monitors and 37–41 active $PM_{2.5}$ monitors (Figure 1).

Residential addresses were linked to the closest active monitors. Birth date and gestational age were used to determine the start and end dates of the full gestational period for each infant, allowing PM exposure to be calculated as the 24-h values averaged over the full gestational period. In order to ensure that PM exposure could be sufficiently characterised, births were excluded if data were available for <75% of the weeks of the entire gestational period. Births were also excluded if PM data were not available for two or more consecutive weeks of gestation. For births with data available for at least 75% of the gestational period and no missing data in consecutive weeks, any weeks of gestation with missing PM data were imputed using the average of the PM monitoring value of the previous and following weeks. Additionally, women were excluded from the study if they lived more than 20 km from an AQS monitor. Overall, the criteria based on sufficient monitoring data and proximity to
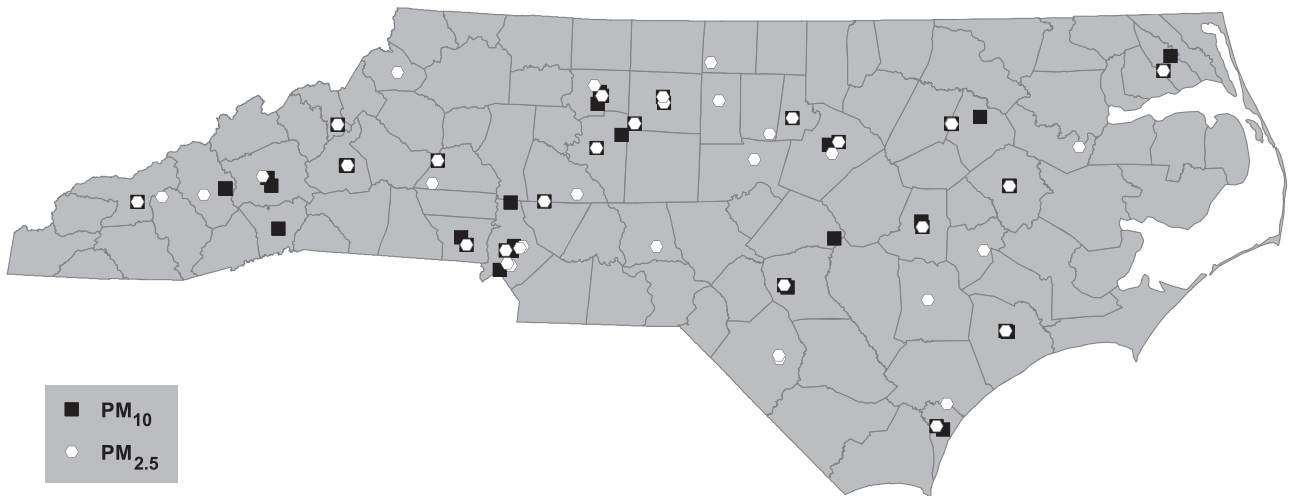
Figure 1. Locations of active PM_{10} and $PM_{2.5}$ monitors during the study period.

the monitor resulted in the exclusion of 146 907 women (approximately 37% of geocoded women).

Neighbourhood data

Tract-level data from the 2000 Census were utilised to create the Neighbourhood Deprivation Index (NDI).[26] This index has been used previously as a measure of neighbourhood poverty in studies of birth outcomes.[26–29] The NDI was created using the first factor loadings from a principal components analysis of the following census variables: per cent of households in poverty, per cent of female-headed households with dependents, per cent with household income <$30 000, per cent of households on public assistance, per cent of males in management/professional occupation, per cent living in crowded housing, per cent unemployed and per cent without a high school education. Quartiles were created for the NDI based on the distribution among census tracts captured by the 20-km buffers around PM_{10} and $PM_{2.5}$ monitors. Women were assigned the NDI quartile for their census tract. Quartiles were created at the census tract level, as opposed to quartiles based on the population distribution, to avoid weighting the quartiles of NDI by population of the census tracts.

Maternal characteristics

Information on maternal characteristics was obtained from the birth records. The variable representing parity was dichotomised based on whether the infant was a first or higher-order birth. Smoking during pregnancy and marital status were also dichotomous variables. Maternal race was categorised as non-Hispanic White (NHW), non-Hispanic Black (NHB) and Hispanic (because of small numbers, women of other races/ethnicities were excluded from the study, $n = 10 437$). The following categories were used for maternal education: <9th grade, attended some high school, completed high school, attended some college and completed college or a higher degree. Maternal age at delivery was categorised in 5-year increments for women between 15 and 44, with two additional categories for <15 years old and >44 years old.

Multi-fetal births ($n = 8441$) and infants with congenital abnormalities ($n = 1711$) were excluded from the analysis. Women who had more than three previous births were also excluded ($n = 6571$). Infants with a birthweight <400 g ($n = 235$) or a gestational age <24 or >42 weeks ($n = 461$) were removed from the study. Additionally, women were excluded if data on key variables were missing ($n = 622$) or they reported having chronic hypertension ($n = 2253$). Figure 2 depicts the process of excluding individuals from the study population. Under these restrictions, a total of 222 775 births were available for analysis.

Statistical analysis

We used modified Poisson regression models with robust standard errors clustered by census tract to assess the associations between GH and PM_{10}, $PM_{2.5}$, maternal characteristics and neighbourhood
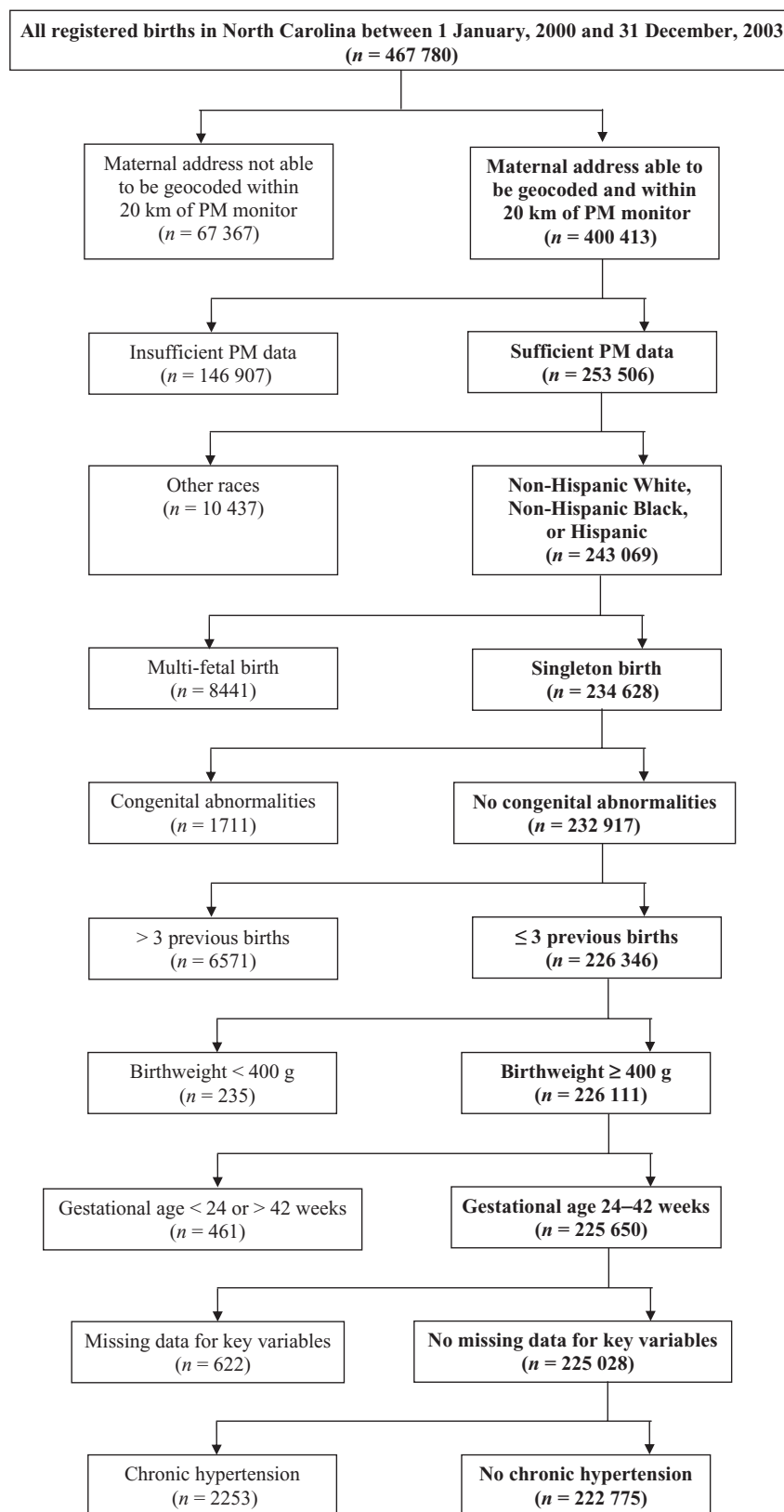
Figure 2. Flowchart of inclusion criteria for the study population. PM, particulate matter.

deprivation. PM_{10} and $PM_{2.5}$ were run as continuous variables in separate models with the same covariates (NDI quartiles and maternal characteristics as described above) included in both models. A linear specification was used for PM_{10} and $PM_{2.5}$ after it was determined that alternate specifications, such as quadratic, did not improve the fit of the model. Clustering by census tract was performed to account for the inclusion of a tract-conditional variable, NDI, in the models. All models were run using SAS 9.2 (SAS Institute Inc., Cary, NC, USA). Additionally, models including interactions of PM_{10} and $PM_{2.5}$ with level of neighbourhood deprivation were included to observe how the associations varied by level of deprivation. This was done by creating an interaction variable that combined PM concentration with a dichotomous variable for neighbourhood deprivation. The interaction was assessed using a likelihood ratio test and results for each deprivation category are presented.

The models were restricted to women living within 20 km of a monitor. To check the sensitivity of our findings to the distance of maternal residence to the PM monitors, models were also run restricting to women living within 10 km of a monitor and women living within 5 km of a monitor. The results did not substantially differ across the buffer sizes. Therefore, we only present results for the women living within 20 km of a monitor.

Results

The total study population after the exclusion criteria were applied was 222 775 women. Of the women in the study population, 5.4% had GH ($n = 12\,085$). Women with GH had similar mean concentrations of PM_{10} and $PM_{2.5}$ exposure during pregnancy compared with normotensive women. The mean PM_{10} levels during pregnancy were $22.4\,\mu g/m^3$ among women with GH and $22.2\,\mu g/m^3$ among women without GH. The respective mean $PM_{2.5}$ levels were $14.7\,\mu g/m^3$ and $14.5\,\mu g/m^3$. A higher proportion of normotensive women were Hispanic (13.4% vs. 9.2%) and a higher proportion had previously given birth (56.9% vs. 38.3%). Maternal age and marital status were similar between the two groups (Table 1).

Results of the regression analysis are shown in Table 2. Positive associations were detected between $PM_{2.5}$ and GH; the risk ratio (RR) for an increase in $PM_{2.5}$ equivalent to the sample's interquartile range (IQR) ($2.24\,\mu g/m^3$) was 1.11 [95% confidence interval

(CI) 1.08, 1.15]. Similarly, there was an association between PM_{10} and GH [RR 1.07 [95% CI 1.04, 1.11] per increase in PM_{10} equivalent to the IQR ($3.92\,\mu g/m^3$)].

Compared with women aged 25–29, women aged 35 years and older were more likely to present with GH, and women under 25 years old were less likely to present with GH. Although no association was seen for most levels of education, women who completed at least a college degree had a lower risk of having GH compared with women completing a high school degree. GH was also inversely associated with maternal smoking, and the risk of GH was elevated among primiparous women. Compared with NHW women, NHB women had a higher risk of GH, whereas Hispanic women had a decreased risk. Marital status showed no association with GH.

The NDI was calculated using principal components analysis and was found to explain 60% of the variance of the census tract variables (alpha = 0.90). Increased risk of GH was associated with higher NDI values; that is, increased neighbourhood deprivation above the first quartile was positively associated with GH (Table 2). We further examined the role of NDI by assessing how deprivation affects the association between GH and concentrations of PM_{10} and $PM_{2.5}$. Because of the similarity of the associations between the second, third and fourth quartiles for NDI, these were collapsed into one category. No interaction was present between NDI categories and $PM_{2.5}$ (P-value for likelihood ratio test 0.24). Using interaction models, the adjusted RRs for each IQR increase in $PM_{2.5}$ were 1.08 [95% CI 1.02, 1.14] for women residing in areas with the lowest deprivation and 1.12 [95% CI 1.08, 1.17] for those in the combined upper 75% of the NDI. A more pronounced difference across NDI levels was observed for PM_{10} and GH (lowest neighbourhood deprivation: RR 1.02 [95% CI 0.98, 1.07] per IQR increase; upper 75% of neighbourhood deprivation: RR 1.10 [95% CI 1.06, 1.14] per IQR increase; P-value for likelihood ratio test <0.05). Despite the changes in the association, PM levels were similar among all categories of the NDI with PM_{10} concentrations in the lowest quartile of deprivation being only slightly higher than the other quartiles (Table 3).

Discussion

Following the conceptual framework laid out in previous work on the multiple factors affecting pregnancy outcomes,[24] this study examined the association of GH

Table 1. Demographic characteristics of women with exposure information concerning PM_{10} and/or $PM_{2.5}$ by gestational hypertension status

	Gestational hypertension (n = 12 085)	No gestational hypertension (n = 210 690)	Total (n = 222 775)
PM_{10} during pregnancy (μg/m³)			
Mean (SD)	22.4 (3.6)	22.2 (3.6)	22.2 (3.6)
Median	22.0	21.9	21.9
Interquartile range	20.1, 24.1	20.0, 23.9	20.0, 23.9
$PM_{2.5}$ during pregnancy (μg/m³)			
Mean (SD)	14.7 (1.7)	14.5 (1.7)	14.5 (1.7)
Median	14.7	14.5	14.5
Interquartile range	13.6, 15.8	13.5, 15.7	13.5, 15.7
	n (%)	n (%)	n (%)
Neighbourhood			
Deprivation Index			
1st quartile	3 290 (27.2)	65 769 (31.2)	69 059 (31.0)
2nd quartile	3 352 (27.7)	54 548 (25.9)	57 900 (26.0)
3rd quartile	2 943 (24.4)	47 024 (22.3)	49 967 (22.4)
4th quartile	2 500 (20.7)	43 349 (20.6)	45 849 (20.6)
Race			
Non-Hispanic White	7 468 (61.8)	129 670 (61.5)	137 138 (61.6)
Non-Hispanic Black	3 506 (29.0)	52 769 (25.0)	56 275 (25.3)
Hispanic	1 111 (9.2)	28 251 (13.4)	29 362 (13.2)
Previous birth			
No	7 457 (61.7)	90 810 (43.1)	98 267 (44.1)
Yes	4 628 (38.3)	119 880 (56.9)	124 508 (55.9)
Smoked during pregnancy			
No	10 969 (90.8)	187 411 (89.0)	198 380 (89.0)
Yes	1 116 (9.2)	23 279 (11.0)	24 395 (11.0)
Maternal age (years)			
<15	40 (0.3)	464 (0.2)	504 (0.2)
15–19	1 474 (12.2)	22 658 (10.8)	24 132 (10.8)
20–24	2 987 (24.7)	55 517 (26.4)	58 504 (26.3)
25–29	3 293 (27.3)	56 189 (26.7)	59 482 (26.7)
30–34	2 730 (22.6)	51 163 (24.3)	53 893 (24.2)
35–39	1 286 (10.6)	21 109 (10.0)	22 395 (10.1)
40–44	264 (2.2)	3 480 (1.7)	3 744 (1.7)
≥45	11 (0.1)	108 (0.1)	119 (0.1)
Maternal education completed			
<9th grade	502 (4.2)	12 060 (5.7)	12 562 (5.6)
Some high school	1 579 (13.1)	29 221 (13.9)	30 800 (13.8)
High school degree	3 490 (28.9)	57 301 (27.2)	60 791 (27.3)
Some college	3 052 (25.3)	45 978 (21.8)	49 030 (22.0)
College degree	3 462 (28.6)	66 130 (31.4)	69 592 (31.2)
Marital status			
Married	8 020 (66.4)	142 634 (67.7)	150 654 (67.6)
Not married	4 065 (33.6)	68 056 (32.3)	72 121 (32.4)

with PM exposure during pregnancy, neighbourhood deprivation and maternal host factors.

We found positive associations between $PM_{2.5}$ and PM_{10} concentrations and the risk of GH. This is especially noteworthy given the relatively low $PM_{2.5}$ and PM_{10} concentrations found in North Carolina compared with other regions of the US. Our findings are similar to a study by Wu *et al.*, which reported a positive association between $PM_{2.5}$ and pre-eclampsia among women living in southern California.[22]

Table 2. Risk ratios [95% confidence intervals] for the associations between particulate matter (PM) exposure during pregnancy, maternal and neighbourhood characteristics and gestational hypertension[a]

	Risk ratio [95% CI]	
	Model including $PM_{2.5}$ (n = 214 325)	Model including PM_{10} (n = 159 358)
PM per interquartile range increase[b]	1.11 [1.08, 1.15]	1.07 [1.04, 1.11]
Neighbourhood Deprivation Index		
1st quartile	1.00 Reference	1.00 Reference
2nd quartile	1.23 [1.15, 1.32]	1.25 [1.16, 1.36]
3rd quartile	1.30 [1.20, 1.40]	1.34 [1.23, 1.46]
4th quartile	1.24 [1.14, 1.35]	1.27 [1.16, 1.39]
Maternal age (years)		
<15	0.79 [0.57, 1.10]	0.82 [0.56, 1.20]
15–19	0.72 [0.67, 0.78]	0.70 [0.64, 0.77]
20–24	0.78 [0.74, 0.83]	0.77 [0.72, 0.82]
25–29	1.00 Reference	1.00 Reference
30–34	1.05 [1.00, 1.11]	1.06 [1.00, 1.13]
35–39	1.29 [1.21, 1.37]	1.29 [1.20, 1.39]
40–44	1.56 [1.37, 1.76]	1.59 [1.37, 1.85]
≥45	1.95 [1.08, 3.52]	1.69 [0.67, 3.68]
Maternal education		
<9th grade	0.96 [0.87, 1.07]	0.96 [0.86, 1.09]
Completed some high school	0.99 [0.93, 1.06]	1.01 [0.93, 1.09]
Completed high school	1.00 Reference	1.00 Reference
Completed some college	0.98 [0.93, 1.03]	0.97 [0.91, 1.03]
Completed college	0.71 [0.67, 0.76]	0.70 [0.65, 0.75]
Smoked during pregnancy		
No	1.00 Reference	1.00 Reference
Yes	0.80 [0.74, 0.85]	0.84 [0.78, 0.91]
Previous birth		
Yes	1.00 Reference	1.00 Reference
No	2.28 [2.19, 2.37]	2.28 [2.18, 2.39]
Race		
Non-Hispanic White	1.00 Reference	1.00 Reference
Non-Hispanic Black	1.15 [1.10, 1.21]	1.14 [1.08, 1.21]
Hispanic	0.67 [0.61, 0.73]	0.67 [0.61, 0.73]
Marital status		
Married	1.00 Reference	1.00 Reference
Not married	0.97 [0.92, 1.03]	0.98 [0.92, 1.05]

[a]Models included all variables listed above.
[b]Interquartile range is 3.92 μg/m³ for PM_{10} and 2.24 μg/m³ for $PM_{2.5}$.

Table 3. Distribution of PM_{10} and $PM_{2.5}$ by quartile of Neighbourhood Deprivation Index

	Neighbourhood Deprivation Index			
	Quartile 1	Quartile 2	Quartile 3	Quartile 4
PM_{10} during pregnancy (μg/m³)				
n	53 726	40 191	33 154	32 287
Mean (SD)	23.00 (3.74)	21.63 (3.27)	21.75 (3.73)	21.88 (3.59)
Median	22.39	21.50	21.58	21.67
Interquartile range	20.80, 24.65	19.69, 23.26	19.48, 23.74	19.66, 23.79
$PM_{2.5}$ during pregnancy (μg/m³)				
n	65 912	55 454	48 356	44 605
Mean (SD)	14.64 (1.59)	14.47 (1.73)	14.40 (1.83)	14.44 (1.83)
Median	14.59	14.49	14.46	14.49
Interquartile range	13.70, 15.62	13.41, 15.65	13.23, 15.67	13.19, 15.74

Additionally, among women in the Netherlands, higher PM_{10} concentrations were associated with increased risk of GH and increased systolic blood pressure measurements in the second and third trimesters.[23] Further work is needed to determine if the association persists in other study populations and to describe the potential mechanism(s) associating PM with GH.

We also observed a positive relationship between neighbourhood deprivation and GH. A few studies have examined socio-economic status of the maternal residential neighbourhood, but the results have been inconsistent. Agyemang *et al.* reported no association between neighbourhood median income or unemployment levels and GH.[18] Another study examining the difference between two areas of Oslo, Norway reported higher rates of pre-eclampsia in areas with lower incomes.[19] Conversely, a study conducted in Sweden reported higher rates of pre-eclampsia in high-income areas.[20] Studies on chronic hypertension have reported positive associations with neighbourhood deprivation.[18,30] In addition, in our study the association between PM_{10} levels and GH varied within categories of neighbourhood deprivation. The association between PM_{10} concentration and GH was greater among those living in neighbourhoods with the highest deprivation compared with those neighbourhoods that had lower levels of neighbourhood deprivation. However, no difference was observed between the two deprivation categories for the association between $PM_{2.5}$ concentration and GH. Future studies of PM and neighbourhood deprivation in relation to GH may help elucidate whether social factors, such as neighbourhood deprivation, influence the association between PM and GH.

Associations were detected with other covariates included in the models. Older maternal age, having no previous births and neighbourhood deprivation were positively associated with GH. Smoking during pregnancy, younger age and high levels of education were inversely associated with GH. Compared with NHW women, the risk of having GH was lower among Hispanic women, but higher among NHB women. Some of these associations have been previously demonstrated. Prior work examining maternal education found that GH was inversely associated with education level[31] and that GH varied by race, with African Americans having higher odds and Hispanics having lower odds.[17,32] Another study of women giving birth in North Carolina reported a positive association between maternal age and hypertension, although this association had a strong interaction with race/ethnicity.[17] Wallis *et al.* also reported a slight positive association between GH and older maternal age.[13] However, these researchers also reported higher risk of GH at younger ages, which was not observed in our study where younger age appeared to reduce the odds of GH. The inverse relationship reported in this study between smoking during pregnancy and GH has been reported in multiple studies, although the mechanism for this association is unknown.[33–35] This study also detected higher risk of GH among primiparous women. Prior research supports the associations between GH and previous pregnancies.[14–16]

This study is not without limitations. PM exposure was averaged over the whole pregnancy regardless of when the GH began. Future studies that match the time of diagnosis for GH during pregnancy with temporal data on air pollution may elucidate this association further. In addition, women were assigned PM exposure levels based on their location of residence; however, no direct sampling was performed to gauge their actual exposure. The actual exposure could vary based on time spent at different locations and doing various activities (such as time spent in traffic during a commute). No wind or other meteorological variables were considered in the determination of PM exposure concentrations. By assigning all women within a given 20-km buffer the same PM monitor value, we may not be accounting for locally varying increased exposures or hot-spots within the radial buffer. However, a sensitivity analysis detected similar results at smaller distances from the monitors (5 and 10 km). This similarity of results across a range of buffers was also observed in another study of this population.[36] Additionally, information was not available on residential mobility during pregnancy, only residence at delivery, so it is possible that some women included in the study lived elsewhere and were exposed to different PM levels during part of their pregnancy. Finally, information on body mass index, which could have been associated with both neighbourhood deprivation and the outcome, was not available.

Despite these limitations, there are multiple strengths of the study. First, it includes a large number of women from across North Carolina. Additionally, multiple risk factors were simultaneously considered; air pollution and neighbourhood deprivation were examined while controlling for multiple covariates that potentially confound the associations. Finally, information on GH, as well as other maternal characteristics,

was provided from vital statistics records, which has been shown to have good reliability.[37]

In summary, a conceptual framework proposed that environmental, social and host factors all contribute to reproductive outcomes,[24] which was true in this study. Among women giving birth in North Carolina between 2000 and 2003, higher levels of both PM_{10} and $PM_{2.5}$ and higher levels of neighbourhood deprivation were positively associated with GH. Additional maternal characteristics also affected the risk of a woman having GH. Future research on the mechanisms by which air pollution and neighbourhood deprivation, or other measures of social and environmental stress, are associated with GH has the potential to improve short- and long-term health of women and their children.

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Disclaimer

The views expressed in this article are those of the authors and do not necessarily reflect the views or policies of the US Environmental Protection Agency.

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