Noise Exposure during Pregnancy: Influence on Adverse pregnancy outcomes

Ruihan Guo*
Shanghai International Studies university Litai-college Alevel Center
*Corresponding author: guanghua.ren@gecademy.cn

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Abstract: There is growing evidence that noise exposure is not merely an annoyance; like other forms of pollution, it has wide-ranging adverse health, social, and economic effects. This paper systematically reviews the published evidence on the relationship between adverse pregnancy outcomes and noise exposure. Results showed that there was an association between noise exposure and adverse pregnancy outcomes, further high-quality studies are needed to establish this association in the future.

1. Introduction
1.1 The factors of leading to preterm birth

Adverse pregnancy outcomes (APO) include pregnancy-induced hypertension (PIH) including gestational hypertension (GH) and preeclampsia (PE), gestational diabetes (GD), preterm birth (PTB), intrauterine growth restriction (IUGR), low birth weight (LBW), and fetal loss (FL) including spontaneous abortion (SA) and stillbirth (SB). APOs are important events determining maternal and neonatal morbidity and mortality. A common finding in patients undergoing apo is the combination of genetic, immune and environmental factors [1].

Noise from the environment, occupation or living environment is considered to be a source of stress, and there is sufficient evidence to show the effects on hearing impairment, hypertension and ischemic heart disease, worry, sleep disorder, decreased academic performance, cardiovascular effects and sleep disorders. Evidence is limited for other effects, such as changes in the immune system and birth defects. There is no doubt that noise and other occupational and environmental conditions are a common source of stress for mothers, which can induce various physiological and psychological changes that may affect pregnancy [2, 3].

There is strong evidence that exposure to ambient air pollution during pregnancy is associated with an increased risk of infant death and reduced birth weight, However, there is evidence that it is not consistent with preterm birth (PTB) and stillbirth. Road traffic is the main cause of air pollution, especially in urban areas, but it is also a source of noise. Adverse health effects associated with road traffic noise (independent of air pollution) include hypertension and cardiovascular consequences, so noise may affect PTB and stillbirth [4, 5].

The purpose of this review is to systemically summarize the published evidence of reproductive results, such as preterm birth (PTB), low birth weight (LBW), spontaneous abortion, stillbirth and so on, and to provide guidance and suggestions for further study of adverse pregnancy outcomes.

1.2 Noise and Preterm birth (PTB)

PTB is defined as birth before 37 weeks of gestation by the World Health Organization (WHO). About 85% of these births are moderate (32–33 weeks) to late preterm babies (34–36 weeks), 10% are very preterm babies (28–31 weeks), and 5% are extremely preterm babies (<28 weeks). and the prevalence ranges from 5% to 18% of births worldwide. According to WHO, every year, an estimated 15 million babies are born preterm (before 37 completed weeks of gestation), and this number is rising [6].
A recent search of the National Library of Medicine database for adverse health effects of noise revealed over 5000 citations. As the population grows and as sources of noise become more numerous and more powerful, there is increasing exposure to noise pollution, which has profound public health implications. Noise, even at levels that are not harmful to hearing, is perceived subconsciously as a danger signal, even during sleep [7].

There is relatively little literature on ambient noise and PTB. A recent systematic review and the only meta-analysis on road traffic noise and PTB to date found that the quality of evidence was very low and did not show any significant impact of noise on the whole (OR 1.00 (0.79, 1.27) per 10 db(A)) or when limited to air pollution adjusted estimates (OR 1.00 (0.79, 1.26) per 10db(A)).

All births (N = 687,147, PTB=33712) occurring during 2006–2010 to mothers residing within middle layer super output areas (MSOAs) completely within the M25 were extracted from the UK National Births/Stillbirth registers supplied by the Office for National Statistics (ONS), to investigate the correlation between noise exposure and preterm birth, which further suggests a possible relationship between long-term traffic noise and an increased risk of preterm birth[9].

A time-series study to assess the impact of mean daily diurnal (Leqd) and nocturnal (Leqn) noise levels (dB(A)), maximum and minimum daily temperatures (°C) on the number of births with PB in Madrid across the period 2001-2009. The mean Leqd value was 64.6 dB(A) [10,11], with a daily maximum value of 69.0 dB(A), while the mean Leqn value was 59.4 dB(A), with a daily maximum value of 67.5 dB(A) noise values were exceeded on 45% of days and 100% of nights across the period analyzed. Their results for preterm birth suggested that noise levels had an impact in two ways, namely: Leqd in week 21 (second trimester), midway through the pregnancy; and Leqn, with a statistically significant association in week 36 (third trimester) of pregnancy. This study showed that special mention should be made of the effect of noise, not only because it acts continuously across the entire pregnancy, but also because it does so in an acute form in its role as a trigger of labor process. However, the study has some limitations, such as maternal confounding factors.

2. Noise and Low birth weight (LBW)

The World Health Organization (WHO) defines LBW as a birth weight of less than 2,500 grams. LBW continues to be an unfinished agenda because it is one of the poor pregnancy outcomes; it is a good indicator of the health of an infant and is a principal factor that determines the infant's physical, survival, and mental growth. It also indicates the past and present health status of the mothers that have caught the attention of WHO [12, 13]. Previous studies have shown negative associations between birth weight and environmental exposures such as air pollution, noise, temperature or lack of green space[14].

A cohort study comprises 31,458 singleton pregnancies from six European birth cohorts based in nine cities, evaluate the association of the urban exposome, which includes the built environment, air pollution, road traffic noise, meteorology, natural space, and road traffic indicators (60 exposures covering 24 environmental indicators) with birth weight, a statistically significant correlation between exposure and fetal growth and a non significant correlation for future meta-analysis were reported[15]. A national cohort study of 857010 infants proved that an increased risk of low birth weight was found in relation to occupational noise exposure during pregnancy in mothers who had worked full time during pregnancy with <21 days (median) leave of absence (OR=1.44 ,95% CI:1.01–2.03)) [16].

Noise exposure is one of the stressor that activates the sympathetic and endocrine systems through the HPA axis. During pregnancy, the HPA axis of the mother and fetus is integrated by the placenta. During long-term stress, these systems function can be three independent neuroendocrine systems with continuously high cortisol levels. These systems may overload and inhibit the placenta from converting active cortisol to inactive cortisol, thereby transferring the effects of hormone unchanged cortisol hormone on the fetus, resulting in reduced growth [17].
3. Noise and Fetal loss (Spontaneous abortion (SA) and Stillbirth (SB))

Spontaneous abortion is the commonest complication of pregnancy[18]. Wang et al. investigated recurrent spontaneous abortions in a case-control study, performed in China and they found noise exposures of more than 6 hours in a residential area was associated with recurrent spontaneous abortion (OR = 5.3, 95% CI 1.0–28.2) [19].

Metal industry workers in Valjevo (Western Serbia) exposed to occupational noise demonstrated a significant number of natural miscarriages. Thus, the noise provoked a constant stress with utero-placental and foeto-placental circulatory disorders resulting in unwanted abortions [20]. Stillbirth is usually characterized as fetal loss over 20 weeks gestation and occurs in 1 in 160–200 pregnancies. Noise exposure during pregnancy was related with antepartum fetal death (OR = 1.9). Another large study in China, investigating 1,875 perinatal deaths and birth defects across 29 hospitals in Shanghai, showed that occupational noise exposure increased the risk of chromosomal anomalies and antepartum fetal death [21, 22]. Animal experiment explored the effects of construction noise on mouse gestation and neonatal growth, Only 1 of the 245 pups born to the control group of 24 mice was stillborn. In comparison, more pups were stillborn when mice were exposed to noise during the first (P = 0.016), second (P = 0.024), or third (P = 0.031) week of pregnancy [23].

There are few published studies on stillbirth and long-term exposure to road traffic noise (or any other transportation noise), Some studies even contradict themselves. In Madrid, a time series study of late fetal death, including late stillbirth, found an increased risk associated with exposure to air pollutants and temperature at specific 2nd and 3rd gestational weeks, but not noise [24].

High levels of noise activate the neuroendocrine response system and increase corticosterone levels in rodents. Corticosteroids have a direct effect on estrogen and progesterone levels. Estrogen and progesterone in turn differentially regulate inflammatory cytokine IL1α and IL6 expression and secretion, which directly affect mouse blastocyst implantation. Increases in IL1α and IL6 expression as a result of noise-induced elevations in corticosterone levels, eventually lead to stillbirth [25, 26].

4. Conclusions

Although more than a century has passed and great medical progress has been made in the prevention of maternal and infant mortality, a question remains an important topic: what is the best strategy to reduce the incidence of preterm infants and improve neonatal outcomes?

High noise exposure during the critical period of pregnancy is a potential stressor, which may increase the risk of preterm birth (PTB), low birth weight (LBW), and fetal loss (FL) including spontaneous abortion (SA) and stillbirth (SB) and so on. Early studies on the association between noise and pregnancy outcomes mainly involved aircraft noise exposure, while recent studies mainly focused on highway and railway traffic noise exposure. Recent studies also tend to better address the combined effects of noise, air pollutants and temperature.

Pregnancy is a physiological state characterized by increased hypothalamic pituitary adrenal (HPA) axis function, and the serum levels of stress hormones, including cortisol and adrenocorticotropic hormone (ACTH), gradually increase after 12 weeks of pregnancy. Noise may lead to activation of the sympathetic nervous system leads to increased catecholamine secretion, which has received much less attention than pressure triggered HPA axis activation. Blood pressure and uterine reactivity may increase by releasing the pressure of maternal catecholamines, thereby reducing placental function and leading to adverse pregnancy outcomes. Even, noise can directly affect the fetus.

However, the number of studies on noise and adverse pregnancy outcomes is still small, so there is an urgent need to further study noise and pregnancy outcomes from different sources. Studies should be conducted in different environments to show consistency and focus on the overall pregnancy outcome, including abortion, fetal growth or congenital malformations.
Reference


