traffic-related air and noise pollution and maternal blood pressure and hypertensive disorders of pregnancy

Introduction

- hypertensive disorders of pregnancy occur in 2–6% of pregnancies
- road traffic is an important source of ambient air and noise pollution, both of which have been associated with adverse birth outcomes and hypertension

- researchers report that exposure to traffic-related air pollution is associated with higher risk for hypertensive disorders of pregnancy

- Identifying the individual and joint effects of these two exposures on cardiovascular health among pregnant women

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The association of traffic-related air and noise pollution with maternal blood pressure and hypertensive disorders of pregnancy in the HOME study cohort

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Methods

- From nine area prenatal clinics, recruited pregnant women between 2003 and 2006 who were: 18 years of age or older; 16 ± 3 weeks gestation; HIV-negative; not taking medications for seizures or thyroid disorders; not diagnosed with diabetes, bipolar disorder, schizophrenia,or cancer; and living in homes built prior to 1978

- 468 pregnant women were enrolled .
- included all women who delivered a live singleton between 2003 and 2006 (n=389) with at least one blood pressure measurement during late pregnancy .
- excluding women who delivered offspring with chromosomal or congenital abnormalities

Traffic-related air pollution exposure

- A diesel-specific traffic signature, determined by elemental source profiles obtained from measurements at cluster sources, was used to quantify the contribution of diesel traffic to PM2.5 concentration

Noise exposure

- Road traffic noise levels were estimated for 1.5m above ground at a 30m grid resolution where the sound pressure level from traffic was 35 dBA or greater

Maternal blood pressure and hypertensive disorders of pregnancy

- The primary outcomes:the two highest maternal SBP and DBP during late pregnancy.

- A secondary outcome in this analysis : risk of hypertensive disorders of pregnancy.

Covariates

- considered the following covariates in this analysis:
- maternal age, race, education, marital status, tobacco smoke exposure, maternal body mass index (BMI), parity, household income, gestation weeks at time of blood pressure measurement, season of conception, and neighborhood socioeconomic status.

Results

Table 1
Summary statistics of hypertensive disorders during pregnancy, road traffic noise (dBA), ECAT concentration (μg/m^3), SBP and DBP (mm Hg) at ≥ 20 weeks gestation according to covariates (The HOME Study, 2003–2006).

Demographic characteristic	Women with HDP	Total	ECAT	Traffic Noise		SBP	DBP
	n (%)[a]	N (%)	Median (25th, 75th)	< 35 dBA n (%)	Median[b] (25th, 75th)	Mean (SD)	Mean (SD)
Overall	35 (9)	370 (100)	0.37 (0.30, 0.46)	226 (61)	47 (39, 54)	115 (13)	69 (9)
Maternal age							
< 25 years	13 (37)	88 (24)	0.41 (0.32, 0.50)	42 (19)	48 (40, 53)	116 (13)	69 (9)
25 to < 35 years	18 (51)	221 (60)	0.35 (0.30, 0.44)	147 (65)	45 (39, 54)	116 (14)	70 (9)
35 + years	4 (11)	61 (16)	0.39 (0.33, 0.48)	37 (16)	50 (43, 57)	114 (11)	68 (8)
Race							
White	14 (40)	231 (62)	0.35 (0.29, 0.44)	157 (69)	45 (39, 52)	115 (13)	69 (8)
Non-white	21 (60)	139 (38)	0.41 (0.33, 0.50)	69 (31)	48 (41, 56)	117 (13)	70 (10)
Education							
High school or less	12 (34)	87 (24)	0.38 (0.30, 0.49)	41 (18)	48 (42, 55)	118 (13)	70 (10)
Some college	9 (26)	95 (26)	0.36 (0.30, 0.47)	57 (25)	47 (38, 52)	114 (12)	69 (9)
Completed college	14 (40)	188 (51)	0.36 (0.30, 0.45)	128 (57)	45 (39, 56)	115 (14)	69 (7)
Marital status							
Unmarried	17 (49)	126 (34)	0.40 (0.32, 0.50)	64 (28)	48 (41, 55)	116 (13)	70 (9)
Married	18 (51)	244 (66)	0.36 (0.30, 0.45)	162 (72)	45 (39, 53)	115 (14)	69 (9)
Household income							
$80,000 +	4 (11)	102 (28)	0.37 (0.32, 0.46)	68 (30)	44 (39, 57)	113 (13)	68 (8)
≥ $40,000 to < 80,000	14 (40)	126 (34)	0.33 (0.29, 0.41)	92 (41)	48 (41, 54)	117 (14)	70 (9)
> $20,000 to < 40,000	4 (11)	63 (17)	0.36 (0.30, 0.47)	33 (15)	45 (38, 52)	115 (12)	68 (9)
≤ $20,000	13 (37)	79 (21)	0.42 (0.33, 0.55)	33 (15)	47 (41, 55)	116 (13)	70 (10)
Maternal tobacco smoke exposure[c]							
None: < 0.015	9 (26)	138 (37)	0.36 (0.30, 0.43)	97 (43)	45 (39, 54)	116 (15)	70 (9)
Secondhand: 0.015–3	22 (63)	195 (53)	0.37 (0.30, 0.46)	107 (47)	47 (39, 54)	115 (12)	69 (9)
Active > 3	4 (11)	37 (10)	0.40 (0.32, 0.49)	22 (10)	46 (40, 55)	115 (14)	70 (9)
Maternal BMI at 16 weeks (kg/m^2)							
< 25	6 (17)	158 (43)	0.37 (0.31, 0.46)	95 (42)	46 (39, 57)	112 (13)	66 (8)
25 to < 30	12 (34)	121 (33)	0.36 (0.30, 0.46)	76 (34)	47 (42, 52)	115 (13)	70 (9)
30 +	17 (49)	91 (25)	0.37 (0.30, 0.47)	55 (24)	47 (40, 53)	121 (12)	73 (9)
Parity							
0	21 (60)	168 (45)	0.37 (0.30, 0.46)	103 (46)	46 (39, 57)	116 (14)	70 (9)
1	5 (14)	114 (31)	0.36 (0.29, 0.46)	69 (31)	46 (39, 52)	114 (12)	68 (9)
2 +	9 (26)	88 (24)	0.37 (0.31, 0.46)	54 (24)	47 (42, 55)	116 (12)	70 (9)
Season of conception							
Spring (March–May)	11 (31)	112 (30)	0.36 (0.30, 0.45)	70 (31)	47 (40, 52)	115 (13)	69 (8)
Summer (Jun- Aug)	7 (20)	89 (24)	0.36 (0.30, 0.43)	63 (28)	46 (39, 52)	116 (13)	69 (8)
Fall (Sept-Nov)	7 (20)	73 (20)	0.39 (0.30, 0.53)	35 (15)	46 (39, 57)	115 (12)	71 (8)
Winter (Dec-Feb)	10 (29)	96 (26)	0.37 (0.32, 0.46)	58 (26)	48 (40, 56)	116 (15)	70 (10)
Neighborhood socioeconomic status							
First (Low)	18 (51)	122 (33)	0.40 (0.33, 0.50)	62 (27)	48 (41, 55)	116 (13)	70 (9)
Second (Medium)	8 (23)	125 (34)	0.35 (0.29, 0.44)	78 (35)	45 (39, 53)	115 (12)	69 (8)
Third (High)	9 (26)	123 (33)	0.36 (0.30, 0.44)	86 (38)	45 (38, 52)	115 (14)	69 (10)
Year residence was built							
1924 or before	11 (31)	122 (33)	0.43 (0.37, 0.52)	64 (28)	47 (39, 56)	117 (14)	70 (9)
1925 to 1955	9 (26)	126 (34)	0.35 (0.30, 0.45)	80 (35)	46 (40, 53)	113 (12)	69 (8)
1956 to 1978	15 (43)	122 (33)	0.33 (0.29, 0.43)	82 (36)	47 (41, 53)	116 (13)	70 (9)

Average SBP and DBP of first highest blood pressure measure collected at ≥ 20 weeks gestation.
Abbreviations: SBP = Systolic blood pressure, DBP = Diastolic blood pressure, HDP = Hypertensive disorders of pregnancy, ECAT = Elemental carbon attributable to traffic. ECAT exposure during pregnancy estimated at residences ~20 weeks gestation. Approximately 10% of women moved residence during pregnancy.

[a] Proportion of women with HDP.
[b] Median (25th, 75th)dBA among traffic noise estimates ≥ 35 dBA.
[c] Maternal tobacco smoke exposure was estimated by the average of serum cotinine (ng/mL)collected at 16 and 26 weeks gestation.

- found a modest correlation between ECAT concentrations and traffic noise ($p < 0.0001$). Women exposed to ≥35 vs<35 dBA of traffic noise were exposed to higher concentrations of ECAT .

- Women living in neighborhoods of lower socioeconomic status and exposed to higher levels of road traffic noise, were also exposed to higher ECAT concentrations compared to women in the middle and high neighborhood socioeconomic groups

- found evidence of a positive monotonic association between ECAT and SBP in late pregnancy

- In early pregnancy, found a positive relationship between traffic noise and SBP

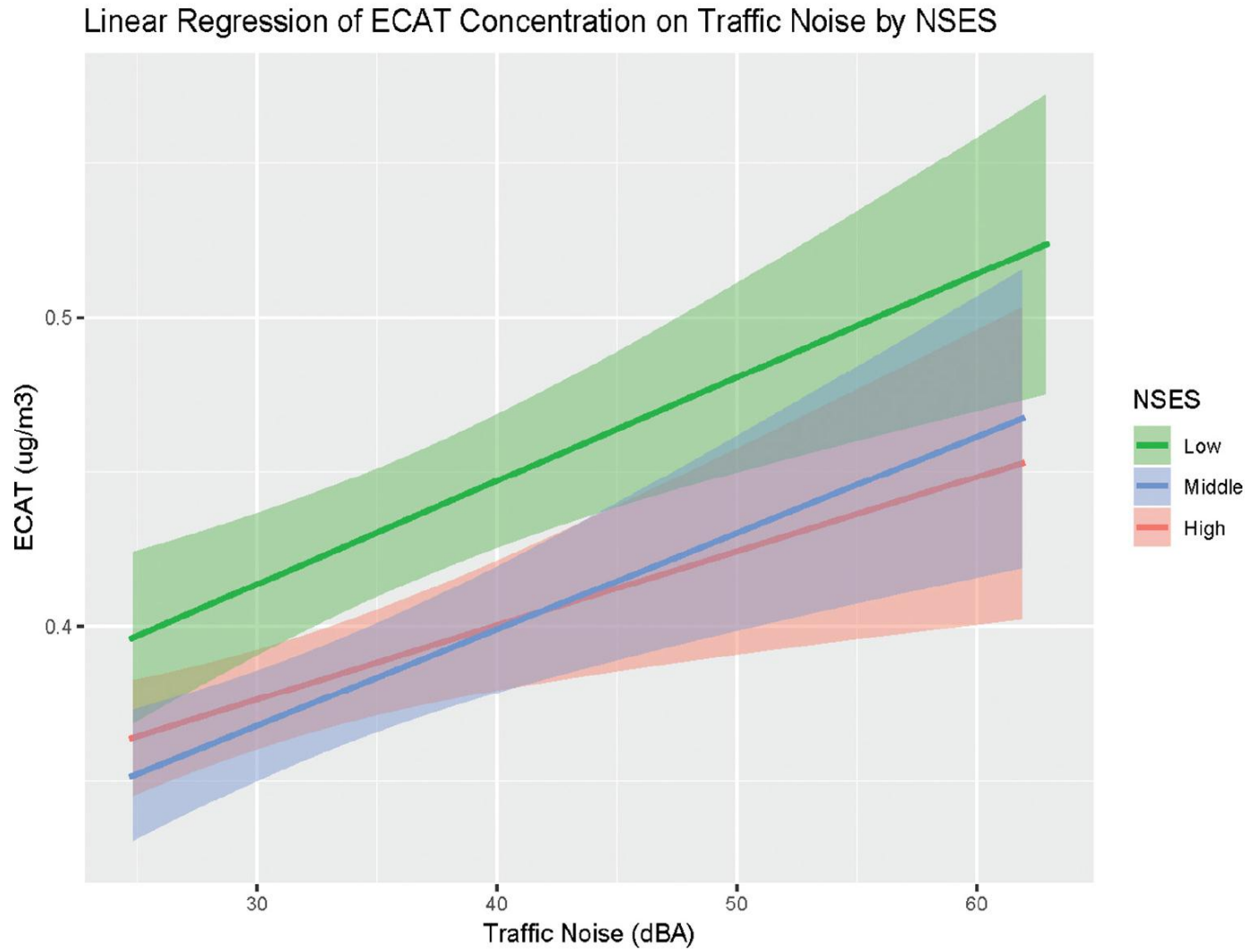

Fig. 1. Linear regression of ECAT concentration (μg/m^3) on traffic noise (dBA) stratified by neighborhood socioeconomic status tercile. Abbreviations: NSES = neighborhood socioeconomic status, ECAT = elemental carbon attributable to traffic. Shaded bands represent 95% confidence intervals.

Table 2
Adjusted differences in SBP and DBP (mm Hg)at ≥20 weeks gestation and risk ratio of HDP across categories of residential ECAT concentrations and traffic noise levels.

	DBP			SBP			HDP		
	Difference (mm Hg)	95% CI	p-Value	Difference (mm Hg)	95% CI	p-Value	RR	95% CI	p-Value
ECAT	0.3	(−0.9, 1.4)	0.63	1.6	(0.02, 3.3)	0.048	0.8	(0.5, 1.3)	0.35
ECAT, adjusted	0.4	(−0.9, 1.6)	0.56	1.9	(0.1, 3.7)	0.035	0.7	(0.5, 1.2)	0.23
Traffic noise	−0.2	(−1.4, 0.9)	0.72	−0.4	(−2.1, 1.2)	0.60	1.2	(0.7, 1.8)	0.52
Traffic noise, adjusted	−0.3	(−1.6, 0.9)	0.62	−1.0	(−2.8, 0.8)	0.28	1.3	(0.8, 2.0)	0.32

Discussion

- In this cohort, found a statistically significant association between residential ECAT concentrations assessed during pregnancy and SBP in late pregnancy.

- This association persisted when also adjusted for traffic noise.

- Exposure to air pollution may impact blood pressure by interacting with the sympathetic nervous system, promoting oxidative stress and circulating cytokines, or altering the vascular endothelium

- traffic noise may impact cardiovascular health by increasing stress hormone concentrations and disrupting sleep patterns

- Previous research has also found a significant relationship between traffic-related air pollution and gestational hypertension risk:
- In an analysis of medical record data from the Consortium on Safe Labor/Air Quality and Reproductive Health Study, each IQR increase in elemental carbon, PM2.5, and nitrogen oxides exposure during weeks 1–20 of gestation was associated with 8%–17% higher risk of gestational hypertension

- An analysis by Pedersen et al. (2017) of data from the Danish National Birth Cohort reported that a 10 dB increase in traffic noise was associated with a small increase in the risk of hypertensive disorders of pregnancy (OR=1.08, 95% CI=1.02, 1.15)(Pedersen et al., 2017).

- Some limitations of this analysis need to be considered:

- First, blood pressure measurements were collected by medical chart review rather than standardized research protocol at specified points during the pregnancy.

- Second, Geocoding errors, housing characteristics and individual time-activity patterns could also contribute to misclassification of exposure, which on average is expected to bias our results towards the null.

- Third, other air pollutants, like nitrogen dioxide, ozone, and carbon monoxide were not considered in this analysis.

Conclusions

- found that residential exposure to traffic-related air pollution during pregnancy was associated with higher SBP during late pregnancy.
- Women who lived in census tracts with lower neighborhood socioeconomic status were exposed to higher concentrations of traffic-related air pollution and traffic noise compared to women in census tracts with higher neighborhood socioeconomic status.
- Future research should assess time points during pregnancy when women may be more susceptible to increases in SBP related to traffic-related air pollution exposure.