



Environmental noise pollution and risk of preeclampsia[☆]

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ABSTRACT

Background: Environmental noise exposure is associated with a greater risk of hypertension, but the link with preeclampsia, a hypertensive disorder of pregnancy, is unclear.

Objectives: We sought to determine the relationship between environmental noise pollution and risk of preeclampsia during pregnancy.

Methods: We analyzed a population-based cohort comprising 269,263 deliveries on the island of Montreal, Canada between 2000 and 2013. We obtained total environmental noise pollution measurements (LA_{eq24} , L_{den} , L_{night}) from land use regression models, and assigned noise levels to each woman based on the residential postal code. We computed odds ratios (OR) and 95% confidence intervals (CI) for the association of noise with preeclampsia in mixed logistic regression models with participants as a random effect, and adjusted for air pollution, neighbourhood walkability, maternal age, parity, multiple pregnancy, comorbidity, socioeconomic deprivation, and year of delivery. We assessed whether noise exposure was more strongly associated with severe or early onset preeclampsia than mild or late onset preeclampsia.

Results: Prevalence of preeclampsia was higher for women exposed to elevated environmental noise pollution levels ($LA_{eq24h} \geq 65$ dB(A) = 37.9 per 1000 vs. <50 dB(A) = 27.9 per 1000). Compared with 50 dB(A), an LA_{eq24h} of 65.0 dB(A) was not significantly associated the risk of preeclampsia (OR 1.09, 95% CI 0.99–1.20). Associations were however present with severe (OR 1.29, 95% CI 1.09–1.54) and early onset (OR 1.71, 95% CI 1.20–2.43) preeclampsia, with results consistent across all noise indicators. The associations were much weaker or absent for mild and late preeclampsia.

Conclusions: Environmental noise pollution may be a novel risk factor for pregnancy-related hypertension, particularly more severe variants of preeclampsia.

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1. Introduction

A growing number of studies suggest that environmental noise pollution is associated with a higher risk of hypertension (Münzel et al., 2014). In a recent meta-analysis, an increase of 5 dB(A) of road noise raised the chance of hypertension by up to 3.4% (van Kempen and Babisch, 2012). Environmental noise pollution is a stressor and affects well-being (Babisch, 2011; Hammer et al., 2014;

Münzel et al., 2014). Studies indicate that exposure to noise can result in increased blood pressure, and that noise evokes secretion of adrenalin, noradrenaline, and cortisol, endocrine hormones that cause vasoconstriction and have a cardiovascular impact (Babisch, 2011; Hammer et al., 2014; Münzel et al., 2014). Moreover, noise is associated with fragmented sleep providing an additional route leading to hypertension (Münzel et al., 2014).

Few studies have investigated the effect of noise in women, despite the possibility that pregnant women may be more sensitive to environmental stressors, particularly those prone to hypertensive diseases of pregnancy such as preeclampsia. Preeclampsia is defined as hypertension with blood pressure 140/90 mmHg or more with clinically significant proteinuria or evidence of other end

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organ involvement (Mol et al., 2016), and is associated with onset of hypertension later in life (Auger et al., 2017). The relationship between noise and risk of preeclampsia has however received limited attention. A recent study of 72,745 Danish women raised the possibility that road traffic noise was associated with mild and early onset preeclampsia, but associations were nonsignificant when models were adjusted for traffic related air pollution (Pedersen et al., 2017). Studies of small samples of women in the workplace are inconsistent, with some reporting no association between occupational noise and pregnancy-related hypertension (Haelterman et al., 2007; Wergeland and Strand, 1997), and others a weak association (Irwin et al., 1994; Saurel-Cubizolles et al., 1991). In light of the conflicting evidence, we sought to determine the association between environmental noise pollution and risk of preeclampsia for a large sample of women in an urban centre in Canada.

2. Materials and methods

2.1. Population

We undertook a retrospective cohort study of all deliveries on the island of Montreal between 2000 and 2013. We extracted pregnant women from the Maintenance and Use of Data for the Study of Hospital Clientele database, which includes discharge abstracts for all hospital deliveries in Quebec, Canada (Ministry of Health and Social Services, 2017). Each abstract includes up to 26 diagnostic codes for medical disorders at the time of delivery. Over 99% of women deliver in hospital in Quebec (Auger et al., 2016), thus we had a nearly representative sample of all pregnancies in Montreal during the study period. We did not consider women whose pregnancies did not reach at least 20 weeks, as preeclampsia is rarely diagnosed before this time (Magee et al., 2014). Moreover, we excluded women with hypertension prior to pregnancy, such as unspecified or preexisting hypertension, and hypertensive heart and renal disease (ICD-9 401–405, 642.0–642.2, 642.7, 642.9; ICD-10 I10–I15, O10, O11, O16).

2.2. Preeclampsia

We identified cases of preeclampsia using International Classification of Diseases (ICD) diagnostic codes (ICD-9 642.3–642.6; ICD-10 O13–O15). These codes include gestational hypertension, which the ICD no longer distinguishes from preeclampsia, and eclampsia (O15), a severe form of preeclampsia characterized by convulsions (Paré et al., 2014). We measured preeclampsia as a binary variable (yes/no), and further determined the severity (mild, severe, no preeclampsia) using diagnostic codes. During the study mild preeclampsia consisted of 300 mg or more of protein in urine during a 24-h period, and severe preeclampsia 3000 mg or more (Magee et al., 2008, 2014). However, exact measures of proteinuria were not available. We included eclampsia in the category of severe preeclampsia as eclampsia was rare and could not be evaluated separately.

We determined the onset time of preeclampsia, including early (<34 weeks of gestation) or late (\geq 34 weeks) (Raymond and Peterson, 2011), using the gestational age at admission derived from the discharge abstract. We separated early from late preeclampsia following evidence that onset before 34 weeks of gestation is due to abnormal placentation, whereas late preeclampsia is more strongly related to factors such as diabetes, obesity, and life habits (Raymond and Peterson, 2011). These two forms of preeclampsia may be separate disease entities that result in similar symptoms of hypertension and proteinuria.

2.3. Noise exposure

We obtained A-weighted total outdoor noise levels (LA_{eq24h}), day-evening-night equivalent noise levels (L_{den}), and nighttime noise levels (L_{night}) from land use regression models for Montreal (Ragetti et al., 2016). Land use regression models were developed using total environmental noise samples collected from 204 sites across Montreal during two consecutive weeks in summer 2010 and five consecutive weeks in spring 2014 (Ragetti et al., 2016). For L_{den} , a 5 dB(A) correction was applied to sound levels between 7 and 10 p.m., and a 10 dB(A) correction was applied between 11 p.m. and 6 a.m. to account for greater sensitivity to noise during these times of day (Ragetti et al., 2016). We used the noise samples to estimate annual sound levels for residential six digit postal codes in land use regression models with road, air and railway traffic, population density, building characteristics and vegetation as predictors (Ragetti et al., 2016). Noise estimates were analyzed as a mean exposure during the study for each postal code in Montreal. Postal codes typically correspond to one block face, and cover a small geographical area, thus maximizing the accuracy of the noise estimates. We assigned noise levels to each woman based on the residential postal code at the time of delivery.

We evaluated each noise indicator as a continuous variable. We did not assume linearity, and used splines to characterize associations over the entire distribution of noise exposure. We specified knots at the 5th, 50th, and 95th percentiles following recommended cutpoints for splines (Durreleman and Simon, 1989).

2.4. Environmental characteristics

We considered two characteristics of the physical environment as potential confounders, including exposure to air pollution and neighbourhood walkability.

2.5. Pollution

We obtained three indicators of ambient air pollution, including daily concentration of ozone (O_3) in ppb, nitrogen dioxide (NO_2) in ppb, and fine particulate matter with diameter less than 2.5 μm ($PM_{2.5}$) in $\mu g/m^3$. These three pollutants have been associated with preeclampsia in literature to date (Hu et al., 2017; Pedersen et al., 2017). Daily pollutant data were collected by Environment Canada from 31 monitoring stations distributed around Montreal. We averaged the daily values to obtain citywide concentration over the first 20 weeks of pregnancy for each woman. We expressed indicators of air pollution as continuous variables in splines for more accurate adjustment, with knots at the 5th, 50th, and 95th percentiles (Durreleman and Simon, 1989).

To better account for spatial variation in air pollutants, we used postal code level measures of NO_2 for 2006, obtained from land use regression models based on information such as road traffic and satellite-derived concentrations of pollutants (Weichenthal et al., 2017), in a sensitivity analysis restricted to deliveries between 2004 and 2008.

2.6. Walkability

We accounted for neighbourhood walkability, as physical activity and lifestyle characteristics have the potential to confound the association between noise and hypertension (Irwin et al., 1994; Rabi et al., 2011). This is particularly relevant for late onset preeclampsia, which is thought to be more closely linked with lifestyle factors associated with obesity and diabetes (Raymond and Peterson, 2011). We obtained a composite score of walkability for local neighbourhoods consisting of residential census

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