Commentary: Could biomass fuel smoke cause anaemia and stunting in early childhood?

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Worldwide, traditional biomass fuels remain the principal source for cooking and heating of homes; these fuels account for about one-third of energy consumption in developing countries. Serious indoor air pollution as a result of burning biomass fuels with inadequate ventilation has been well documented. Several decades ago, Kirk Smith and colleagues demonstrated remarkably high levels of particles in homes in rural India where biomass fuels were being burned, reviewed in Smith (1987). Measured values of indicator pollutants in such environments, including particles and carbon monoxide, are typically many-fold greater than permitted under typical regulatory limits. Epidemiological studies have linked exposure to smoke indoors from biomass fuel consumption to increased risk for acute lower respiratory illnesses (ALRI) in infants and children, to increased risk for chronic obstructive pulmonary disease (COPD) in adults, to increased risk for lung cancer with indoor coal burning, and to low birth weight.5,6

The resulting burden of disease is substantial. Smith et al., as part of the most recent global burden of disease estimates for the World Health Organization, attributed 1.6 million deaths and 38.5 million disability-adjusted life-years (DALYs) in the year 2000 to indoor smoke from solid fuels. The increased risk for ALRI was the dominant contributor, accounting for 59% of premature deaths and 78% of the DALYs. The size of the disease burden reflects the substantial population of exposed children, in fact the majority in many regions of the world, and the strength of the effect of exposure on risk for ALRI, an approximate doubling. These estimates place indoor air pollution from biomass fuels among the leading contributors to the global burden of disease. For India specifically, Smith has attributed 4–6% of the national burden of disease to indoor air pollution.

Mishra and Retherford provide findings from a national survey in India, which suggest that anaemia and stunting in children under 3 years of age may also result from indoor air pollution. The findings are based on a survey of nearly 30,000 children carried out in 1998–99. Weight and height/length were measured, as was haemoglobin and exposure to smoke was classified by types of fuel used in the home for cooking and heating. With consideration of potential confounding, moderately increased risks for moderate-to-severe anaemia and for severe stunting were found for exposed children in comparison with those living in homes where cleaner fuels were used. Perinatal consequences of biomass fuel exposure of the mother have been reported, but this study is the first to show stunting and anaemia during early childhood.

In considering whether the new findings of Mishra and Retherford could reflect a causal association, the possibility of residual confounding merits particular attention. Not surprisingly, the mix of fuels varies by location and socioeconomic characteristics of the household; the biomass smoke-exposed children are more likely to live in rural areas, to be in households with a lower standard of living and to live in lower-quality housing. Their mothers are less likely to have received iron supplementation during pregnancy and to be anaemic at the time of survey. Mishra and Retherford use multinomial logistic regression models in an attempt to control potential confounding by a suite of 20 factors. With inclusion of increasing numbers of potential confounding factors in the models, they find persistent, statistically significant effects of exposure.

Nonetheless, residual confounding may still be contributing to the observed associations. This possibility is indicated by the tendency of the risk estimates for exposure to biomass fuels to drop as more variables are added to the models; for example, the odds ratio for severe stunting is 5:2 in the unadjusted model and 1:84 in the fully adjusted model (Table 4). The extent to which confounding can be controlled depends on accurate measurement of the full set of confounding factors and their inclusion in an appropriately specified model. The reliance on national survey data inherently limits confounder assessment. For example, there is no information on intensity of hookworm or other geohelminth infection, which are critical causal factors for anaemia and malnutrition and highly granular geographically. There is also uncertainty as to the most suitable approach to model the effects of the complex and correlated set of predictors included in these models.

The plausibility of the findings in relation to the effects of inhaling biomass smoke is a further consideration in evaluating causality. Evidence from other settings suggests that biomass smoke exposure can actually increase haemoglobin levels among women with iron deficiency and some studies show that women who smoke have higher haemoglobin levels as well. Positive associations of smoke and CO exposure with increased haemoglobin are plausible, as the reduced oxygen delivery would prompt an increase in erythropoietin production and mobilization of iron reserves for erythropoiesis. Similarly, long-term biomass smoke exposure may cause significant lung damage due to inflammation, possibly leading to mild hypoxia and an elevated erythropoietic response. Whether such adverse effects in young children can also affect their growth to the degree reported in this study is unknown.

The observational evidence on the adverse pulmonary effects of biomass fuel smoke exposure is extremely strong and consistent and suggests that a substantial portion of the adverse health burden in low and middle income countries

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is caused by such exposures. The findings of Mishra and Retherford indicate another set of outcomes that need exploration in further research. However, there is already sufficient evidence to motivate interventions, as Smith and others have long proposed and that have been tried recently in Guatemala. A wave of intervention studies is needed to learn how to best reduce an avoidable cause of disease in children and adults.

References