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Where there's smoke... there's tuberculosis

Stephen Gordon, Jamie Rylance

Recent observations have demonstrated precisely the increased life expectancy associated with clean environmental air in the USA.¹ This dramatic health benefit has been achieved at levels of pollution very much lower than those found indoors in underdeveloped countries. Traditional cooking fires using biomass (organic material) fuel are inefficient and the smoke levels measured in poorly ventilated homes are often 100 times that regarded as dangerous in the affluent industrial world. High levels of indoor air pollution have been associated in epidemiological studies with upper and lower respiratory tract acute infections (ALRIs), chronic obstructive pulmonary disease (COPD) and lung cancer.² There has been less evidence to support an association of indoor air pollution with biomass fuel use and tuberculosis, and so the report by Kolappan and Subramani (*see page 705*) in this issue is most welcome.³

Three billion people use biomass fuel globally, almost entirely in areas with high rates of tuberculosis. The confounding effects of poverty, crowding, malnutrition and increased exposure make an association between biomass fuel use and tuberculosis very hard to dissect. In a well-conducted study in Chennai, India, the authors report that 36% of proven tuberculosis cases can be associated with biomass fuel usage. Confounding factors including cigarette smoking, alcohol use, socioeconomic status and case-contact are

also examined. This study is likely to underestimate the true burden of tuberculosis transmission as young children are intensely exposed to biomass smoke in the home and are a very difficult group in which to diagnose tuberculosis.

Given the association of ALRIs with biomass smoke exposure, and the association of cigarette smoking with tuberculosis, the finding of this study is not surprising. Two major unanswered questions are immediately provoked. First: what is the mechanism behind this association? Secondly: what can be done to protect biomass smoke-exposed adults and children in areas with high tuberculosis transmission?

The mechanism by which biomass smoke exposure results in increased tuberculosis disease is likely to relate to established effects on alveolar macrophages and pulmonary epithelium. Smoke particle size, form and surface chemistry initially promote a proinflammatory state and oxidative damage to the lung. Laboratory models have demonstrated upregulation of tumour necrosis factor α (TNF α), interleukin 6 (IL6) and IL8, nuclear factor- κ B (NF- κ B) activation and cellular lipid peroxidation. Apoptosis of alveolar macrophages is also increased, and, although this is usually considered a positive host defence response, cytotoxic effects may predominate. Phagocytosis is reduced in some non-tuberculosis bacterial studies, and pulmonary mycobacterial load is increased by diesel exhaust particulates. Peak respiratory burst activity may be decreased, but inducible nitric oxide synthase (iNOS) expression is

upregulated. There is no clear narrative to this disruption, and many as yet uninvestigated possibilities. As the epidemiological evidence becomes stronger, there is a need to strengthen mechanistic explanations using relevant in vitro models of biomass particulates.

More importantly to the 7 million people per year who are diagnosed as having tuberculosis in underdeveloped countries, what can be done to prevent this burden of disease? The answer here can be stated with more certainty. Multiple new technologies exist to improve fuel efficiency and domestic ventilation, even in the most resource-deprived situations (www.hedon.info). At the Partnership for Clean Indoor Air Forum in Kampala in March 2009 (www.pciaonline.org), 265 non-governmental organisations (NGOs) re-asserted their intention to improve indoor air quality not only because of the environmental imperative to do so, but also because of the probable health benefits of improved stove technology.

As chest physicians, we look forward to the results of the RESPIRE trial from Guatemala, due to be published this year, which is expected to show for the first time a direct health benefit from an indoor air intervention. A stove intervention alone, however, will not reduce the global burden of tuberculosis.

Reducing biomass smoke-related disease will require multifaceted action to improve fuel security, health education (particularly for women), diet and environmental conservation coupled with excellent tuberculosis case management.⁴ These are exciting and challenging times.

Competing interests: None.

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