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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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ABSTRACT

Traffic-related air and noise pollution may increase the risk for cardiovascular disorders, especially among susceptible populations like pregnant women. The objective of this study was to evaluate the association of exposure to traffic-related air pollution and traffic noise with blood pressure in pregnant women. We extracted systolic blood pressure (SBP) and diastolic blood pressure (DBP) at ≥20 weeks gestation, as well as hypertensive disorders of pregnancy from medical records in the HOME Study, a prospective pregnancy and birth cohort from Cincinnati, OH (n = 370). We estimated exposure to elemental carbon attributable to traffic (ECAT),[1] a marker of traffic-related air pollution, at women's residences at ~20 weeks gestation using a validated land use regression model and traffic noise using a publicly available transportation noise model. We used linear mixed models and modified Poisson regression adjusted for covariates to examine associations of ECAT and traffic noise with blood pressure and hypertensive disorders of pregnancy risk, respectively. In adjusted models, we found a 1.6 (95% CI = 0.02, 3.3; p = 0.048) mm Hg increase in SBP associated with an interquartile range increase in ECAT concentration; the association was stronger after adjusting for traffic noise (1.9 mm Hg, 95% = 0.1, 3.7; p = 0.035). ECAT concentrations were not significantly associated with DBP or hypertensive disorders of pregnancy, and traffic noise was not associated with SBP, DBP, or hypertensive disorders of pregnancy. There was no evidence of a joint effect of traffic noise and ECAT on any outcome. In this cohort, higher residential traffic-related air pollution exposure at ~20 weeks gestation was associated with higher SBP in late pregnancy. It is important for future studies of traffic-related air or noise pollution to jointly consider both exposures and neighborhood characteristics given their correlation and potential cumulative impact on cardiovascular health.

1. Introduction

Hypertensive disorders of pregnancy, including gestational hypertension, pre-eclampsia, and eclampsia, are among the leading causes of maternal mortality worldwide (Lo et al., 2013; Say et al., 2014). The diagnosis of hypertensive disorders of pregnancy, which occurs in 2–6% of pregnancies, requires a measurement of at least two occurrences of systolic blood pressure (SBP) greater than or equal to 140 mm Hg or diastolic blood pressure (DBP) greater than or equal to 90 mm Hg after 20 weeks gestation (Wallis et al., 2008; Lo et al., 2013; Umesawa and Kobashi, 2017). Given the high maternal and fetal morbidity associated with hypertensive disorders of pregnancy and limited treatment options, identification of modifiable risk factors is of significant public health interest.

In urban settings, road traffic is an important source of ambient air and noise pollution, both of which have been associated with adverse birth outcomes and hypertension (Leon Bluhm et al., 2007; Fuks et al., 2011; van Kempen and Babisch, 2012; Lee et al., 2013; Tétreault et al., 2013; Gehring et al., 2014; Hu et al., 2014; Pedersen et al., 2014; Lavigne et al., 2016; Pedersen et al., 2017; Zhu et al., 2017; Honda et al., 2017). Some researchers, using proximity to traffic as a measure of exposure, have not found an association between traffic and

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[1] ECAT = elemental carbon attributable to traffic.

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hypertensive disorders of pregnancy (van den Hooven et al., 2009); however, other researchers studying the relationship between constituents of traffic-related air pollution, such as elemental carbon, fine particulate matter ($PM_{2.5}$), and nitrogen oxides, report that exposure to traffic-related air pollution is associated with higher risk for hypertensive disorders of pregnancy (Pedersen et al., 2017; Zhu et al., 2017; Pedersen et al., 2014). Additionally, the adverse health effects of traffic-related air pollution could be exacerbated by traffic noise pollution, which has also been linked to higher risk of hypertension (Leon Bluhm et al., 2007; van Kempen and Babisch, 2012; Pedersen et al., 2017). Identifying the individual and joint effects of these two exposures on cardiovascular health among pregnant women may help guide regulatory interventions for susceptible populations (Tétreault et al., 2013).

Traffic-related air and noise pollution have both been independently associated with risk for hypertensive disorders of pregnancy, but few studies have specifically assessed the joint association between these exposures and maternal blood pressure (Hu et al., 2014; Pedersen et al., 2014; Pedersen et al., 2017; Zhu et al., 2017). Furthermore, we are unaware of any studies simultaneously evaluating the relationship of both road traffic air and noise pollution with maternal blood pressure in the United States. We are aware of only one other study, conducted by Pedersen et al. (2017), which assessed the association of both traffic air and noise pollution with risk for hypertensive disorders of pregnancy in a Danish cohort (Pedersen et al., 2017).

Accordingly, the objective of this study was to evaluate the independent and joint associations of traffic-related air and noise pollution with maternal blood pressure and hypertensive disorders of pregnancy. We hypothesized that higher exposure to traffic-related air pollution and traffic noise would be independently associated with higher maternal systolic and diastolic blood pressure in late pregnancy, as well as risk for hypertensive disorders of pregnancy.

2. Methods

We used data from The Health Outcomes and Measures of the Environment (HOME) Study, a prospective pregnancy and birth cohort of women and children in greater Cincinnati, Ohio. Detailed information about recruitment and data collection has been published elsewhere (Braun et al., 2017). From nine area prenatal clinics associated with three delivery hospitals, we 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. Approval for the study was obtained from the institutional review boards of Cincinnati Children's Hospital Medical Center and the participating delivery hospitals. Women provided written informed consent after study protocols were explained by trained research assistants.

Overall, 468 pregnant women were enrolled in the HOME Study. For this analysis, we included all women who delivered a live singleton between 2003 and 2006 (n = 389) with at least one blood pressure measurement during late pregnancy (≥20 weeks gestation), excluding women who delivered offspring with chromosomal or congenital abnormalities. Women with missing covariate (n = 10) and outcome (n = 9) data were also excluded from this analysis, resulting in a sample size of 370 pregnant women.

2.1. Traffic-related air pollution exposure

We assessed traffic-related air pollution exposure using a previously validated land use regression model developed for the greater Cincinnati area, as described elsewhere (Ryan et al., 2005; Hu et al., 2006; Le Masters et al., 2006; Brokamp et al., 2017). Briefly, from 2001 to 2006, we conducted 24-h ambient air-sampling of $PM_{2.5}$ on a rotating basis at 24 sites distributed throughout the area where participants lived (Supplemental Fig. 1). After sample collection, gravimetric analysis was used to determine $PM_{2.5}$ concentration. Teflon filters underwent X-ray fluorescence to quantify concentrations of 39 elements, and quartz filters were analyzed by thermal-optical transmittance to quantify elemental and organic carbon concentrations (Ryan et al., 2007). A multivariate receptor model, UNMIX, was used to determine traffic sources contributing to total $PM_{2.5}$ concentration. 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 $PM_{2.5}$ concentration (Hu et al., 2006). For each sampling site, the concentration of elemental carbon attributable to traffic (ECAT) ($\mu g/m^3$), a measure of elemental carbon generated from traffic-related diesel combustion, was calculated (Hu et al., 2006; Sahu et al., 2011). Thus, ECAT serves as our marker for traffic-related air pollution. Spatial variation in ambient ECAT concentration at each participant's residential address around approximately 20 weeks gestation was estimated using a validated land use regression (LUR) model ($R^2 = 0.75$). The LUR model provides estimates of the long-term spatial distribution of ECAT because we developed the model using the arithmetic mean of all 24-hour samples collected at each site (Ryan et al., 2007). We used the ECAT concentration at the residential address as an estimate of maternal exposure to traffic-related air pollution throughout pregnancy.

2.2. Noise exposure

We obtained information on 2014 trends in average noise energy due to road traffic across the Cincinnati region from the U.S. Department of Transportation (DOT), National Transportation Noise Mapping Tool (U.S. Department of Transportation, 2017). Using ArcGIS, version 10.3, we used the CONUS Road Noise Image Service to estimate 24-h average A-weighted road traffic noise levels (Leq) at participants' geocoded addresses around 20 weeks gestation. The DOT road traffic noise model was based on acoustical algorithms and information about vehicle type, speed, and Average Annual Daily Traffic (AADT) from the Federal Highway Administration's Traffic Noise Model. Road traffic noise levels were estimated for 1.5 m above ground at a 30 m grid resolution where the sound pressure level from traffic was 35 dBA or greater (U.S. Department of Transportation, 2017). For the regression analyses, we assigned participants living in areas with < 35 dBA of traffic noise values of 35 divided by the square root of two.

2.3. Maternal blood pressure and hypertensive disorders of pregnancy

The primary outcomes in this analysis were the two highest maternal SBP and DBP measurements during late pregnancy (≥20 weeks gestation), which we extracted from medical charts. We used the two highest SBP and DBP measures because The American College of Obstetricians and Gynecologists (ACOG) diagnostic criteria for hypertension during pregnancy is based on two measurements collected after 20 weeks gestation (American College of Obstetricians and Gynecologists, 2013). For women using hypertension medication prior to pregnancy (n = 20), we added 10 mm Hg to SBP measurements and 5 mm Hg to DBP measurements to adjust for treatment effects (Balakrishnan et al., 2017; Tobin et al., 2005).

A secondary outcome in this analysis was risk of hypertensive disorders of pregnancy. Consistent with our prior study, a participant was considered to have a diagnosis of hypertensive disorders of pregnancy if her medical chart indicated that she had one or more of the following diagnoses: gestational hypertension (n = 13), pre-eclampsia (n = 22), eclampsia (n = 0), or HELLP (hemolysis, elevated liver enzyme levels, and low platelet levels) syndrome (n = 2) (Werner et al., 2015). In addition, participants were considered to have gestational hypertension if they had two blood pressure measurements collected at ≥20 weeks gestation and the SBP and DBP measurements met at least one of the