

Biomass Fuels for Cooking: Clear that Smoke

To the Editor:

I read with interest the recent article by Regalado and colleagues, wherein the authors have assessed the impact of cooking with biomass fuels (BMFs) on respiratory symptoms and lung function among rural women in a developing country (1). They found the presence of increased respiratory symptoms (predominantly an increase in phlegm) as well as a decline in spirometric indices, both of which correlated with the duration of exposure. These findings reinforce the observations of previous studies done at my institute that BMFs have deleterious effects on respiratory functions of nonsmoking individuals. However, two important aspects related to cooking with domestic fuels merit attention.

First, it is worthwhile to note that exposure to kerosene (another commonly used fuel for domestic cooking in rural areas that do not have access to liquefied petroleum gas [LPG]) also leads to an increase in respiratory symptoms. In fact, increase in respiratory symptoms was maximal with the use of mixed fuels (both BMFs as well as kerosene) compared with either of the two alone (2, 3). These effects occur not only in adults but also in children (4). Moreover, patients with preexisting obstructive airway disease tend to have an increase in respiratory symptoms as well as worsening lung functions after exposure to domestic cooking fuels (5). Second, in addition to obstructive airway disease, exposure to BMFs and mixed fuels can lead to bronchial hyperresponsiveness as well as restrictive defects on spirometry (6, 7). Combined exposure to tobacco smoke and domestic cooking fuels (like BMFs and mixed fuels) leads to much more morbidity than either of them alone (2, 7).

In developing countries, and especially in rural areas, using LPG for cooking is often not possible either due to economic constraints or lack of access to the same. The need to use BMFs and other alternate fuels for domestic cooking is compounded by the absence of proper ventilation in houses, and this enhances the adverse effects of such fuels. The key to avoiding the deleterious effects of these fuels (on respiratory symptoms and function) is to advocate the use of smokeless devices for cooking as well as to improve the ventilation in houses, especially in the area where domestic cooking is done. After all, prevention is better than cure.

Conflict of Interest Statement: N.S. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

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From the Authors:

We thank Dr. Navneet Singh for his interest in our recent article (1). Dr. Singh's comments related to the role of kerosene exposure on respiratory symptoms of adults and children are certainly plausible. In our survey, we explored the use of multiple cooking fuels, including liquefied petroleum gas (LPG), petroleum, charcoal, biomass, and electricity. In the studied community, and in other areas of Mexico, the use of kerosene as cooking fuel is very uncommon. This may explain why in our study only cooking with biomass stoves was associated with reports of increased phlegm production and the slight decrease of lung function. We did not analyze the presence of bronchial hyperresponsiveness among participants, but it is clear that women with chronic obstructive pulmonary disease (COPD) due to wood smoke exposure have this and, at least in one study conducted at our institute, the degree of hyperresponsiveness was greater in patients with COPD exposed to biomass than in those exposed to tobacco smoke (2).

At the time of the survey, tobacco smoking was uncommon in women (3%), and we decided to analyze the impact of cooking with biomass stoves in nonsmoking women. Therefore, we were unable to study the impact of cooking with biomass stoves in smokers. However, it is now more common to see women with COPD who cooked with biomass stoves and were smokers. We should increase public awareness of the health impact of traditional cooking with solid fuels and other polluting fuels, as it is very unlikely that poor communities will change to cleaner fuels in the near future. The alternative is the dissemination of improved biomass stoves and better house ventilation as Dr. Singh suggests.

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Mice Running Late: T-Cell Kinetics in Models of Asthma

To the Editor:

We read with interest the article by Drs. Wenzel and Holgate discussing the shortcomings of mouse models in asthma research (1). The authors highlight the profound differences in anatomy and airway remodeling between murine and human lungs. They also speculate on differences in Th1/Th2 immune pathways between the two species. To their conclusions we would like to add that there are crucial differences in the immunology of "asthma" between men and mice. In a study that compared the

kinetics of allergen-induced leukocyte infiltration into human and murine airways, T-cell kinetics were completely different in the two species (2). While T-cell recruitment to human airways peaks within 24 h after allergen challenge, and precedes the peak of eosinophil infiltration, which occurs several days after allergen challenge, T-cell infiltration into murine airways peaks 14 d after allergen exposure, at a time point when eosinophil numbers in the airways have returned to baseline (2).

T cells are currently considered as key regulators of allergic airway inflammation, and it is assumed that they orchestrate the recruitment and the activation of effector cells such as eosinophils (3). This would fit the findings that T-cell infiltration precedes eosinophil infiltration in human asthma. However, the very late infiltration of T cells into murine airways is not only in contrast to human asthma but also in contrast to current concepts on the role of T cells in asthma. We do acknowledge that we cannot exclude that certain T-cell subsets might be involved in the orchestration of some aspects of airway inflammation in mice. However, the profound differences in the kinetics of T cells

have strongly dampened our enthusiasm for mice as a meaningful tool in asthma research.

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