

Early-life exposures to environmental tobacco smoke and indoor air pollution in the Drakenstein Child Health Study: Impact on child health

Lower respiratory tract infections (LRTIs) are the leading cause of childhood morbidity and mortality in South Africa (SA). Despite sustained efforts to decrease this, including better access to vaccination and strengthening of primary healthcare services, childhood LRTIs continue to impact significantly on child health.^[1,2]

SA, a middle-income country, has undergone much social and political change in the past two decades, resulting in urban migration and the mushrooming of peri-urban communities with subsequent health, education and environmental challenges.^[3] Despite an increase in electrification, many households continue to rely on alternative fuel sources for cooking and heating.^[4] Burning of alternative fuels (such as paraffin, wood, coal and other biomass substances), often in inadequately ventilated homes, contributes to indoor air pollution, a recognised risk factor for respiratory disease.^[5] Further, environmental tobacco smoke exposure continues to be problematic despite anti-smoking legislation.^[6]

The Drakenstein Child Health Study (DCHS), an SA birth cohort study of 1 000 mother-child pairs, longitudinally investigates the epidemiology, risk factors, aetiology and long-term outcome of childhood diseases, including respiratory illnesses.^[7] The study site is in a peri-urban, poor socioeconomic community in the Drakenstein subdistrict, 50 km from Cape Town. Pregnant women were enrolled from two public primary healthcare clinics, Mbekweni (serving a predominantly black African population) and Newman (predominantly mixed-ancestry population), and all deliveries occurred at Paarl Hospital. Children are followed up until at least 5 years of age.

The impact of indoor air pollution (IAP) and environmental tobacco smoke (ETS) exposure on child health was investigated in the DCHS. To measure exposures comprehensively, two home visits, one in the antenatal period (third trimester) and the second postnatally (between 4 and 6 months of the infant's life), were conducted to assess the home environment and to measure the most common indoor air pollutants and byproducts of combustion. Devices placed in participants' homes measured exposure to particulate matter (PM₁₀), carbon monoxide (CO), nitrogen dioxide (NO₂), sulphur dioxide (SO₂) and volatile organic compounds (VOCs).^[8] Measurements of IAP were obtained from 863 antenatal and 723 postnatal home visits, providing important SA data on IAP and potential sources of pollution. Measured benzene (VOC) levels were significantly above acceptable SA ambient standards,^[9] and together with CO and NO₂, increased levels were associated with fossil fuel use.^[8]

Tobacco smoking by pregnant women is often under-reported globally, although household ETS exposure may be high.^[10,11] In the DCHS, urine cotinine measures were used to validate maternal self-reported smoking and exposure.^[12] Tobacco smoking and exposure was found to be highly prevalent, with a smoking prevalence of >50% in mixed-ancestry mothers. Alarming, 18% of infants were born with urine cotinine levels in keeping with active smoking, while a further 30% had levels indicating passive smoke exposure.^[12] The impact of the exposures on birth outcomes was significant, with antenatal maternal smoking associated with lower birth weight.^[12]

The timing of environmental exposures on the subsequent development of LRTI in infancy has not been well described. Most interestingly, we found that antenatal exposures were the main risk factors associated with LRTI, with maternal smoking in pregnancy or PM₁₀ exposure most strongly associated with LRTI. Further, maternal smoking in pregnancy or antenatal passive smoke or PM₁₀ exposure was associated with wheezing in infants.^[13] Interestingly, toluene, a volatile organic compound, was a novel exposure associated with severe LRTI requiring hospitalisation.^[13]

Environmental exposures therefore had a substantial impact on child health and on LRTI. The effect on LRTI of antenatal compared with postnatal exposure suggests an *in utero* developmental lung effect. This study highlights antenatal and early life as a critical period for lung development. Urgent and effective smoking cessation programmes as well as public health interventions focusing on IAP are required. Women of childbearing age, pregnant women and children in poor communities represent vulnerable populations at risk for long-term health effects of these exposures. Early-life LRTI and environmental exposures have increasingly been associated with the development of chronic obstructive pulmonary disease in adulthood. Further longitudinal study of this cohort will provide important information on the long-term respiratory outcomes.

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