ORIGINAL ARTICLE

Acute Air Pollution Exposure and Blood Pressure at Delivery Among Women With and Without Hypertension

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BACKGROUND
Chronic air pollution exposure increases risk for hypertensive disorders of pregnancy, but the effect of acute air pollution exposure on blood pressure during pregnancy is less well known.

METHODS
We studied 151,276 singleton term deliveries from the Consortium on Safe Labor (2002–2008) with clinical blood pressure measured at admission to labor/delivery and diagnoses of hypertensive disorders collected from electronic medical records and hospital discharge summaries. Air pollution exposures were estimated for the admission hour and the 4 hours preceding admission using a modified version of the Community Multiscale Air Quality models and observed air monitoring data. Blood pressure was categorized as normal; high normal; and mild, moderate, or severe hypertension based on pregnancy cut points. Adjusted ordinal logistic regression estimated the odds of women having a higher admission blood pressure category as a function of air pollutant, hypertensive disorders, and their interaction effect.

RESULTS
Odds of high blood pressure at admission to labor/delivery were increased in normotensive women after exposure to nitrogen oxides (by 0.2%/5 units), sulfur dioxide (by 0.3%/1 unit), carbon monoxide and several air toxics (by 3%–4%/high exposure). The effects were often similar or stronger among women with gestational hypertension and preeclampsia. Exposure to particulate matter <10 μm increased odds of high blood pressure in women with preeclampsia by 3%/5 units.

CONCLUSIONS
Air pollution can influence admission blood pressure in term deliveries and may increase likelihood of preeclampsia screening at delivery admission.

Keywords: air pollution; blood pressure; epidemiology; hypertension; pregnancy.

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Air pollution is a complex mixture of gases and particulates formed from manmade and natural processes (e.g., power plants, industry, traffic, forest fires, and volcanic eruptions) or from chemical reactions between pollutants in the atmosphere.[1] Long-term ambient air pollution exposure accounts for 2% of all cardiopulmonary disease worldwide,[2] and even low levels of air pollution increase cardiac event and hypertension risk in the general population.[1] Several criteria of air pollutants and toxics are monitored and regulated because of their health threats, including carbon monoxide (CO), nitrogen oxides (NO_x, comprising mostly nitrogen monoxide and dioxide), sulfur dioxide (SO_2), particulate matter <10 μm (PM_{10}) and <2.5 μm ($PM_{2.5}$) in aerodynamic diameter, and ozone.[1] Pregnant women might be vulnerable to adverse effects of air pollution because of cardiovascular changes and increased oxygen consumption occurring as part of normal pregnancy.[3]

Blood pressure decreases until midgestation but increases to prepregnancy levels or higher at term (≥37 weeks' gestation).[3] Women with hypertensive disorders of pregnancy have higher blood pressure throughout pregnancy with a smaller midpregnancy nadir and a greater late-pregnancy elevation.[4] Most women with hypertensive disorders of pregnancy have clustering of cardiovascular risk factors, and preeclampsia (new-onset hypertension with proteinuria or other multisystem signs) is also thought to have a placental or immunological origin.[5]

Chronic air pollution exposure increases blood pressure and the risk of hypertensive disorders of pregnancy, preterm birth, and stillbirths.[6–13] However, the reported effects

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of acute/recent air pollution exposure on blood pressure during pregnancy have not been consistent and appear to change by trimester.[7,9,14] Because any hypertensive blood pressure measured during pregnancy increases the risk of later cardiac diseases,[15] studying triggers for blood pressure elevations is important.

We studied the effects of acute air pollution exposure on blood pressure measured at admission to labor/delivery in a large population with and without hypertensive disorders of pregnancy, hypothesizing that women with hypertensive disorders have different effects than normotensive women because of their underlying differences in cardiovascular risk factors.

METHODS

The Consortium on Safe Labor (2002–2008) included 228,562 deliveries (233,736 newborns) at ≥23 weeks' gestation from 19 hospitals across the United States.[16] Data on maternal demographics, medical history, labor and delivery, and discharge diagnoses were extracted from electronic medical records. The study was approved by the institutional review boards of all participating institutions. Multifetal pregnancies (n = 5,050), singleton deliveries <37 weeks' gestation (n = 26,144), women with eclampsia (n = 93), and women missing data on blood pressure (n = 45,625), blood pressure measurement timing (n = 182), or age (n = 192) were excluded, rendering a final sample of 138,802 women with 151,276 singleton term deliveries. Most women (126,829; 91.4%) contributed only one pregnancy.

Clinical blood pressure was measured upon admission to labor/delivery by hospital staff using standard equipment. The first systolic and diastolic blood pressures and the exact date/time of admission were extracted from the electronic medical records. The following categories were created using relevant cutoffs for adult and pregnant populations:[5,17,18]

1. Normal blood pressure: systolic blood pressure <120 mm Hg and diastolic blood pressure <80 mm Hg.
2. High-normal blood pressure: systolic blood pressure 120–139 mm Hg and diastolic blood pressure <90 mm Hg or diastolic blood pressure 80–89 mmHg and systolic blood pressure <140 mm Hg.
3. Mild hypertension: systolic blood pressure 140–149 mm Hg and diastolic blood pressure <100 mm Hg or diastolic blood pressure 90–99 mm Hg and systolic blood pressure <150 mm Hg.
4. Moderate hypertension: systolic blood pressure 150–159 mm Hg and diastolic blood pressure <110 mm Hg or diastolic blood pressure 100–109 mm Hg and systolic blood pressure <160 mm Hg.
5. Severe hypertension: systolic blood pressure ≥160 mm Hg with any diastolic blood pressure or diastolic blood pressure ≥110 mm Hg with any systolic blood pressure.

Pregnancies with gestational hypertension (n = 3,935), preeclampsia (n = 5,744), chronic hypertension (n = 2,490), and superimposed preeclampsia (n = 637) were identified through electronic medical records and/or maternal discharge summaries using *International Classification of Diseases, Version 9* codes. Women were deemed normotensive (n = 138,470) if they had no indication of hypertensive disorders during pregnancy.

Air pollutant exposures were estimated with a modified version of the three-dimensional multipollutant regional air quality model developed by the US Environmental Protection Agency, the Community Multiscale Air Quality model (version 4.7.1).[19] The model used data on pollutant emission (from the National Emission Inventories), meteorology (from the Weather Research and Forecasting model), and chemical reactions between pollutants for the air quality simulations. A 36-km resolution domain covered the entire continental United States for years 2001–2009 in the simulations. Exposure was based on the predicted hourly air pollutant concentrations within 15 nonoverlapping delivery hospital referral regions, weighted to reflect the populated areas, discounting places where women were unlikely to live and work.

The modeled estimates of ozone, CO, NO_x, SO_2, PM_{10}, and $PM_{2.5}$ were fused with observed data from the US Environmental Protection Agency Air Quality System using inverse distance weighting to correct for measurement error between modeled and observed exposures (G. Chen *et al.*, unpublished data). Only modeled estimates were obtained for major $PM_{2.5}$ constituents (elemental carbon, organic compounds, ammonium, sulfate, and nitrate) and the unspeciated portion of $PM_{2.5}$ (dust particles) and for ambient air toxics because they were rarely measured by air quality monitors. Hourly averages of air pollutants were obtained for admission hour (time 0) and for the 4 hours preceding it (lags 1–4 hours). These times were chosen based on our previous analysis (T. Männistö *et al.*, unpublished data).

Statistics

Differences in demographics were tested by linear, logistic, or multinomial regression with generalized estimating equations for continuous, binary, and categorical variables, respectively. The effect of each air pollutant at the specified lag time (0, 1, 2, 3, and 4 hours preceding admission) on categorized blood pressure measured at labor/delivery admission was modeled as a function of the air pollutant, hypertensive disorder diagnosis, the interaction between air pollutant and hypertensive disorder diagnosis, and covariables. The coefficients were respectively interpreted as the pollutant effect among normotensive women, hypertensive disorder effect (pooled to a single estimate), and the pollutant effect among women with hypertensive disorders on blood pressure category. Normotensive women acted as the comparison group in the analyses. Air pollutants were fitted in the model independently because the air quality model accounted for the biochemical reactions between species and the effects of weather, other natural phenomena, and long-term pollutant sources.[19] We used ordinal logistic regression to estimate the effect of air pollutants on blood pressure categories, where the single obtained estimate (odds ratio (OR) with 95% confidence intervals (CIs)) was interpreted as the odds of being in a higher blood pressure category, assuming a similar effect between all pairs of outcome groups. Robust standard errors with clustering within a woman were calculated to account

for multiple pregnancies of the same woman. We evaluated pollutant concentrations (Supplementary Table S1) and estimated the effect of 1 μg/m^3 increase in $PM_{2.5}$, 1 parts per billion (ppb) increase in SO_2, 0.1 μg/m^3 increase in $PM_{2.5}$ constituents, 5 μg/m^3 increase in PM_{10}, 5 ppb increase in NO_x and ozone, and 10 ppb increase in CO to assess linear relationships. Air toxics were dichotomized at the 75th percentile because modeled concentrations were quite small and we determined that the most likely risk signal would be at the upper end of the distribution. All models were adjusted for site, maternal age, race/ethnicity, insurance status, smoking during pregnancy, and time of day of admission. The analyses of $PM_{2.5}$ constituents were also adjusted for total $PM_{2.5}$ mass to estimate the independent effect of constituents. Because the unadjusted and adjusted estimates were similar, only the adjusted models are presented. We also estimated the effect of air pollutants on systolic and diastolic blood pressure using linear regression with generalized estimating equations and robust standard errors (Supplementary Tables S2–S7).

Two sensitivity analyses were performed: one excluding all women taking antihypertensive medications at any time during pregnancy and one restricted to women in spontaneous labor.

All analyses were conducted using SAS version 9.3 (SAS Institute, Cary, NC).

RESULTS

Women with chronic hypertension and superimposed preeclampsia were older, less often non-Hispanic white, and less likely privately insured than normotensive women (Table 1). All hypertensive women were less likely to be normal weight, have spontaneous labor, and be admitted to labor/delivery at night, but they used antihypertensive medications more often and had higher blood pressure at admission than normotensive women. Overall, 12% of normotensive women, 59% of women with gestational hypertension or preeclampsia, 50% of women with chronic hypertension, and 72% of women with superimposed preeclampsia had hypertensive admission blood pressure. Increased ORs of having a higher admission blood pressure category were associated with all hypertension diagnoses: gestational hypertension (OR = 9.69; 95% CI = 9.00–10.43), preeclampsia (OR = 12.86; 95% CI = 12.02–13.76), chronic hypertension (OR = 7.89; 95% CI = 7.12–8.75), and superimposed preeclampsia (OR = 25.49; 95% CI = 20.83 to 31.25).

Normotensive women had increased odds of higher blood pressure category with exposure to nitrate and dust particles (by 0.1%–0.2%/0.1 units), CO (by 0.04%/10 units), NO_x (by 0.2%–0.3%/5 units), SO_2 (by 0.3%–0.4%/1 unit) (Table 2), acenaphthene, acenaphthylene, benz[a]pyrene, fluoranthene, fluorene, phenanthrene, pyrene (by 4%–7%/high exposure) (Table 3), 1,3-butadiene, propene, and styrene (by 3%–6%/high exposure) (Table 4). Exposure to sulfate particles and sesquiterpene were associated with opposing effects (Tables 2 and 4). Similar effects were seen in the linear models, where additional associations were seen between PM_{10} and lower diastolic blood pressure and between ozone, chrysene, cyclohexane, and *n*-hexane and higher systolic blood pressure (Supplementary Tables S2–S7).

Women with gestational hypertension had increased odds of higher blood pressure category with exposure to dust particles (by 0.3%/0.1 units), acenaphthene, acenaphthylene, chrysene, fluoranthene, phenanthrene, pyrene, benzene, methyl-tertiary-butyl ether, *n*-hexane, xylenes, propene, and toluene (by 18%–27%/high exposure) (Tables 2–4). Additionally, in the linear models, higher systolic or diastolic blood pressure was associated with exposures to elemental carbon, cyclohexane, NO_x, anthracene, ethylbenzene, and toluene, and lower diastolic blood pressure was associated with exposure to CO (Supplementary Tables S2–S7).

Preeclamptic women had increased odds of higher blood pressure with exposure to PM_{10} (by 3%/5 units), elemental carbon (by 1%/0.1 units), nitrate and dust particles (by 0.4%–0.6%/0.1 units), benz[a]anthracene, chrysene, fluorene, phenanthrene, pyrene, benzene, cyclohexane, *n*-hexane, xylenes, and sesquiterpene (by 12%–23%/high exposure) but lower odds with exposure to sulfate particles, ozone, CO, and indeno[1,2,3-Cd]pyrene (Tables 2–4). In the linear models, NO_x, SO_2, and benz[a]pyrene were also associated with lower systolic or diastolic blood pressure, and methyl-tertiary-butyl ether was associated with higher diastolic blood pressure (Supplementary Tables S2–S7).

Women with chronic hypertension had increased odds of higher blood pressure category with exposure to CO (by 0.3%–0.4%/10 units), NO_x (by 1%–2%/5 units), and SO_2 (by 2%–3%/1 unit), whereas opposing effects were observed with exposure to sulfate and dust particles and chrysene (Tables 2–4). Acenaphthene, o-xylene, and toluene exposures were also associated with higher systolic or diastolic blood pressure in the linear models whereas ammonium particles associated with lower diastolic blood pressure (Supplementary Tables S2–S7).

Women with superimposed preeclampsia had increased odds of higher blood pressure with exposure to CO (by 0.6%–0.7%/10 units), ozone (by 8%–11%/5 units), and anthracene (by 49%/high exposure) but lower odds with exposure to SO_2, chrysene, fluoranthene, naphthalene, pyrene, sesquiterpene, and styrene (Tables 2–4). In the linear models, benz[a]anthracene, indeno[1,2,3-Cd]pyrene, naphthalene, 1,3-butadiene, xylenes, and toluene were also associated with lower systolic blood pressure, whereas naphthalene, methyl-tertiary-butyl ether, and xylenes increased diastolic blood pressure (Supplementary Tables S2–S7).

Sensitivity analyses

All results were similar after excluding women who took antihypertensive medications (data not shown). Results among normotensive women persisted after restricting data to spontaneous labors, with some loss of precision due to smaller sample sizes. In women with hypertensive disorders, the results were more attenuated, but the association between PM_{10} and SO_2 exposure and higher blood pressure category persisted in women with preeclampsia and chronic hypertension, respectively, and novel association between $PM_{2.5}$ and higher blood pressure category was seen in women with preeclampsia and superimposed preeclampsia (data not shown).

Table 1. Characteristics of women with term singleton pregnancies and measured blood pressure

Characteristic	Normotensive 138,470 (91.5)	Gestational hypertension 3,935 (2.6)		Preeclampsia 5,744 (3.8)		Chronic hypertension 2,490 (1.7)		Superimposed preeclampsia 637 (0.4)	
No. pregnancies in the cohort									
1	124,992 (90)	3,730 (95)	<0.0001	5,403 (94)	<0.0001	2,208 (89)	0.006	606 (95)	<0.0001
2	12,825 (9)	197 (5)		319 (6)		260 (10)		31 (5)	
≥3	653 (1)	8 (0)		22 (0)		22 (1)		0 (0)	
Mean age (SD), y	27.2 (5.9)	26.7 (6.1)	<0.0001	26.9 (6.6)	<0.0001	30.7 (6.5)	<0.0001	29.6 (6.8)	0.0003
BP category on labor/delivery admission									
Normal	55,292 (40)	230 (6)	<0.0001	379 (7)	<0.0001	250 (10)	<0.0001	28 (4)	<0.0001
High-normal	66,814 (48)	1,402 (36)		1,986 (35)		999 (40)		152 (24)	
Mild hypertension	13,186 (10)	1,464 (37)		1,925 (34)		746 (30)		189 (30)	
Moderate hypertension	2,508 (2)	619 (16)		978 (17)		301 (12)		144 (23)	
Severe hypertension	670 (0)	220 (6)		476 (8)		194 (8)		124 (20)	
Nulliparous	52194 (38)	2270 (58)	<0.0001	3334 (58)	<0.0001	738 (30)	<0.0001	246 (39)	0.03
Race/ethnicity									
Non-Hispanic white	70,621 (51)	2,230 (57)	<0.0001	2,665 (46)	0.002	735 (30)	<0.0001	149 (23)	<0.0001
Non-Hispanic black	26,910 (19)	951 (24)		1,512 (26)		1,105 (44)		269 (42)	
Hispanic	25,030 (18)	508 (13)		918 (16)		449 (18)		164 (26)	
Asian/Pacific Islander	5,336 (4)	66 (2)		234 (4)		39 (2)		9 (1)	
Other	3557 (3)	62 (2)		172 (3)		110 (4)		32 (5)	
Unknown	7,016 (5)	118 (3)		243 (4)		52 (2)		14 (0)	
Insurance									
Private	83,868 (61)	2,279 (58)	0.0317	3,557 (62)	0.0043	1,100 (44)	<0.0001	212 (33)	<0.0001
Public/self-pay	44,921 (32)	1,452 (37)		1,883 (33)		1,029 (41)		309 (49)	
Other	121 (0)	10 (0)		3 (0)		3 (0)		0 (0)	
Unknown	9,560 (7)	194 (5)		301 (5)		358 (14)		116 (18)	
Pre-pregnancy BMI[a]									
Underweight	4,982 (4)	62 (2)	<0.0001	114 (2)	<0.0001	25 (1)	<0.0001	10 (2)	<0.0001
Normal weight	51,164 (37)	1,080 (28)		1,358 (24)		380 (15)		124 (20)	
Overweight	21,430 (16)	744 (19)		877 (15)		394 (16)		138 (22)	
Obese	9,627 (7)	465 (12)		551 (10)		365 (15)		118 (19)	
Severely obese	6,725 (5)	468 (12)		524 (9)		570 (23)		128 (20)	
Unknown	44,542 (32)	1,116 (28)		2,320 (40)		756 (30)		119 (19)	

Continued

Table 1. Continued

Characteristic	Normotensive 138,470 (91.5)	Gestational hypertension 3,935 (2.6)		Preeclampsia 5,744 (3.8)		Chronic hypertension 2,490 (1.7)		Superimposed preeclampsia 637 (0.4)	
Time of day at admission									
Night	29,507 (21)	491 (13)	<0.0001	813 (14)	<0.0001	357 (14)	<0.0001	99 (16)	<0.0001
Morning	53,764 (39)	1,369 (35)		1,535 (27)		995 (40)		150 (24)	
Day	27,022 (20)	1,079 (27)		1,813 (32)		582 (23)		236 (37)	
Evening	28,177 (20)	996 (25)		1,583 (28)		556 (22)		152 (24)	
Smoking	7,761 (6)	197 (5)	0.7003	273 (5)	0.9486	163 (7)	0.1838	48 (8)	0.7689
Antihypertension medication use	203 (0)	152 (4)	<0.0001	450 (8)	<0.0001	334 (13)	<0.0001	99 (16)	<0.0001
Spontaneous labor	82,878 (60)	1,318 (34)	<0.0001	2,192 (38)	<0.0001	759 (31)	<0.0001	127 (20)	<0.0001

All figures are No. (%) unless otherwise specified. *P* values are obtained from linear, logistic, or multinomial logistic regression with generalized estimating equations for continuous, binary, and categorical variables, respectively.

Abbreviations: BMI, body mass index; BP, blood pressure; SD, standard deviation.

[a]Calculated as weight/height^2 (in kg/m^2) and categorized as the following: underweight, BMI <18.5 kg/m^2; normal weight, BMI 18.5–24.9 kg/m^2; overweight, BMI 25.0–29.9 kg/m^2; obese, BMI 30.0–34.9 kg/m^2; and severely obese, BMI ≥35.0 kg/m^2.

Table 2. Odds ratios (95% confidence intervals) of a higher blood pressure category on admission to labor/delivery after exposure to criteria air pollutants, by hypertension status[a]

Exposure by hypertensive status	Time (hours) 0	1	2	3	4
Normotensive women					
PM_{10}	0.997 (0.992–1.001)	0.997 (0.992– 1.001)	0.997 (0.993–1.002)	0.997 (0.993–1.002)	0.998 (0.993–1.002)
$PM_{2.5}$	1.000 (0.999–1.001)	1.000 (0.999–1.002)	1.000 (0.999–1.002)	1.000 (0.999–1.002)	1.000 (0.999–1.002)
Elemental carbon[b]	1.000 (0.996–1.004)	0.999 (0.995–1.003)	0.999 (0.995–1.003)	1.000 (0.996–1.004)	1.000 (0.996–1.004)
Organic compounds[b]	1.000 (0.997–1.002)	1.000 (0.997–1.002)	1.000 (0.997–1.002)	1.000 (0.997–1.002)	1.000 (0.997–1.002)
Ammonium particles[b]	0.998 (0.995–1.001)	0.998 (0.995–1.001)	0.998 (0.995–1.002)	0.998 (0.995–1.001)	0.997 (0.994–1.001)
Nitrate particles[b]	**1.002 (1.001–1.003)**	**1.002 (1.001–1.003)**	**1.002 (1.001–1.003)**	**1.002 (1.001–1.003)**	**1.002 (1.001–1.003)**
Sulfate particles[b]	**0.998 (0.998–0.999)**	**0.998 (0.998–0.999)**	**0.998 (0.998–0.999)**	**0.998 (0.998–0.999)**	**0.998 (0.998–0.999)**
Dust particles[b]	1.001 (1.000–1.001)	1.001 (1.000–1.001)	**1.001 (1.000–1.001)**	**1.001 (1.000–1.002)**	**1.001 (1.000–1.002)**
CO	1.000 (1.000–1.000)	1.000 (1.000–1.000)	1.000 (1.000–1.000)	**1.000 (1.000–1.001)**	**1.000 (1.000–1.001)**

Continued

Table 2. Continued

Exposure by hypertensive status	Time (hours)				
	0	1	2	3	4
Ozone	0.997 (0.993–1.001)	0.998 (0.995–1.002)	0.999 (0.995–1.003)	0.998 (0.994–1.001)	0.997 (0.993–1.000)
NO_x	1.001 (0.999–1.002)	1.001 (0.999–1.002)	1.002 (1.000–1.003)	**1.002 (1.001–1.004)**	**1.003 (1.001–1.005)**
SO_2	**1.003 (1.001–1.006)**	**1.004 (1.002–1.007)**	**1.003 (1.001–1.006)**	**1.003 (1.000–1.005)**	1.002 (1.000–1.005)
Gestational hypertension					
PM_{10}	1.013 (0.987–1.039)	1.014 (0.988–1.040)	1.016 (0.990–1.043)	1.016 (0.990–1.042)	1.015 (0.999–1.041)
$PM_{2.5}$	1.006 (0.998–1.014)	1.006 (0.999–1.014)	1.007 (0.999–1.015)	1.006 (0.998–1.014)	1.005 (0.997–1.014)
Elemental carbon[b]	1.016 (1.000–1.033)*	1.016 (1.000–1.032)*	1.016 (0.999–1.032)*	1.015 (0.998–1.032)	1.013 (0.996–1.030)
Organic compounds[b]	1.006 (0.999–1.014)	1.006 (0.999–1.014)	1.006 (0.999–1.013)	1.005 (0.998–1.013)	1.005 (0.997–1.012)
Ammonium particles[b]	1.001 (0.995–1.008)	1.002 (0.995–1.008)	1.002 (0.995–1.008)	1.001 (0.994–1.007)	0.999 (0.993–1.006)
Nitrate particles[b]	1.001 (0.997–1.006)	1.002 (0.998–1.006)	1.003 (0.999–1.007)	1.002 (0.998–1.006)	1.002 (0.998–1.006)
Sulfate particles[b]	1.000 (0.998–1.003)	1.000 (0.998–1.002)	1.000 (0.998–1.002)	1.000 (0.997–1.002)	0.999 (0.997–1.002)
Dust particles[b]	1.001 (0.998–1.004)	1.002 (0.999–1.005)	1.003 (1.000–1.005)	**1.003 (1.000–1.006)**	**1.003 (1.000–1.006)**
CO	0.999 (0.998–1.001)	0.999 (0.998–1.001)	1.000 (0.998–1.001)	1.001 (0.999–1.002)	1.001 (0.999–1.003)
Ozone	**0.983 (0.966–1.000)**	**0.983 (0.966–1.000)**	**0.982 (0.965–0.999)**	**0.980 (0.963–0.997)**	**0.980 (0.963–0.998)**
NO_x	0.995 (0.987–1.003)	0.997 (0.989–1.005)	1.000 (0.992–1.008)	1.003 (0.995–1.011)	1.006 (0.997–1.014)
SO_2	1.006 (0.990–1.022)	1.004 (0.988–1.019)	1.004 (0.989–1.020)	1.009 (0.993–1.026)	0.998 (0.982–1.015)
Preeclampsia					
PM_{10}	**1.031 (1.009–1.053)***	**1.033 (1.011–1.055)***	**1.032 (1.010–1.054)***	**1.029 (1.007–1.051)***	**1.025 (1.003–1.047)***
$PM_{2.5}$	1.004 (0.998–1.010)	1.004 (0.998–1.010)	1.004 (0.998–1.010)	1.003 (0.997–1.009)	1.003 (0.997–1.009)
Elemental carbon[b]	**1.010 (1.001–1.019)**	**1.011 (1.003–1.020)***	**1.011 (1.003–1.020)***	**1.011 (1.002–1.019)***	**1.010 (1.001–1.018)***
Organic compounds[b]	1.004 (0.999–1.009)	1.005 (1.000–1.010)*	1.005 (1.000–1.010)*	1.004 (0.999–1.009)	1.002 (0.998–1.007)
Ammonium particles[b]	1.002 (0.997–1.007)	1.002 (0.997–1.007)	1.002 (0.997–1.008)	1.002 (0.997–1.007)	1.001 (0.996–1.006)
Nitrate particles[b]	**1.006 (1.003–1.008)**	**1.006 (1.003–1.009)***	**1.006 (1.003–1.009)***	**1.006 (1.003–1.008)***	**1.006 (1.003–1.008)***
Sulfate particles[b]	0.998 (0.997–1.000)	0.998 (0.996–1.000)	**0.998 (0.996–1.000)**	**0.998 (0.996–1.000)**	**0.998 (0.996–1.000)**
Dust particles[b]	**1.004 (1.002–1.006)***	**1.005 (1.003–1.007)***	**1.005 (1.003–1.007)***	**1.005 (1.003–1.007)***	**1.004 (1.002–1.007)***
CO	**0.998 (0.997–1.000)***	0.999 (0.997–1.000)	1.000 (0.998–1.001)	1.001 (0.999–1.002)	1.000 (0.999–1.002)
Ozone	**0.984 (0.970–0.998)**	**0.984 (0.970–0.998)***	**0.983 (0.969–0.997)***	**0.981 (0.967–0.995)***	**0.979 (0.965–0.993)***
NO_x	0.995 (0.988–1.002)	0.999 (0.992–1.006)	1.002 (0.995–1.009)	1.002 (0.996–1.009)	1.002 (0.995–1.009)
SO_2	1.002 (0.994–1.011)	1.001 (0.994–1.009)	1.003 (0.995–1.012)	1.002 (0.994–1.011)	1.001 (0.992–1.009)
Chronic hypertension					
PM_{10}	0.968 (0.935–1.002)	0.968 (0.936–1.002)	0.970 (0.937–1.004)	0.974 (0.941–1.009)	0.986 (0.952–1.020)
$PM_{2.5}$	0.994 (0.984–1.004)	0.994 (0.984–1.004)	0.994 (0.984–1.004)	0.996 (0.985–1.006)	0.998 (0.988–1.008)

Continued

Table 2. Continued

Exposure by hypertensive status	Time (hours)				
	0	1	2	3	4
Elemental carbon[b]	0.988 (0.971–1.006)	0.988 (0.971–1.007)	0.990 (0.971–1.009)	0.994 (0.975–1.014)	1.002 (0.982–1.023)
Organic compounds[b]	0.999 (0.990–1.007)	0.999 (0.990–1.007)	0.999 (0.990–1.008)	1.000 (0.991–1.009)	1.002 (0.993–1.012)
Ammonium particles[b]	0.993 (0.986–1.001)	0.994 (0.986–1.001)	0.994 (0.986–1.001)	0.994 (0.987–1.001)	0.995 (0.988–1.003)
Nitrate particles[b]	1.000 (0.996–1.004)	1.000 (0.996–1.003)	1.000 (0.996–1.003)	1.000 (0.996–1.004)	1.001 (0.997–1.005)
Sulfate particles[b]	**0.997 (0.994–1.000)**	**0.997 (0.994–1.000)**	**0.997 (0.994–1.000)**	**0.997 (0.994–1.000)**	0.997 (0.994–1.000)
Dust particles[b]	**0.996 (0.993–0.999)***	**0.996 (0.993–0.999)***	**0.997 (0.993–1.000)***	0.998 (0.994–1.001)	0.999 (0.995–1.002)
CO	1.002 (0.999–1.004)	1.002 (0.999–1.004)	1.002 (0.999–1.004)	**1.003 (1.000–1.006)***	**1.004 (1.001–1.007)***
Ozone	1.012 (0.988–1.037)	1.016 (0.991–1.041)	1.014 (0.989–1.039)	1.014 (0.990–1.039)	1.013 (0.988–1.038)
NO_x	1.007 (0.995–1.019)	1.008 (0.997–1.020)	1.010 (0.999–1.021)	**1.011 (1.001–1.022)**	**1.017 (1.005–1.029)***
SO_2	1.015 (0.993–1.038)	**1.023 (1.003–1.043)**	**1.027 (1.005–1.049)***	1.009 (0.988–1.032)	**1.023 (1.002–1.045)**
Superimposed preeclampsia					
PM_{10}	1.039 (0.959–1.125)	1.048 (0.966–1.136)	1.053 (0.971–1.142)	1.047 (0.967–1.134)	1.043 (0.964–1.129)
$PM_{2.5}$	1.013 (0.991–1.037)	1.016 (0.993–1.039)	1.017 (0.995–1.039)	1.015 (0.994–1.037)	1.015 (0.995–1.036)
Elemental carbon[b]	0.984 (0.944–1.026)	0.990 (0.950–1.032)	0.996 (0.955–1.038)	0.997 (0.956–1.039)	1.000 (0.959–1.042)
Organic compounds[b]	1.006 (0.987–1.026)	1.008 (0.989–1.028)	1.009 (0.990–1.029)	1.007 (0.988–1.027)	1.007 (0.987–1.027)
Ammonium particles[b]	0.994 (0.978–1.010)	0.996 (0.980–1.012)	0.999 (0.984–1.015)	0.998 (0.983–1.013)	0.998 (0.983–1.013)
Nitrate particles[b]	0.992 (0.981–1.003)	0.995 (0.984–1.005)	0.996 (0.986–1.006)	0.996 (0.986–1.006)	0.996 (0.987–1.006)
Sulfate particles[b]	1.002 (0.996–1.009)	1.002 (0.996–1.008)	1.002 (0.996–1.008)	1.002 (0.996–1.007)	1.001 (0.996–1.007)
Dust particles[b]	0.998 (0.990–1.006)	0.999 (0.990–1.009)	1.000 (0.991–1.009)	0.999 (0.991–1.008)	0.999 (0.991–1.008)
CO	1.001 (0.995–1.007)	1.004 (0.997–1.010)	1.005 (0.998–1.011)	**1.007 (1.000–1.013)**	**1.006 (1.000–1.012)**
Ozone	**1.111 (1.061–1.164)***	**1.108 (1.057–1.160)***	**1.104 (1.054–1.156)***	**1.090 (1.040–1.142)***	**1.080 (1.029–1.132)***
NO_x	0.986 (0.958–1.014)	0.991 (0.967–1.016)	0.995 (0.972–1.017)	1.003 (0.977–1.031)	1.009 (0.982–1.035)
SO_2	0.983 (0.945–1.023)	0.999 (0.971–1.029)	0.979 (0.943–1.016)	**0.963 (0.930–0.997)***	0.993 (0.952–1.036)

Abbreviations: CO, carbon monoxide; NO_x, nitrogen oxides; $PM_{2.5}$, particulate matter <2.5 μm; PM_{10}, particulate matter <10 μm; ppb, parts per billion; SO_2, sulfur dioxide.

[a]Ordinal logistic regression with a function of air pollutant, hypertensive disorder, and their interaction and results clustered within woman estimated the effect of 1-unit increase in elemental carbon, organic compounds, and ammonium, nitrate, sulfate and dust particles (in $\mu g/m^3$) and SO_2 (in ppb); 0.1-unit increase in $PM_{2.5}$ constituents (in $\mu g/m^3$); 5-unit increase in PM_{10} (in $\mu g/m^3$), NO_x (in ppb) and ozone (in ppb); and 10-unit increase in CO (in ppb), adjusted for site, maternal age, race/ethnicity, insurance, smoking during pregnancy, and time of day.

[b]Model is further adjusted for total $PM_{2.5}$ mass.

*Interaction term is significant ($P < 0.05$), suggesting that the response is different than in the normotensive women.

Estimates that are statistically significant are given in bold.

Table 3. Odds ratios (95% confidence intervals) of a higher blood pressure category on admission to labor/delivery after high exposure to polycyclic aromatic hydrocarbons, by hypertension status[a]

Exposure by hypertensive status	Time (hours)				
	0	1	2	3	4
Normotensive women					
Acenaphthene	**1.067 (1.039–1.095)**	**1.065 (1.037–1.094)**	**1.072 (1.044–1.101)**	**1.070 (1.042–1.099)**	**1.073 (1.045–1.102)**
Acenaphthylene	**1.077 (1.048–1.106)**	**1.072 (1.043–1.101)**	**1.073 (1.044–1.102)**	**1.068 (1.040–1.097)**	**1.069 (1.041–1.098)**
Anthracene	1.010 (0.983–1.037)	1.006 (0.979–1.033)	1.006 (0.979–1.033)	1.015 (0.988–1.042)	1.019 (0.992–1.046)
Benz[a]anthracene	0.997 (0.971–1.024)	1.003 (0.977–1.031)	1.011 (0.985–1.039)	1.012 (0.985–1.040)	1.004 (0.978–1.032)
Benz[a]pyrene	1.014 (0.986–1.043)	1.023 (0.995–1.052)	**1.029 (1.001–1.058)**	**1.031 (1.003–1.06)**	1.020 (0.992–1.049)
Chrysene	1.006 (0.978–1.034)	1.009 (0.982–1.038)	1.016 (0.988–1.045)	1.022 (0.994–1.051)	1.020 (0.992–1.049)
Fluoranthene	**1.067 (1.039–1.096)**	**1.063 (1.035–1.092)**	**1.064 (1.035–1.093)**	**1.059 (1.031–1.088)**	**1.062 (1.034–1.091)**
Fluorene	**1.053 (1.026–1.081)**	**1.049 (1.022–1.077)**	**1.047 (1.020–1.074)**	**1.053 (1.025–1.081)**	**1.060 (1.032–1.088)**
Indeno[1,2,3Cd]pyrene	0.994 (0.969–1.020)	0.998 (0.973–1.024)	1.001 (0.975–1.026)	0.995 (0.970–1.021)	0.999 (0.974–1.025)
Naphthalene	1.012 (0.984–1.040)	1.026 (0.998–1.055)	**1.039 (1.011–1.068)**	**1.051 (1.023–1.080)**	**1.038 (1.010–1.067)**
Phenanthrene	**1.065 (1.037–1.094)**	**1.065 (1.037–1.094)**	**1.062 (1.033–1.090)**	**1.064 (1.036–1.093)**	**1.064 (1.036–1.093)**
Pyrene	**1.053 (1.025–1.081)**	**1.058 (1.030–1.087)**	**1.063 (1.034–1.091)**	**1.056 (1.028–1.085)**	**1.060 (1.031–1.088)**
Gestational hypertension					
Acenaphthene	1.134 (0.980–1.312)	1.115 (0.964–1.290)	1.106 (0.955–1.282)	**1.174 (1.014–1.360)**	1.135 (0.981–1.312)
Acenaphthylene	**1.259 (1.089–1.455)***	**1.189 (1.029–1.374)***	1.154 (0.999–1.333)	1.151 (0.995–1.331)	1.109 (0.960–1.282)
Anthracene	1.110 (0.973–1.267)	1.093 (0.958–1.247)	1.121 (0.983–1.279)	1.113 (0.975–1.270)	1.092 (0.957–1.247)
Benz[a]anthracene	1.026 (0.889–1.183)	1.051 (0.911–1.214)	1.125 (0.976–1.297)	1.048 (0.909–1.207)	0.978 (0.850–1.125)
Benz[a]pyrene	1.014 (0.874–1.175)	1.096 (0.945–1.271)	1.096 (0.946–1.270)	1.096 (0.945–1.271)	1.108 (0.955–1.285)
Chrysene	1.050 (0.902–1.221)	1.108 (0.953–1.288)	**1.201 (1.032–1.398)***	**1.249 (1.073–1.455)***	**1.259 (1.080–1.468)***
Fluoranthene	1.148 (0.987–1.336)	1.127 (0.968–1.313)	1.138 (0.978–1.325)	**1.188 (1.021–1.382)**	**1.172 (1.010–1.360)**
Fluorene	1.136 (0.984–1.311)	1.138 (0.985–1.313)	1.094 (0.947–1.262)	1.131 (0.979–1.307)	1.124 (0.975–1.295)
Indeno[1,2,3Cd]pyrene	0.939 (0.809–1.089)	0.984 (0.847–1.142)	0.985 (0.849–1.144)	0.976 (0.840–1.134)	0.908 (0.782–1.054)
Naphthalene	1.010 (0.861–1.185)	1.019 (0.867–1.198)	1.038 (0.884–1.218)	0.990 (0.844–1.161)	1.002 (0.857–1.171)
Phenanthrene	**1.162 (1.004–1.344)**	**1.193 (1.031–1.381)**	**1.188 (1.025–1.378)**	**1.166 (1.007–1.349)**	1.119 (0.968–1.295)
Pyrene	1.139 (0.978–1.327)	1.146 (0.983–1.336)	1.163 (0.996–1.357)	**1.223 (1.051–1.424)**	**1.249 (1.076–1.451)***
Preeclampsia					
Acenaphthene	1.011 (0.906–1.129)	1.030 (0.923–1.149)	1.010 (0.905–1.129)	1.027 (0.919–1.147)	1.066 (0.955–1.190)
Acenaphthylene	1.067 (0.955–1.192)	1.063 (0.952–1.188)	1.051 (0.940–1.174)	1.060 (0.948–1.185)	1.042 (0.933–1.163)
Anthracene	1.052 (0.941–1.177)	1.086 (0.971–1.215)	1.067 (0.955–1.193)	1.071 (0.958–1.198)	1.045 (0.935–1.168)

Continued

Table 3. Continued

Exposure by hypertensive status	Time (hours)				
	0	1	2	3	4
Benz[a]anthracene	**1.158 (1.039–1.291)***	**1.201 (1.077–1.339)***	**1.164 (1.044–1.299)***	**1.194 (1.071–1.331)***	1.081 (0.971–1.205)
Benz[a]pyrene	0.938 (0.838–1.050)	0.952 (0.851–1.066)	0.936 (0.836–1.047)	0.942 (0.842–1.054)	0.957 (0.856–1.070)
Chrysene	**1.176 (1.051–1.317)***	**1.154 (1.031–1.292)***	**1.229 (1.098–1.375)***	**1.174 (1.049–1.314)***	**1.125 (1.005–1.258)***
Fluoranthene	1.106 (0.991–1.234)	1.078 (0.966–1.203)	1.046 (0.937–1.168)	1.042 (0.933–1.164)	1.064 (0.954–1.187)
Fluorene	**1.135 (1.016–1.267)**	**1.124 (1.006–1.256)**	1.096 (0.980–1.226)	1.077 (0.964–1.204)	1.081 (0.968–1.207)
Indeno[1,2,3Cd]pyrene	**0.861 (0.769–0.965)***	**0.870 (0.775–0.975)***	0.891 (0.793–1.001)	**0.878 (0.781–0.987)***	0.929 (0.827–1.045)
Naphthalene	1.076 (0.965–1.199)	1.067 (0.957–1.190)	1.086 (0.974–1.212)	1.077 (0.966–1.200)	1.056 (0.948–1.176)
Phenanthrene	1.115 (0.998–1.246)	**1.124 (1.006–1.256)**	1.117 (1.000–1.248)	1.075 (0.962–1.202)	1.083 (0.970–1.209)
Pyrene	**1.163 (1.041–1.299)**	**1.160 (1.037–1.297)**	**1.130 (1.011–1.262)**	1.037 (0.928–1.158)	1.047 (0.938–1.168)
Chronic hypertension					
Acenaphthene	1.057 (0.883–1.267)	1.059 (0.884–1.269)	0.974 (0.812–1.169)	0.993 (0.829–1.189)	1.052 (0.877–1.261)
Acenaphthylene	1.043 (0.871–1.250)	1.020 (0.851–1.221)	1.008 (0.842–1.208)	0.986 (0.822–1.182)	1.028 (0.856–1.234)
Anthracene	1.011 (0.848–1.206)	0.977 (0.819–1.165)	0.990 (0.830–1.182)	0.958 (0.804–1.143)	1.005 (0.843–1.197)
Benz[a]anthracene	0.925 (0.780–1.097)	0.918 (0.774–1.089)	0.868 (0.731–1.031)	0.949 (0.798–1.128)	1.042 (0.876–1.240)
Benz[a]pyrene	0.938 (0.782–1.125)	0.933 (0.779–1.116)	0.960 (0.803–1.149)	0.992 (0.829–1.188)	1.027 (0.858–1.229)
Chrysene	**0.804 (0.680–0.951)***	**0.789 (0.667–0.934)***	**0.814 (0.687–0.964)***	0.893 (0.754–1.057)	0.917 (0.775–1.086)
Fluoranthene	0.965 (0.807–1.154)	1.020 (0.851–1.222)	1.023 (0.853–1.227)	1.016 (0.848–1.218)	1.088 (0.910–1.300)
Fluorene	1.010 (0.847–1.205)	0.929 (0.778–1.110)	0.940 (0.786–1.123)	0.965 (0.807–1.154)	0.996 (0.835–1.189)
Indeno[1,2,3Cd]pyrene	0.967 (0.811–1.152)	1.001 (0.841–1.190)	1.029 (0.865–1.223)	1.075 (0.906–1.277)	1.013 (0.854–1.202)
Naphthalene	0.957 (0.800–1.146)	0.935 (0.782–1.118)	1.025 (0.857–1.225)	1.116 (0.934–1.334)	1.114 (0.932–1.331)
Phenanthrene	1.105 (0.924–1.321)	1.072 (0.896–1.283)	1.062 (0.888–1.270)	0.956 (0.801–1.142)	1.061 (0.891–1.262)
Pyrene	0.915 (0.765–1.095)	0.933 (0.778–1.119)	1.014 (0.845–1.217)	1.046 (0.873–1.253)	1.068 (0.892–1.279)
Superimposed preeclampsia					
Acenaphthene	0.945 (0.648–1.378)	0.854 (0.575–1.270)	0.888 (0.601–1.310)	0.882 (0.596–1.306)	1.101 (0.745–1.627)
Acenaphthylene	0.844 (0.569–1.251)	0.775 (0.521–1.152)	0.797 (0.536–1.187)	0.866 (0.588–1.275)	0.940 (0.636–1.388)
Anthracene	1.259 (0.879–1.805)	**1.494 (1.044–2.140)***	1.265 (0.879–1.821)	1.173 (0.819–1.680)	1.110 (0.776–1.586)
Benz[a]anthracene	0.759 (0.542–1.064)	0.767 (0.549–1.072)	0.832 (0.594–1.166)	0.958 (0.688–1.335)	0.823 (0.593–1.142)
Benz[a]pyrene	0.693 (0.451–1.063)	0.788 (0.509–1.219)	0.884 (0.571–1.368)	1.079 (0.701–1.662)	0.935 (0.593–1.474)

Continued

Table 3. Continued

Exposure by hypertensive status	Time (hours)				
	0	1	2	3	4
Chrysene	**0.700 (0.498–0.983)***	0.742 (0.524–1.050)	0.869 (0.612–1.233)	0.817 (0.581–1.150)	0.965 (0.690–1.348)
Fluoranthene	0.728 (0.497–1.068)	0.685 (0.463–1.014)*	**0.609 (0.409–0.908)***	0.703 (0.473–1.044)*	0.867 (0.584–1.288)
Fluorene	0.881 (0.605–1.281)	0.819 (0.557–1.205)	0.870 (0.587–1.288)	0.847 (0.572–1.255)	0.991 (0.671–1.463)
Indeno[1,2,3Cd]pyrene	0.765 (0.530–1.105)	0.951 (0.658–1.376)	0.938 (0.646–1.362)	0.982 (0.673–1.433)	1.141 (0.782–1.666)
Naphthalene	**0.688 (0.483–0.980)***	0.717 (0.502–1.023)*	0.827 (0.576–1.187)	0.988 (0.694–1.407)	0.903 (0.633–1.289)
Phenanthrene	0.890 (0.597–1.325)	0.790 (0.531–1.174)	0.743 (0.498–1.107)	0.812 (0.545–1.212)	0.991 (0.668–1.470)
Pyrene	0.702 (0.486–1.015)*	**0.616 (0.426–0.891)***	**0.635 (0.436–0.924)***	0.750 (0.515–1.092)	0.827 (0.572–1.198)

[a]Ordinal logistic regression with linear function of air pollutant, hypertensive disorder, and their interaction and results clustered within woman estimated the effect of high air toxics exposure (≥75th percentile, in parts per billion), adjusted for site, maternal age, race/ethnicity, insurance, smoking during pregnancy, and time of day.

*Interaction term is significant ($P < 0.05$), suggesting that the response is different than in the normotensive women.

Estimates that are statistically significant are given in bold.

Table 4. Odds ratios (95% confidence intervals) of higher blood pressure category on admission to labor/delivery by volatile organic compounds[a]

Exposure by hypertensive status	Time (hours)				
	0	1	2	3	4
Normotensive					
Benzene	1.001 (0.973–1.030)	1.009 (0.981–1.038)	1.006 (0.978–1.035)	0.997 (0.970–1.026)	0.995 (0.967–1.023)
1,3-butadiene	1.013 (0.985–1.043)	1.018 (0.990–1.047)	**1.038 (1.010–1.067)**	**1.051 (1.022–1.081)**	**1.058 (1.028–1.089)**
Ethylbenzene	0.997 (0.968–1.028)	1.001 (0.971–1.032)	0.999 (0.969–1.030)	0.999 (0.969–1.030)	0.986 (0.956–1.016)
Cyclohexane	1.008 (0.977–1.040)	1.016 (0.985–1.048)	1.013 (0.982–1.045)	1.016 (0.985–1.048)	1.015 (0.984–1.047)
Methyl-tertiary-butyl ether	0.983 (0.956–1.010)	0.992 (0.965–1.019)	0.997 (0.971–1.025)	0.994 (0.968–1.022)	0.980 (0.953–1.007)
N-hexane	1.000 (0.972–1.029)	1.010 (0.982–1.039)	1.007 (0.979–1.036)	1.011 (0.983–1.040)	0.994 (0.966–1.023)
Ethyl-methyl ketone	0.979 (0.951–1.008)	1.001 (0.972–1.030)	0.999 (0.970–1.028)	0.995 (0.967–1.025)	0.988 (0.959–1.017)
M-xylene	0.996 (0.966–1.027)	1.009 (0.979–1.040)	1.005 (0.975–1.036)	1.002 (0.972–1.033)	1.001 (0.971–1.032)
O-xylene	0.993 (0.962–1.024)	0.985 (0.955–1.017)	1.000 (0.969–1.032)	1.007 (0.976–1.039)	0.998 (0.968–1.030)
P-xylene	0.996 (0.966–1.026)	1.008 (0.978–1.039)	1.003 (0.973–1.033)	1.000 (0.970–1.030)	0.983 (0.954–1.014)
Propene	1.000 (0.974–1.027)	1.014 (0.988–1.041)	**1.036 (1.009–1.064)**	**1.047 (1.020–1.075)**	**1.039 (1.012–1.067)**

Continued

Table 4. Continued

Exposure by hypertensive status	Time (hours)				
	0	1	2	3	4
Sesquiterpene	**0.971 (0.948–0.995)**	0.979 (0.955–1.003)	0.992 (0.968–1.017)	0.992 (0.969–1.016)	0.992 (0.968–1.016)
Styrene	0.986 (0.958–1.016)	1.000 (0.972–1.029)	1.017 (0.989–1.046)	**1.031 (1.002–1.061)**	**1.035 (1.005–1.066)**
Toluene	0.988 (0.958–1.019)	0.982 (0.952–1.013)	0.995 (0.964–1.026)	1.004 (0.973–1.035)	1.004 (0.973–1.036)
Gestational hypertension					
Benzene	1.153 (0.992–1.340)	**1.183 (1.014–1.379)***	**1.216 (1.045–1.415)***	**1.190 (1.025–1.383)***	**1.203 (1.037–1.394)***
1,3-butadiene	0.960 (0.837–1.101)	0.991 (0.861–1.140)	1.008 (0.874–1.162)	1.010 (0.875–1.165)	1.077 (0.932–1.244)
Ethylbenzene	1.163 (0.989–1.369)	1.119 (0.951–1.318)	1.103 (0.935–1.300)	1.141 (0.972–1.340)	1.143 (0.974–1.342)
Cyclohexane	1.164 (0.986–1.375)	1.140 (0.966–1.345)	1.176 (0.996–1.387)	**1.198 (1.015–1.415)**	1.132 (0.961–1.332)
Methyl-tertiary-butyl ether	1.144 (0.992–1.320)*	**1.221 (1.059–1.409)***	**1.259 (1.092–1.451)***	**1.186 (1.031–1.366)***	**1.154 (1.003–1.328)***
N-hexane	**1.266 (1.088–1.472)***	**1.252 (1.076–1.456)***	**1.270 (1.092–1.476)***	**1.275 (1.097–1.481)***	**1.218 (1.048–1.416)***
Ethyl-methyl ketone	1.055 (0.901–1.236)	1.080 (0.921–1.266)	1.096 (0.936–1.284)	1.093 (0.933–1.280)	1.080 (0.924–1.262)
M-xylene	1.132 (0.961–1.332)	**1.203 (1.019–1.422)***	**1.190 (1.010–1.402)***	**1.235 (1.048–1.456)***	**1.229 (1.046–1.443)***
O-xylene	**1.184 (1.001–1.402)***	1.183 (0.997–1.404)*	1.179 (0.995–1.398)	1.178 (0.995–1.395)	**1.198 (1.015–1.413)***
P-xylene	1.085 (0.920–1.279)	1.110 (0.942–1.307)	1.144 (0.971–1.347)	1.153 (0.981–1.355)	1.098 (0.935–1.288)
Propene	**1.203 (1.048–1.382)***	**1.222 (1.06–1.408)***	**1.211 (1.049–1.397)***	**1.255 (1.085–1.452)***	**1.242 (1.073–1.437)***
Sesquiterpene	1.017 (0.888–1.165)	0.992 (0.866–1.136)	1.044 (0.913–1.195)	1.092 (0.954–1.249)	1.010 (0.881–1.156)
Styrene	1.008 (0.878–1.158)	1.044 (0.907–1.203)	1.062 (0.922–1.224)	1.026 (0.890–1.182)	1.054 (0.914–1.214)
Toluene	1.174 (0.992–1.390)	1.171 (0.988–1.387)*	**1.219 (1.028–1.444)***	**1.213 (1.027–1.431)***	**1.209 (1.025–1.426)***
Preeclampsia					
Benzene	**1.162 (1.046–1.291)***	**1.179 (1.061–1.309)***	**1.194 (1.074–1.327)***	**1.160 (1.043–1.289)***	**1.115 (1.003–1.238)***
1,3-butadiene	0.946 (0.842–1.063)	0.936 (0.831–1.056)	0.947 (0.836–1.073)	0.992 (0.874–1.127)	0.966 (0.849–1.100)
Ethylbenzene	1.110 (0.998–1.234)	1.101 (0.990–1.224)	1.078 (0.970–1.199)	1.053 (0.947–1.171)	1.029 (0.925–1.144)
Cyclohexane	1.104 (0.992–1.228)	**1.129 (1.014–1.256)**	**1.145 (1.029–1.274)***	**1.129 (1.015–1.256)**	1.096 (0.985–1.219)
Methyl-tertiary-butyl ether	1.084 (0.976–1.205)	1.107 (0.997–1.230)*	1.110 (0.999–1.234)	1.074 (0.966–1.193)	1.028 (0.925–1.142)
N-hexane	**1.265 (1.138–1.406)***	**1.236 (1.112–1.375)***	**1.180 (1.060–1.312)***	**1.193 (1.073–1.326)***	**1.142 (1.027–1.270)***
Ethyl-methyl ketone	1.070 (0.962–1.191)	1.104 (0.993–1.229)	1.067 (0.959–1.187)	1.039 (0.934–1.156)	1.030 (0.926–1.146)
M-xylene	**1.117 (1.004–1.242)***	**1.128 (1.014–1.255)***	1.106 (0.995–1.231)	1.042 (0.937–1.159)	1.006 (0.905–1.119)
O-xylene	1.058 (0.951–1.176)	1.042 (0.936–1.159)	1.026 (0.922–1.141)	1.013 (0.911–1.127)	0.980 (0.881–1.089)
P-xylene	1.101 (0.990–1.224)	**1.125 (1.012–1.251)***	1.088 (0.979–1.211)	1.043 (0.938–1.159)	0.992 (0.892–1.103)
Propene	1.019 (0.914–1.136)	1.017 (0.911–1.135)	1.079 (0.965–1.206)	1.096 (0.979–1.227)	1.027 (0.916–1.15)

Continued

Table 4. Continued

Exposure by hypertensive status	Time (hours)				
	0	1	2	3	4
Sesquiterpene	**1.130 (1.010–1.264)***	**1.125 (1.005–1.258)***	**1.122 (1.002–1.256)***	1.066 (0.953–1.192)	1.112 (0.996–1.242)
Styrene	0.980 (0.871–1.103)	0.939 (0.832–1.059)	0.996 (0.879–1.128)	1.022 (0.899–1.161)	1.033 (0.908–1.176)
Toluene	1.057 (0.950–1.176)	1.047 (0.941–1.165)	1.050 (0.944–1.168)	1.021 (0.918–1.135)	0.998 (0.898–1.109)
Chronic hypertension					
Benzene	0.890 (0.743–1.065)	0.957 (0.799–1.147)	0.975 (0.811–1.171)	0.996 (0.829–1.197)	1.041 (0.868–1.249)
1,3-butadiene	1.022 (0.862–1.213)	0.946 (0.800–1.119)	0.972 (0.823–1.149)	0.954 (0.806–1.129)	0.903 (0.763–1.068)
Ethylbenzene	0.911 (0.760–1.093)	0.917 (0.765–1.099)	0.922 (0.767–1.109)	1.060 (0.883–1.273)	1.070 (0.893–1.282)
Cyclohexane	0.885 (0.741–1.057)	0.931 (0.777–1.115)	0.919 (0.767–1.102)	0.947 (0.791–1.133)	1.011 (0.847–1.207)
Methyl-tertiary-butyl ether	0.961 (0.810–1.140)	0.972 (0.820–1.153)	1.024 (0.864–1.213)	0.976 (0.824–1.155)	1.101 (0.929–1.304)
N-hexane	0.852 (0.714–1.017)	0.908 (0.760–1.085)	0.889 (0.743–1.063)	1.007 (0.844–1.202)	1.036 (0.868–1.237)
Ethyl-methyl ketone	0.967 (0.808–1.158)	0.853 (0.712–1.023)	0.879 (0.735–1.052)	1.004 (0.842–1.198)	0.973 (0.816–1.160)
M-xylene	0.905 (0.755–1.084)	0.904 (0.755–1.082)	0.956 (0.797–1.149)	0.986 (0.821–1.183)	1.054 (0.880–1.261)
O-xylene	0.946 (0.788–1.137)	0.983 (0.819–1.181)	1.032 (0.858–1.241)	1.094 (0.911–1.313)	1.057 (0.878–1.272)
P-xylene	0.915 (0.765–1.095)	0.942 (0.787–1.127)	0.934 (0.777–1.123)	1.052 (0.876–1.264)	1.092 (0.911–1.308)
Propene	0.953 (0.800–1.135)	0.919 (0.771–1.096)	0.913 (0.766–1.088)	0.941 (0.789–1.123)	1.038 (0.871–1.237)
Sesquiterpene	0.891 (0.747–1.062)	0.957 (0.804–1.141)	1.050 (0.881–1.253)	0.968 (0.814–1.152)	0.978 (0.821–1.166)
Styrene	1.000 (0.845–1.184)	0.989 (0.836–1.169)	0.921 (0.779–1.088)	0.853 (0.722–1.008)	0.892 (0.755–1.053)
Toluene	1.006 (0.837–1.208)	1.012 (0.843–1.216)	1.037 (0.862–1.248)	1.084 (0.904–1.301)	1.062 (0.884–1.275)
Superimposed preeclampsia					
Benzene	0.778 (0.547–1.105)	0.793 (0.554–1.135)	0.870 (0.606–1.249)	0.928 (0.652–1.321)	0.910 (0.640–1.292)
1,3-butadiene	0.765 (0.553–1.057)	0.767 (0.551–1.067)	0.809 (0.580–1.129)	0.797 (0.569–1.115)	**0.702 (0.504–0.979)***
Ethylbenzene	0.748 (0.527–1.061)	0.774 (0.544–1.100)	0.793 (0.558–1.128)	0.887 (0.629–1.253)	0.909 (0.646–1.280)
Cyclohexane	0.774 (0.545–1.099)	0.867 (0.612–1.227)	0.814 (0.571–1.160)	0.886 (0.630–1.248)	0.891 (0.634–1.253)
Methyl-tertiary-butyl ether	0.960 (0.692–1.333)	1.077 (0.775–1.497)	1.126 (0.809–1.567)	1.285 (0.928–1.779)	1.250 (0.901–1.734)
N-hexane	0.794 (0.561–1.123)	0.759 (0.535–1.076)	0.731 (0.513–1.043)	0.895 (0.632–1.266)	0.849 (0.603–1.195)
Ethyl-methyl ketone	0.812 (0.573–1.150)	0.858 (0.608–1.212)	0.847 (0.598–1.200)	0.868 (0.618–1.218)	0.916 (0.657–1.276)
M-xylene	0.719 (0.507–1.017)	0.775 (0.544–1.105)	0.889 (0.618–1.280)	0.898 (0.634–1.272)	0.854 (0.603–1.208)
O-xylene	0.716 (0.501–1.024)	0.770 (0.539–1.098)	0.850 (0.590–1.224)	0.992 (0.694–1.417)	0.986 (0.693–1.403)

Continued

Table 4. Continued

Exposure by hypertensive status	Time (hours)				
	0	1	2	3	4
P-xylene	0.762 (0.537–1.081)	0.820 (0.578–1.163)	0.871 (0.613–1.238)	0.892 (0.634–1.255)	0.870 (0.618–1.224)
Propene	0.914 (0.655–1.275)	1.026 (0.732–1.438)	0.903 (0.634–1.287)	0.797 (0.562–1.130)	0.916 (0.652–1.286)
Sesquiterpene	**0.655 (0.467–0.918)**	0.727 (0.514–1.027)	0.734 (0.515–1.046)	0.795 (0.563–1.123)	0.926 (0.658–1.304)
Styrene	0.872 (0.629–1.209)	0.830 (0.596–1.155)	0.764 (0.549–1.062)	0.763 (0.547–1.065)	**0.694 (0.498–0.967)***
Toluene	0.745 (0.520–1.066)	0.738 (0.516–1.055)	0.860 (0.599–1.236)	0.944 (0.661–1.350)	1.013 (0.713–1.440)

[a]Ordinal logistic regression with linear function of air pollutant, hypertensive disorder, and their interaction and results clustered within woman estimated the effect of high air toxics exposure (≥75th percentile in parts per billion), adjusted for site, maternal age, race/ethnicity, insurance, smoking during pregnancy, and time of day.
*Interaction term is significant ($P < 0.05$), suggesting that the response is different than in the normotensive women.
Estimates that are statistically significant are given in bold.

DISCUSSION

In this large study of women with term deliveries, acute exposure to several air pollutants increased the odds of having higher blood pressure on admission to labor/delivery. Although most associations with blood pressure elevations were seen among normotensive women, we also observed air pollutant effects among women with preeclampsia and gestational hypertension. To our knowledge, this is the first large study evaluating the association between acute air pollution exposure and blood pressure during pregnancy. Pregnancy is associated with changes in the cardiovascular system and blood pressure, but blood pressure measured at term typically is comparable with or exceeds prepregnancy values.[3]

We found higher blood pressure on admission to labor/delivery among normotensive women after exposure to CO, NO_x, SO_2, some $PM_{2.5}$ constituents, and several air toxics. In the general population, acute and recent exposures to PM_{10}, $PM_{2.5}$, NO_x, SO_2, and ozone increased blood pressure in most studies,[20–24] although in nonsmoking adults, PM_{10} and ozone exposure decreased systolic BP.[23] Previous studies have also consistently shown that long-term air pollution exposure increases blood pressure and/or risk of hypertensive disorders in pregnancy.[6–11] Acute or recent PM_{10} or NO_x exposures increased blood pressure during pregnancy in some, but not all, studies.[7,9,14] These studies in pregnant women have generally estimated the effect of short-term air pollution (e.g., daily pollution averages), excluded women with chronic hypertension, and lacked power to evaluate specific hypertensive disorders, which may explain why their results are somewhat different from ours.

We found that normotensive women and those with hypertensive disorders of pregnancy often had blood pressure effects in similar direction with air pollution exposure, but the magnitude of effect was often stronger in women with hypertensive disorders. Preeclamptic women seemed susceptible to the effects of particulates because total PM_{10} and several $PM_{2.5}$ constituents increased blood pressure, whereas opposing effects were sometimes seen among normotensive women. In women with chronic hypertension and superimposed preeclampsia, air pollution exposure generally was either associated with no effect on blood pressure or lower odds of high blood pressure. Most of our observed negative effects remained after excluding women using antihypertensive medication. Results of previous studies have also been inconsistent regarding air pollution effects on blood pressure among people with chronic hypertension, as in 1 study, air pollution increased blood pressure only in elderly persons taking antihypertensive medication,[25] whereas opposing effects were seen in another study.[26] The underlying chronic hypertension and associated vascular changes may mask any small effects air pollutants have on blood pressure among women with chronic hypertension and superimposed preeclampsia, leading to seemingly negative or null effects.

We observed several novel associations between high blood pressure on admission to labor/delivery and air toxics, mostly among normotensive women and/or among women with gestational hypertension or preeclampsia. Previously, increased blood pressure was observed after

acute exposure to oxygenated polycyclic aromatic hydrocarbons in particulates among elderly people using antihypertensive medication,[25] and long-term exposure to some air toxics has been associated with ischemic heart disease and total mortality.[27,28] Our results support increases in blood pressure associated with acute air toxics exposure in pregnancy.

Inhaled air pollutants have been found to have systemic effects in the general and pregnant populations, including elevations in inflammatory markers, oxidative stress, plasma viscosity, fibrinogen, and atherosclerosis, and endothelial dysfunction.[1,21,29–31] The very acute effects of air pollution are likely mediated by autonomic nervous system imbalance, leading to vasoconstriction.[21] Women with preeclampsia have increases in peripheral vascular resistance and may be hyperresponsive to vasoactive factors and even have reversal of the circadian rhythm of blood pressure.[5] Additionally, women with hypertensive disorders of pregnancy have higher rates of cardiovascular risk factors, and especially women with preeclampsia have higher inflammation and more endothelial dysfunction,[5] which may precipitate and enhance any effects of air pollution on blood pressure. Some of these underlying differences may explain why women with preeclampsia and gestational hypertension at term seemed to experience stronger effects of air pollution than normotensive women.

Our study had several strengths, including large sample size, which allowed us to study women with specific hypertensive disorders of pregnancy. We used established clinical cut points in pregnancy for our blood pressure categories to ensure our findings would be clinically meaningful.[5,17,18] The modeled air pollution data allowed us to include women even from areas without air monitors. In addition, the models accounted for atmospheric reactions between pollutants, the effects of weather and season on the pollutant levels, and source pollution, rendering a sophisticated model estimate of pollutant mixtures at a given time. The biggest limitation in our study was the use of clinical blood pressure, measured only once during pregnancy. However, we expect that blood pressure was measured with good practice by experienced staff using validated instruments because these measures are routinely taken and can be used in clinical decision-making concerning labor and delivery. Also, because we used data on blood pressure at labor/delivery admission, these measures may be influenced by the pain and stress associated with labor and uterine contractions. Indeed, only 40% of all normotensive women had normal blood pressure on admission to labor/delivery. We believe that these 1-time measurements at labor/delivery are most comparable with blood pressure measured at emergency department visits, which also are prone to be in the high-normal or hypertensive range.[32] One-time hypertensive blood pressure during pregnancy has been shown to increase risk of subsequent hypertension and cardiac events,[15] similar to the effects of high blood pressure measured at emergency department visits,[32] and even high-normal blood pressures are known to increase risk of chronic hypertension.[18] In our study, 0.3% of all hypertensive admission blood pressures among normotensive women could be attributed to small increases in SO_2, whereas 6.5% of all hypertensive admissions were attributable to air toxics. Blood pressures this high could trigger a preeclampsia work-up in normotensive women, which has implications on a population level. Among the 4 million US births per year, at least 28,000 hypertensive admissions and/or preeclampsia work-ups can be attributed to air toxics.

In our large study of women with singleton term deliveries, exposure to several air pollutants in the hours before admission was associated with increased odds of normotensive women having high normal or hypertensive blood pressure. We also found that women with gestational hypertension and preeclampsia had higher blood pressure after air pollution exposure, whereas small or no effects were seen among women with chronic hypertension or superimposed preeclampsia. Acute air pollution exposure may trigger a hypertensive response especially in relation to the stresses of labor and delivery, which may trigger a preeclampsia workup in previously normotensive women, lead to more intense follow-up during labor, and even have long-term implications by increasing risk of subsequent hypertension and cardiac events.

SUPPLEMENTARY MATERIAL

Supplementary materials are available at *American Journal of Hypertension* (http://ajh.oxfordjournals.org).

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DISCLOSURE

The authors declared no conflict of interest.

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