INTRODUCTION

Air pollution is currently one of the main public health problems, affecting the health of human beings, animals, and plants. The rapid technological advances of the modern world has resulted in an increase in the quantity and variety of pollutants eliminated in the atmosphere, affecting the quality of life on the planet. Exposure to air pollutants has shown to be associated with several deleterious effects to health, even at levels considered safe by environmental legislation. Ambient airborne particulate matter (PM) is one of the leading preventable threats to global health.

In newborns, measurements of upper body skin-fold thickness may provide an estimate of the total body fat of the infant. Postnatal fat accumulation occurs predominantly in the extremities; hence triceps (upper arm) skin-fold thickness provides an indication of the peripheral body fat mass, and sub scapular (upper back) skin-fold thickness reflects the subcutaneous/visceral fat. Fifteen million babies are born premature (less than 37 weeks) worldwide each year. Low birth weight (less than 2500 grams at birth) affects more than 20 million infants worldwide, representing 15% of all births. Differences in newborn body composition and the subsequent increased risk of adverse cardiometabolic outcomes has been linked to both newborn’s size and prenatal exposure to ambient air pollution.

Exposure to Pollutants

During the past two decades, the chemical characteristics and the geographic distribution of sulfur oxide and particulate pollution have been altered by control strategies, specifically taller stacks for power plants, put in place in response to air pollution regulations adopted in the early 1970s.

While the increasing stack heights have lowered local ambient levels, the residence time of SOx and particles in the air have been increased, thereby promoting transformation to various particulate sulfate compounds, including acidic sulfates. These sulfate particles constitute a large fraction of the total mass of smaller particles. Researchers have previously reported associations between exposure to air pollution during pregnancy and decreased birth weights. However, in any given location there is usually very little variation in air pollutant concentrations over short time periods, barring events such as wildfires and other seasonally influenced sources of pollution. It has therefore been difficult to pinpoint a particular window of time during gestation when an exposed foetus might be particularly susceptible to air pollutants.

Air Pollution and Birth Defects

At present, the evidence on the relation between outdoor air pollution and birth defects is limited to only one report. The results evaluated the effect of CO, NO2, O3, and PM10 on the occurrence of birth defects in Southern California for the period 1987–1993. The average monthly exposure for each pollutant throughout pregnancy was calculated. Dose-response patterns were observed for CO exposure in the second month of gestation and ventricular septal defects (AOR for the highest vs. lowest quartile of exposure, 2.95; 95% CI, 1.44–6.05) and for exposure to O3 in the second month and aortic artery and valve defects (AOR, 2.68; 95% CI, 1.19–6.05). Given the lack of studies on air pollution and birth defects, the evidence base available so far is insufficient to draw conclusions about causality.

The possible impact of CO, NO2, O3, and PM10 on premature birth was seen in Southern California. After adjustment for a number of biologic, social, and ethnic covariates, premature births were associated with CO and...
PM10 in the first gestational month and during late pregnancy. The RR associated with PM10 during the first gestational month was 1.16 (95% CI, 1.06–1.26); exposure in the last 6 weeks of gestation was associated with an RR of 1.20 (95% CI, 1.09–1.33). The association of premature birth with CO levels was not consistent throughout the study area.13

**Air Pollution and Low Birth Weight**

The potential effects of air pollutants on birth weight were first examined in a small case–control study by Alderman; the study did not find any relationship between neighbourhood ambient levels of CO during the third trimester of pregnancy and LBW. A graded dose–effect relationship was found between maternal exposure to SO2 and TSP during the third trimester and infant birth weight. The authors speculated that SO2 and particles, or some complex mixtures associated with these pollutants, during late gestation contributed to the LBW risk in the studied population.14

Numerous epidemiological studies have documented the correlation between PM exposure and low birth weight.15 Studies found out that increased ozone exposure throughout pregnancy, increased CO exposure throughout the first trimester of pregnancy, and increased PM10 exposure during the third trimester of pregnancy are all associated with lower birth weight. Although tobacco smoke and PM exposure are not equitable, the similarities in effect are worthy of consideration. A study in Seoul, South Korea demonstrated that exposure to elevated levels of CO, PM10, SO2, and NO2 between the second and fifth months of pregnancy contributed to LBW.16 Studies revealed that pregnant women exposed to high concentrations of PM2.5 have children with LBW, particularly exposure during the third-trimester.

Pollutant clusters with elevated NO2, NO, and PM2.5 concentrations exhibited increased log odds of TLBW, and those with low PM2.5, NO2, and NO concentrations showed lower log odds of TLBW. The spatial patterning of pollutant cluster effects on mothers, combined with between-pollutant correlations within pollutant clusters, imply that traffic-related primary pollutants influence pollutant cluster TLBW risks. Furthermore, contextual clusters with the greatest log odds of TLBW had more adverse neighbourhood socioeconomic, demographic, and housing conditions.17

**Air Pollution and IUGR**

IUGR is defined as birth weight below the 10th percentile of birth weight for gestational age and sex. IUGR is an interesting end point that may predict functional changes in adulthood, such as hypertension and coronary heart disease. A significantly increased risk of giving birth to a child with IUGR was established for mothers who were exposed to PM10 levels > 40 µg/m3 or PM2.5 > 27µg/m3 during the first month of gestation. The adjusted odds ratio (AOR) associated with a 10-µg/m3 increase in PM10 was 1.25 (95% CI, 1.08–1.56); a similar, although weaker, association was seen for PM2.5. There was no association between IUGR and particulate levels in later gestational months or with SO2, NOx, or O318. The studies reviewed above indicate that ambient air pollution is inversely associated with a number of birth outcomes. This is a relatively new area of environmental epidemiology; most reports have emerged over the last 10 years. Low birth weight was associated with maternal marital status and gender of the newborn. Despite the predominance of male births, the prevalence of underweight was higher in female infants. Regarding the type of delivery, cesarean birth appears with a lower chance of insufficient weight; however, there was no statistically significant result in the present study. Similar results were observed in their study, and hypothesised that as lower weight gain may be associated with a lower socioeconomic level, women belonging to this socioeconomic stratum19.

**CONCLUSION**

The primary effects of prenatal pollutants exposure affect particularly BW and other parameters of fetal biometry. There is strong evidence regarding the impact of PM and NO2 on wheezing and respiratory infection in infancy but not on the role of the pollutants in the induction of asthma.

There is a need for additional prospective cohorts that begin in early pregnancy with follow-up to the children’s first years of life that may provide information on causal relationships. The main aspects to be clarified include the role of specific pollutants, prenatal windows of vulnerability, and the importance of confounders as socioeconomics status and nutrition. Health impact/risk assessors should consider these limitations and future well-designed pregnancy cohort studies are needed to aid understanding of these important issues.

On the basis of this review, we suggest several priorities for future research.

First, it remains to be confirmed that the effects on birth weight, prematurity, and IUGR are genuine and causal. Second, it is important to identify the most vulnerable period of exposure in pregnancy. Third, the contribution of different pollutants needs to be established.

Fourth, the biologic pathways require further clarification. And finally, with the increasing attention to the life course, it would be interesting to examine whether early exposures and impaired reproductive outcome have any long-term consequences in later life.

**REFERENCES**


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