Traffic-related air pollution: an avoidable exposure to improve respiratory health

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Respiratory disease taken as a whole is a major global burden contributing significantly to morbidity and mortality at all stages of life. According to WHO estimates, asthma is the most prevalent respiratory condition with over 300 million people affected globally and an additional 100 million diagnoses forecast by 2025. The second most prevalent condition is COPD, with an estimated 210 million suffering globally. Pneumonia and acute lower respiratory illnesses are the leading causes of death in children under 5 years of age, responsible for an estimated 1.4 million deaths annually. It is currently understood that low lung function is a major risk factor linking all these entities, increasing susceptibility to these global health problems.

Lung function is known to track throughout childhood,[1 2] so to a very real extent the lung function one is born with is a major determinant of future respiratory health.[3–5] However, lung function may grow better[6] or less well[7–9] than anticipated depending on environmental exposures, especially those occurring in early life. Airway branching pattern develops during the first 16–18 weeks of fetal development, with alveolar development beginning later and continuing in early postnatal life. Thus, the respiratory system is vulnerable to adverse environmental exposures in both prenatal and postnatal life. The most compelling data for prenatal exposures influencing lung function at birth[10] and beyond[11] are for fetal exposure to tobacco smoking, especially from the mother smoking during pregnancy. However, other prenatal exposures to environmental toxicants are thought to limit respiratory development in utero, as exemplified by maternal exposures to household chemicals during pregnancy.[12] There are two potential pathways by which prenatal exposures might result in low lung function at birth; by direct effects on lung development or via effects on somatic growth of the fetus. Birth weight has a small but significant direct effect on adult lung function, independent of factors known to influence adult lung function, including parental and personal heights and tobacco smoking.[13] However, to date, the direct effects of prenatal exposures on lung function are presumptive as no study has been conducted using a technique that directly measures airway calibre or airway mechanics at or soon after birth.

Morales *et al*[14] report on the effects of intrauterine and early postnatal exposure to ambient air pollution on lung function in preschool-aged children (mean age 4.5 years). They have used sophisticated modelling to estimate individual exposures to benzene and nitrogen dioxide to indicate exposure to traffic-related pollution and found significant reductions in FEV_1 with both pollutants. A dose–response relationship was demonstrated with the risk of low lung function, defined as FEV_1 <80% predicted, increasing with increasing pollutant exposures. In addition, Morales *et al*[14] were able to demonstrate that the effects of exposure were most pronounced during the second trimester. These data are credible and consistent with previous reports of maternal exposures to ambient air pollution decreasing fetal somatic growth[15] and infant lung function measured during tidal breathing.[16] Hansen *et al*,[15] using less sophisticated modelling of pollution exposure than used by Morales *et al*,[14] also showed that effects of exposures varied during gestation; however, in their study the impacts were more variable. There are also numerous reports of the adverse effects of ambient air pollution, either from traffic-related or industrial sources, on lung function in older children.[9 17 18] What the study from Morales *et al*[14] adds to the literature is the increased precision of the exposure estimates and the age of the children studied. They provide convincing evidence that prenatal exposures to traffic-related pollution have long-term effects on lung function in otherwise healthy children.

Despite the value of this study, it does have limitations. The model validation used passive samplers distributed over the study area, rather than in the individual children's homes and the exposure estimates were averaged over various periods (annual, pregnancy and each trimester) to represent mean levels for each period. There are no data on which to judge whether such mean levels better represent the potential for 'damage' than peak levels, the number of occasions above a threshold value or some type of integrated exposure, such as area under an exposure–time curve. Clearly personal monitoring in a study of the scope (both in time frame and in numbers of subjects studies) undertaken by Morales *et al*[14] would not be possible and they have probably done as much as they could. Another limitation is the use of spirometry in preschool-aged children. This test is not easy to perform in young children and indeed, as shown in their Table 2, Morales *et al*[14] were only able to obtain reproducible spirometry in fewer than 50% of the children tested. Another issue with spirometry in this age group is the physiological interpretation of FEV_1. The utility and validity of standard spirometry performed during maximal forced expiration are that expiratory flow limitation is induced so that the flow obtained is independent of effort and, as such, reflects the mechanical properties of the airways and lungs. However, young children are unlikely to be able to maintain flow limitation to very low lung volumes; this is likely to be one of the reasons for the normal fall in FEV_1/FVC ratio reported through childhood.[19] The FEV_1/FVC ratio reported by Morales *et al*[14] of 93%–94% is normal for this age range,[20] but very different from the 80% that is taken to be the lower limit of normal in older children or adults. Young children's lungs are essentially empty within 1 s and thus FEV_1 and the FEV_1/FVC ratio need to be interpreted differently in young children. Morales *et al*[14] may have been better to have measured lung function with the forced oscillation technique, which has both a higher success rate in young children and measures the mechanical properties of airways and lungs with a similar physiological interpretation in all ages, with the exception of infants measured via a face mask where the impedance of the nasal pathways is also included.

Despite the study limitations, Morales *et al*[14] should be lauded for producing convincing data in a large number of children highlighting the long-term consequences of exposure to traffic-related pollution and the lifelong increased risk of respiratory disease. This exposure is potentially avoidable and can be influenced by regulation and legislation. Policy makers need to heed data such as those presented by Morales *et al*[14] as limiting exposure to traffic-related pollution during fetal

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development and early postnatal life is one way that the burden of respiratory disease can be decreased.

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PRESS RELEASE

THORAX

Exposure to traffic pollution during pregnancy can damage future child's lungs

Policies to cut exposure to traffic air pollution could prevent damage to future children's lungs, say researchers

[Intrauterine and early postnatal exposure to outdoor air pollution and lung function at preschool age Online First doi 10.1136/thoraxjnl-2014-205413]

[Traffic-related air pollution: an avoidable exposure to improve respiratory health Online First doi 10.1136/thoraxjnl-2014-206227]

Women who are exposed to traffic pollution while pregnant are increasing the chances of damaging the lungs of their unborn children, concludes a study published online in the journal **Thorax**.

Exposure to pollution during the second trimester of pregnancy in particular raises the risk of harm to a child's lungs, underlining the multiple public health benefits of policies to reduce exposure to air pollution, say researchers.

Existing research has often highlighted the adverse effects of air pollutants on lung function in school-age children and adolescents, but the effects of a mother's exposure to pollution on the lung function of her unborn child and shortly after birth are less well known.

Researchers led by Dr Eva Morales of the Centre for Research in Environmental Epidemiology (CREAL), an ISGlobal research centre, Barcelona, Spain, set out to examine the association of exposure to air pollution during specific trimesters of pregnancy and postnatal life with lung function in preschool children.

Using data from the INfancia y Medio Ambiente (INMA) Project – a population-based mother–child cohort study set up in several geographic areas in Spain – the researchers assessed lung function with spirometry (measuring of breath).

From the 1,295 women enrolled in the study at the beginning of pregnancy, the researchers obtained data on exposure to both air pollution and lung function assessment at 4.5 years old for 620 (48%) of their children.

Nitrogen dioxide (NO_2) is a widely used marker of traffic-related air pollution, and benzene levels can reflect industrial activities and are considered as a surrogate for a mixture of predominantly traffic-driven pollutants. Both were used as indicators of pollution in the areas in which the women lived.

Analysis of the results showed that exposure to higher levels of benzene and NO_2 in pregnancy was associated with reduced lung function parameters in breathing tests.

The volume of breath that has been exhaled at the end of the first second of forced expiration, known as the FEV1 (forced expiratory volume) was –18.4 mL for benzene and −28.0 mL, for NO_2 in women exposed to pollution during the second trimester of pregnancy. FEV1 is a marker of airway obstruction.

Children whose mothers lived in a high traffic air pollution area for benzene during the second trimester of pregnancy had a 22% higher risk of impaired lung function than those living in less polluted areas.

The risk for children of mothers living in a high traffic air pollution area for NO_2 during their second trimester was 30% higher than those from less polluted areas.

Stronger associations between higher levels of pollution around pregnant women and poorer lung function in their subsequent children appeared among allergic children and those of lower social class.

However, there was no significant evidence of an association between early postnatal life (during the first year of life), recent and current exposures to outdoor air pollutants with lung function at preschool age.

The researchers conclude: “Results suggest that exposure to traffic-related air pollutants acting during the prenatal period could adversely impact the developing lung.

“Public policies to reduce exposure to traffic-related air pollution may avoid harmful effects on lung development and function with substantial public health benefits.”

In a linked editorial, Professor Peter Sly, deputy director of the executive of the Queensland Children’s Medical Research Institute, University of Queensland, Brisbane, Australia says the study presents convincing evidence that prenatal exposure to pollution has long-term effects on lung function of children.

“Policy makers need to heed data such as those presented by Morales et al as limiting exposure to traffic-related pollution during fetal development and early postnatal life is one way that the burden of respiratory disease can be decreased.”

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