Published in final edited form as:

Clin Chest Med. 2012 December ; 33(4): 649–665. doi:10.1016/j.ccm.2012.08.003.

Indoor fuel exposure and the lung in both developing and developed countries: An update

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Synopsis

Almost 3 billion people worldwide burn solid fuels indoors. These fuels include biomass and coal. Although indoor solid fuel smoke is likely a greater problem in developing countries, wood burning populations in developed countries may also be at risk from these exposures. Despite the large population at risk worldwide, the effect of exposure to indoor solid fuel smoke has not been adequately studied. Indoor air pollution from solid fuel use is strongly associated with COPD (both emphysema and chronic bronchitis), acute respiratory tract infections, and lung cancer (primarily coal use) and weakly associated with asthma, tuberculosis, and interstitial lung disease. Tobacco use further potentiates the development of respiratory disease among subjects exposed to solid fuel smoke. There is a need to perform additional interventional studies in this field. It is also important to increase awareness about the health effects of solid fuel smoke inhalation among physicians and patients as well as trigger preventive actions through education, research, and policy change in both developing and developed countries.

Keywords

Biomass; Solid fuel; COPD; Asthma; Lung cancer; Respiratory tract infection

I. INTRODUCTION

There are four principal categories of indoor air pollution - combustion products, chemicals, radon, and biological products. This article focuses on the respiratory health effects of pollutants from combustion of various types of indoor fuels, which is currently a major public health problem in the world.

Types of Indoor fuels

Indoor fuels include solid, liquid and gas fuels (Table 1). Solid fuels include biomass and coal. Biomass fuel refers to any living or recently living plant and/or animal-based material

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Disclosure Statement

The author has no relationship with a commercial company that has a direct financial interest in the subject matter or materials discussed in the article or with a company making a competing product.

that is deliberately burned by humans as fuel such as wood, twigs, dried animal dung (e.g., cow dung), charcoal (a product of incomplete burning of wood), grass, or agricultural crop residues (e.g., corn husk, straw, and bagasse - biomass remaining after processing sugar-cane). Coal, as distinct from charcoal, is a naturally occurring fossil fuel formed from preserved compressed and partially metamorphosed organic material. Coal includes 'smoky' coal (bituminous coal) and 'smokeless' coal (anthracite coal). Liquid fuel includes kerosene and liquefied petroleum gas (LPG). Gas fuels include methane and natural gas. LPG and natural gas, in addition to electricity, are widely viewed as clean fuels. The most important determinant of the choice of fuel in a given region of the world is its cost, giving rise to the 'energy ladder' depicted in Figure 1. Worldwide, wood is the most common solid fuel used, although coal is predominantly used in China and dried cow dung is commonly used in rural South Asia ¹. The primary focus of this review is indoor solid fuels.

Burden of solid fuel-related adverse effects

In 2007 worldwide, approximately 42% of all households and 76% of all rural households utilized solid fuels (Figure 2) ². Most solid fuel users are poor, and live in developing countries. Smoke that emanates from the household combustion of solid fuels is thus the most widespread traditional source of indoor air pollution on a global scale. It should however be pointed out that solid fuel, primarily wood, is also used in developed countries - 28% of a cohort based in New Mexico, United States reported wood smoke exposure in a 2010 report ³. Poorly maintained stoves are an important contributor to indoor air pollution in developed countries as well. Thus, solid fuel use is prevalent in all inhabited continents of the world. Because almost 3 billion people worldwide are exposed to solid fuel smoke, the population at risk worldwide for adverse respiratory effects is very large.

Use of indoor solid fuel

In developing countries, a variety of solid fuels are used primarily for cooking purpose in unvented, inefficient and leaky but inexpensive stoves ^{4,5}. These stoves typically consist of simple arrangements such as a few stones or a U-shaped hole or a rounded pit and operate under poorly ventilated conditions ⁵⁻⁷. In developed countries (as well as in developing countries with cold climates), primarily wood is used in home fireplaces and stoves for heating indoors (Figure 3) ^{8,9}.

Contents of indoor solid fuel smoke

Indoor solid-fuel smoke contains a complex mixture of a large number of pollutants ^{10,11} that include respirable particulate matter (PM), carbon monoxide, oxides of nitrogen and sulfur, benzene, formaldehyde, 1,3-butadiene, polycyclic aromatic hydrocarbons such as benzo(a)pyrene, free radicals, aldehydes, volatile organic compounds, chlorinated dioxins, oxygenated and chlorinated organic matter, and endotoxin (the latter is raised particularly in smoke from burning maize crop residue and cow dung ¹).

PM content in indoor solid fuel smoke

PM₁₀ are particles with mass median aero-dynamic diameter of < 10 µm. These particles are easily inhaled and reach the deeper portions of the lung, causing a spectrum of adverse cardiovascular and pulmonary health effects. More recently PM_{2.5} (aero-dynamic diameter <2.5 µm) has been used as a PM metric, reflecting the likelihood that greater toxicity may be attributable to smaller particulates. PM₁₀ values ranging from 10,000 to 20,000 µg/m³ have been reported in households in developing countries ¹² that are well above the U.S. Environmental Protection Agency (EPA) national 24-hour standard for PM₁₀ of 150 µg/m³ ¹³. In households with limited ventilation (as is common in many developing countries),

exposures experienced by household members may be 100 times higher than World Health Organization (WHO) and EPA guidelines.

Further, smoke from the various solid fuels is not alike. Ellegard reported that wood-burning stoves were associated with a significantly higher release of respirable particles ($1,260 \mu\text{g}/\text{m}^3$) compared with either charcoal ($540 \mu\text{g}/\text{m}^3$) or liquefied petroleum gas stoves ($200\text{--}380 \mu\text{g}/\text{m}^3$) during cooking time ¹⁴. Some biomass smokes, such as agricultural crop residue smoke, primarily contain particles that are less than $10 \mu\text{m}$ in diameter and thus may have disproportionately greater respiratory effects than smoke from other biomass fuels ¹⁵.

Comparison of solid fuel smoke with other common combustion emissions

Compared to cleaner indoor fuels (such as kerosene and gas fuels), biomass fuel use is associated with higher concentrations of respirable particulate matter ¹⁶. Biomass smoke is also associated with greater levels of inflammation and oxidative stress in sputum as well as greater DNA damage in buccal epithelium, as compared with the cleaner fuel LPG ¹⁷. Further, the combustion of biomass is qualitatively similar to the burning of tobacco in terms of emissions of particulate matter and gases, although without the nicotine. Additionally, particles from wood smoke are similar to those from traffic emission in their pro-inflammatory potential, although mediated by different particle characteristics. For example, the organic fraction is the most important particle component in the response to wood smoke, whereas this fraction plays a lesser role in the response to traffic-derived particles ¹⁸. Wood smoke particulate matter also generates greater DNA damage than traffic-generated particulate matter per unit mass in human cell lines, possibly due to the higher level of polycyclic aromatic hydrocarbons in the former ¹⁹.

Populations most affected by solid fuel smoke

In developing countries, women and young children have the greatest exposure since they spend the most time near the domestic hearth. Exposure for infants and toddlers is further increased if they are carried on their mother's back while she cooks, a common cultural practice in some regions ²⁰. The greatest burden among adults in these countries is borne by nonsmoking women ²¹, but the process by which solid fuel smoke leads to respiratory disease may begin in early childhood, or even *in utero*.

In cold climates and highland areas, people spend more time indoors where they are exposed to fires that burn over extended periods in homes that may have tight construction for space heating ²². Thus, both pollution levels and exposure times increase, affecting both sexes and children. It is further possible that elderly subjects who spend a majority of their time indoors may also be more vulnerable to the health effects of solid fuel smoke.

Mechanisms by which solid fuel smoke affects respiratory outcomes

There are limited data on the mechanisms by which solid fuel smoke affects the lung (Table 2). Macrophage dysfunction; increased proteolytic activity of matrix metalloproteinases (MMP); greater gene expression of MMP; pulmonary surfactant deactivation; reduced bacterial clearance; and reduced mucociliary clearance have all been reported ^{23–29}. COPD subjects exposed to wood smoke demonstrate an upregulation of arginase activity in their platelets and erythrocytes, which is linked with greater oxidative stress ³⁰. The high levels of oxidative stress related to wood smoke exposure further mediates increased apoptosis in human cells such as pulmonary artery endothelial cells ³¹. In addition, solid fuel smoke also produces DNA damage and inflammatory and oxidative stress response gene expression in cultured human cells ³². Exposure to wood smoke of healthy human volunteers is followed by a rise in serum and urine concentrations of Clara cell protein 16 (CC16), a marker of the integrity of the pulmonary airblood barrier ^{33, 34}.

Lung histopathological effects of solid fuel smoke exposure

Rats exposed to chronic wood smoke develop mild bronchiolitis, epithelial cell hyperplasia and hypertrophy, alveolar septal thickening, and emphysema³⁵. Similarly, rabbits exposed to dried animal dung smoke show respiratory epithelial cell proliferation, alveolar destruction, and emphysema³⁶. Bronchoalveolar lavage obtained from healthy human volunteers acutely exposed to wood smoke reveals a neutrophilic influx³⁷.

Useful Interventions

Intervention studies to modify adverse respiratory outcomes from solid fuel smoke exposure are limited. The most effective way of eliminating exposure to smoke from solid fuels is to switch to cleaner fuels such as electricity, but this option is not always feasible. Other useful interventions reported in the literature for developing countries include the following.

- i. Outdoor relocation of cooking with solid fuels - Women cooking with biomass fuel outdoors are exposed to lower levels of particulate matter as compared with those cooking indoors^{38, 39}.
- ii. Partitioning of kitchen from living space - The presence of partition to the kitchen does not reduce the exposure of the cook, but reduces exposure to other members in the household³⁸.
- iii. Window in kitchen - The presence of an additional window in the kitchen may be associated with lower adverse respiratory effects⁴⁰.
- iv. Improved cook stoves: Improved cook stoves are characterized by a higher efficiency in thermal conversion, a higher heat transfer ratio, and a more complete combustion (and therefore a lower emission of smoke), compared with the “traditional” counterparts. Improved cook stoves may be accompanied by better removal of smoke by the addition of a chimney. In a parallel randomized woodstove intervention trial in highland Guatemala, the intervention group received an improved stove with chimney, the *plancha*, whereas the control group continued using open indoor fires⁶. A 50% mean reduction in 48-hour average personal carbon monoxide exposure levels among children 0–18 months of age was associated with the improved stove intervention⁶.

Outcomes from interventions, such as better stove design and maintenance, have not been adequately studied in developed countries.

II. RESPIRATORY OUTCOMES RELATED TO SOLID FUEL SMOKE EXPOSURE

Indoor air pollution from solid fuel use is strongly associated with premature mortality as well as COPD (both emphysema and chronic bronchitis), acute respiratory tract infections, and lung cancer and weakly associated with asthma, tuberculosis, and interstitial lung disease (Table 3).

II.A. Overall mortality

The WHO in its annual report estimated that in 2004 indoor air pollution from solid fuel use was responsible for 2.7% of the total annual global burden of disease (over 41 million disability-adjusted life years), and 3.3% of annual premature deaths (almost 2 million), primarily in developing countries⁴¹. In that report, indoor air pollution ranked tenth on the examined list of risk factor causes of death globally and first among environmental risk factors (ahead of unsafe water/sanitation/hygiene)⁴¹. Further, indoor smoke from solid fuel caused about 21% of lower respiratory tract infections worldwide, 35% of chronic

obstructive pulmonary deaths, and about 3% of lung cancer deaths⁴¹. Unfortunately, the greatest burden of indoor air pollution–related premature deaths is among children from pneumonia²¹.

II. B. COPD Outcomes

While cigarette smoking is the leading preventable cause of COPD in the developed world, indoor solid fuel smoke exposure may be the leading preventable cause in lesser developed countries, particularly among women⁴². COPD outcomes studied in relation to solid fuel smoke exposure include emphysema, chronic bronchitis and poor lung growth. Longitudinal change in lung function attributable to indoor solid fuel smoke exposure has not been studied.

II.B. i. Emphysema—The odds for developing COPD with biomass fuel exposure is about two to three-fold higher, disproportionately affecting women and young adults. In one cohort of Colombian women, the population attributable risk for wood smoke causing COPD was 50%⁴³.

Cross-sectional studies: Multiple cross-sectional studies in developing countries have established an association between biomass fuel use and chronic airflow obstruction^{44–49}. A prevalence study of COPD (as defined by post-bronchodilator ratio of forced expiratory volume in one second to forced vital capacity or $FEV_1/FVC < 0.70$) in five cities of Colombia found that biomass stove use for 10 years was associated with COPD (O.R. 1.50; 95% CI, 1.22–1.86)⁴⁸. Further, a study from rural Mexico found that biomass stove use was associated with a 2.8% adjusted decrease in FEV_1/FVC ratio⁴⁹. Additionally, a cross-sectional study of smokers in New Mexico in the United States showed wood smoke exposure to be associated with low postbronchodilator % predicted FEV_1 and prevalence of COPD with remarkably similar odds ratios (O.R. 1.96; 95% C.I. 1.52–2.52) as described in the literature originating from developing countries³.

Case-control studies: Multiple case-control studies have consistently found an association between cooking with biomass fuel and airflow obstruction^{43, 50, 51}. These studies are from developing countries, with the exception of a study from Barcelona, Spain⁹. The study from Spain (as well as the cross-sectional study from southwestern United States) suggests that exposed people in developed countries may not be spared from the ill-effects of wood smoke^{3, 9}.

Longitudinal studies: Longitudinal studies of the impact of biomass smoke exposure on the development of COPD are few in number, but one study did follow 520 COPD patients over 7 years in Mexico City to assess their mortality risk⁵². Multivariable survival analysis showed that biomass smoke exposed COPD subjects experienced a similar mortality rate as tobacco smoke-exposed COPD subjects, after adjusting for severity of disease⁵².

Meta-analyses: Three meta-analyses show consistent results between biomass smoke exposure and lung function-diagnosed COPD. Kurmi *et al.* examined 13 COPD studies and showed a 3.0-fold increased risk (O.R. = 2.96, 95% C.I. 2.01, 4.37)⁵³. Hu *et al.* examined 15 studies and showed a 2.4-fold increased risk (O.R. = 2.44, 95% C.I. 1.9–3.33)⁵⁴. Po *et al.* showed a similar risk in women, using six studies (O.R. = 2.40, 95% C.I. 1.47–3.93, Table 4)⁵⁵. The pooled effect estimates for lung function-diagnosed COPD are generally comparable to those published for chronic bronchitis and higher than those published for doctor-diagnosed COPD⁵³.

Interventional Studies: A Chinese retrospective cohort study found significant reduction in incidence of doctor-diagnosed COPD (relative risk of 0.58 in men, 0.75 in women) in homes where coal was used in improved cooking stoves with chimneys, the effect increasing with time since adoption⁵⁶. There is a need for additional prospective studies and clinical trials to examine changes in incidence and severity of COPD following interventions to decrease solid fuel smoke exposure.

Dose-response relationship: Exposure-response analyses show a positive trend, with correlation between developing COPD and increasing level or duration of exposure to biomass smoke^{9, 43, 48, 51, 57, 58}.

Interaction between host characteristics and solid fuel smoke on COPD: Sex: While solid fuel exposed women apparently are at higher risk for developing COPD in developing countries, exposed men may be at higher risk in developed countries³. There is possibly no sex predilection towards these outcomes. The higher burden of disease in a certain gender in a region likely reflects the sex-related differences in activities that lead to greater exposure. **Hispanic ethnicity** In the New Mexico state in southwestern United States, Hispanic ethnicity may be protective against low lung function and COPD related to wood smoke exposure³. Interestingly, Hispanics of New Mexico, characterized by high Native American genetic ancestry, are also similarly protected from COPD due to cigarette smoke exposure⁵⁹. **Cigarette smoking:** Tobacco use may potentiate the development of COPD among subjects exposed to solid fuel smoke⁵⁵. Epidemiologic studies from developing countries demonstrate a greater prevalence of respiratory symptoms and greater degree of airway obstruction in tobacco smokers concomitantly exposed to biomass smoke than either non-biomass-exposed smokers or nonsmokers exposed to biomass emissions^{54, 60, 61}. A similar additive interaction between wood smoke and cigarette smoke exposures on COPD is described in a study from the United States³.

Differences between smoking-related COPD and biomass-related COPD: Biomass smoke-related COPD is *similar* to tobacco-smoke related COPD with respect to its clinical, physiological and radiological presentation (dyspnea, airway obstruction, air trapping, increased airway resistance, chronic bronchitis, centrilobular emphysema, and pulmonary hypertension); impact on quality of life; mortality rate; histopathological findings (of anthracosis and bronchial squamous metaplasia); and levels of inflammatory cells (neutrophils, eosinophils) and mediators (interleukin-8, MMP-9 and 8-isoprostanes) in induced sputum^{52, 62, 63}. In contrast, Rivera *et al* reported that biomass-related COPD was associated with a lesser extent of emphysema and goblet cell metaplasia but greater fibrosis and pigment deposition in the lung parenchyma and thicker pulmonary arterial intima, as compared to cigarette smoke-exposed COPD⁶⁴.

Epigenetic susceptibility: Wood smoke exposure may be more strongly associated with COPD outcomes in the presence of methylated p16 or GATA4 genes in sputum³. The study did not find that promoter methylation caused or explained away the wood smoke association with COPD. However, promoter methylation enhanced the susceptibility towards COPD among wood smoke exposed subjects.

II.B.ii. COPD-chronic bronchitis—The most common manifestations of domestically acquired particulate lung disease in adults in developing countries are cough-related complaints, specifically chronic bronchitis.

Prevalence: The prevalence rates for chronic bronchitis in communities exposed to indoor biomass smoke is high^{39, 46, 57, 58, 60, 65}. Some of the highest prevalence rates were reported

in rural parts of Nepal (19.8% in nonsmoking women who spent more than 4 hr/day near the fireplace) and Bolivia (22% for all nonsmokers in a village that cooked indoors with cow dung)^{39, 65}.

Strength of association: Users of biomass fuel had approximately two-fold greater rates of chronic bronchitis than those using cleaner fuels such as kerosene or LPG^{60, 66}.

Additionally, a cross-sectional study of smokers in New Mexico in the United States showed wood smoke exposure to be associated with chronic bronchitis with somewhat similar odds ratios (O.R. 1.64; 95% C.I. 1.31–2.06), as described in the literature originating from developing countries³.

Dose-response relationship: In a case-control study of rural non-smoking Mexican women, the odds for chronic bronchitis were directly related to total cumulative exposure to biomass smoke⁵⁸. Thus, a cumulative exposure of 100 hour-years was associated with an adjusted odds ratio of 9.3 while 200 hour-years of exposure was associated with a higher odds ratio of 15.0 for chronic bronchitis⁵⁸. However, since lower exposures may be associated with higher socioeconomic status and more efficient stove design, these studies are limited by their inability to control for these potential confounders. An interesting occurrence in rural Bolivia has provided an unusual opportunity to control for these factors³⁹. Two rural Bolivian villages with no reported tobacco use, identical in every way except for the location of their cooking stoves, were studied³⁹. One village had their kitchens indoors (consistent with traditional Bolivian custom), while the other moved their stoves outdoors 50 years earlier due to the perceived ill effects of smoke inhalation on health. The study recorded a two-fold higher prevalence of chronic bronchitis in the indoor group compared with the outdoor group (22% vs. 13%)³⁹. By controlling for all confounders including socioeconomic status, floor type, and fuel type, this study provides excellent evidence for the pathogenic role for biomass combustion in the development of chronic bronchitis.

Interventional studies: In a parallel randomized woodstove intervention trial of Mayan women in highland Guatemala, the intervention group received an improved stove with chimney, the *plancha*, whereas the control group continued using open indoor fires^{6, 67}. The intervention group reported a significant reduction in the prevalence of chronic respiratory symptoms, especially wheeze (relative risk for wheeze of 0.42, 95% C.I. 0.25, 0.70)⁶⁷.

II.B.iii. Poor lung growth and development—Exposure to biomass smoke from an early age may retard lung growth. Various indices of lung function were lower among adolescent and young adult men and women in Nepal (16–25 years old) who were biomass smoke exposed, compared to those who were not⁴⁷. Similarly, lower lung function were observed in school children aged 10–13 years living in homes with coal stoves in Chengde and Shanghai cities of China⁶⁸ and in 7–12 year old school children exposed to indoor wood stoves in Kuala Lumpur, Malaysia⁶⁹. A retrospective cohort study of 1,036 nineyear old Polish children living in households heated with gas or coal in the first six months of their life, showed that a higher indoor pollution score during the first six months of life was strongly associated with lower lung function at age nine years⁷⁰. These findings suggest an adverse effect of solid fuel smoke exposure on early lung growth. On the other hand, longitudinal decline in lung function has not yet been studied in relation to solid fuel smoke exposure.

II. C. Acute respiratory tract infections

Acute respiratory tract infections (ARI) can be divided into two types - upper respiratory tract infections (AURI) and lower respiratory tract infections (ALRI) of which ALRI is

associated with a greater risk for death. Infants and children living in homes using solid fuels are at increased risk for developing both AURI and ALRI and dying from ALRIs ²³.

Burden Statement—ALRI is a leading contributor to the global burden of disease, accounting for 6.2% of the total disability-adjusted life-years for all ages in the year 2004 ⁷¹. ALRI is the primary cause of death in children under 5 years old globally (accounting for 1.4 million deaths in 2010 in this age group)². Almost half of deaths in this age group from ALRI are attributable to indoor air pollution from household solid fuel use ⁷²

Strength of association—The odds for ALRIs for children exposed to household biomass smoke has been quantified in a number of studies, the majority from developing countries, but also from the United States and Italy ^{50, 73, 74}. Most of these studies have used a case-control design, although several cohort studies have also been done ⁷⁵. The overall estimate of the risk of ALRI from 13 studies selected for the meta-analysis conducted by Smith *et al.* for the WHO comparative risk assessment was 2.3 (95% CI 1.9–2.7) ⁷⁶. The risk was higher among children younger than 2 years (O.R. 2.5). The highest O.R. was found for children carried on their mothers' backs while cooking (O.R. 3.1). An update of this meta-analysis was recently published with a total of 24 studies selected ⁷⁷. The overall pooled OR for ALRI in this updated meta-analysis was 1.8 (95% CI, 1.5–2.2). On the other hand, combining acute upper and lower respiratory tract infections in another meta-analysis, higher OR of 3.5 (1.9, 6.4) were described by Po *et al.* ⁵⁵.

Dose-response relationship—The relationship of the rate of increase in ALRIs on average daily exposure level to PM₁₀ is a non-linear or concave function, with the rate of increase declining for exposures above approximately 1,000–2,000 µg/m³ ⁷⁵.

Mechanism—Impaired respiratory tract defense mechanisms may explain the association between biomass smoke exposure and ARI risk in children ^{25, 50}. Acute exposure to particulate matter reduces mucociliary clearance, resulting in increased residence time of inhaled particles, including microorganisms ^{23, 24}. Animal studies demonstrate impairment in macrophage phagocytic function and surface adherence; and reduction in bacterial clearance ^{25, 26} in response to wood smoke (Table 2).

Interventional Trials—In a parallel randomized woodstove intervention trial in highland Guatemala, the intervention group received an improved stove with chimney, the *plancha*, whereas the control group continued using open indoor fires (Figure 4) ⁶. The intervention group experienced significant and robust reductions for three secondary outcomes – fieldworker-assessed severe pneumonia, physician-diagnosed severe pneumonia, as well as respiratory syncytial virus-negative severe pneumonia ⁶.

II. D. Lung cancer

Although smoking is the major risk factor for lung cancer worldwide, approximately 1.5% of annual lung cancer deaths are attributed to exposure to carcinogens from indoor solid fuel use ⁷⁶. The International Agency for Research on Cancer (IARC) has classified combustion products from coal and biomass fuels as Group 1 and 2A carcinogens respectively (*i.e.* known and probable carcinogens respectively) ⁷⁸. Consistent with the IARC classification, the data suggests a stronger association for coal smoke with lung cancer, as compared to exposure to other biomass fuel smoke, in both animals and humans ^{1, 79}.

Cross-sectional, case-control and retrospective longitudinal studies have examined the association between solid fuel smoke and lung cancer. The majority of the published studies in this field originate from China where 'smoky' (bituminous) coal is widely used for

cooking. After adjustment for smoking and chronic airway disease, the 2004 meta-analysis by Smith *et al.* indicated a two-fold increase in risk among women from indoor coal smoke exposure (O.R. of 1.94, 95% C.I. 1.09–3.47) with a lower risk among men (O.R. 1.51; 95% C.I. 0.97–2.46)⁷⁶. There are fewer studies that examine the risk for lung cancer associated with biomass fuel use. One such study by Behera *et al.* reported an adjusted O.R. of 3.59 (95% C.I. 1.07–11.97) in an Indian population⁸⁰. Results from a pooled analysis among 4,181 cases and 5,125 controls from Europe and North America who reported predominant use of wood fuels in their house also showed that wood smoke exposure was associated with increased risk for lung cancer (O.R. 1.21, 95% CI: 1.06–1.38)⁸¹.

Mechanism—Smoke from solid fuel, particularly bituminous coal, contains high concentrations of carcinogens such as polycyclic aromatic hydrocarbons and benzo[*a*]pyrene and produces high levels of free radicals and DNA damage in cultured human cells³². Further, comparison of various biomass fuels showed that the DNA damage was most significant with animal dung cake¹⁷. The direct genotoxicity of solid fuel smoke may thus contribute to the development of lung cancer.

Although it is unclear if the solid fuel smoke affects the histological distribution of lung cancer among those exposed, a mutation spectrum comprising of TP53 and codon 12 K-ras gene mutations are noted in lung cancers from nonsmokers exposed to ‘smoky’ coal^{82, 83}. This mutation spectrum is consistent with an exposure to polycyclic aromatic hydrocarbons, which are the primary component of the ‘smoky’ coal emissions. In addition, overexpression of the p53 protein is described in coal smoke-related lung cancer tissue^{84, 85}.

Host susceptibility factors—Cigarette smokers may be more susceptible than non-smokers to developing lung cancer related to exposure to indoor air pollutants^{81, 86}. This interaction is plausible, given that both tobacco smoke and solid fuel smoke share similar carcinogenic constituents and together may increase the risk for lung cancer to a greater extent than either exposure alone. Further, there may be genetic and epigenetic susceptibility factors. Thus, gene polymorphisms in Glutathione-S-Transferase family such as GSTM1 null genotype may increase the susceptibility to lung cancer related to both wood smoke and coal smoke exposures^{84, 87}. Additionally, promoter methylation of genes (such as p16 gene) in sputum may be associated with greater odds for lung cancer among individuals exposed to ‘smoky’ coal emissions⁸⁸.

Interventional study—A retrospective cohort study performed on a cohort of over 20,000 farmers from Xuan Wei county of China where ‘smoky’ coal is used for cooking indoors, lung cancer incidence was noted to be almost halved (hazard ratios of 0.59 among men and 0.54 among women) among residents who had changed from unvented firepits to stoves with chimneys⁸⁹.

II.E. Tuberculosis

There is inconsistent evidence that exposure to biomass smoke increases the risk of either acquiring tuberculosis or progression of tuberculosis to clinical disease.

Mechanism—Exposure to wood smoke results in impaired macrophage phagocytic function and surface adherence; reduced mucociliary clearance²³; and reduced bacterial clearance^{25, 26}. These mechanisms may predispose exposed subjects to tuberculosis^{90, 91}.

Strength of Association—A meta-analysis of 10 studies revealed a pooled effect estimate (OR) of 1.55 (95% CI 1.11, 2.18) for tuberculosis disease¹. Earlier meta-analyses had yielded similar results⁹².

II. F. Asthma

Asthma prevalence—Published effect sizes for asthma prevalence in relation to biomass exposure vary considerably. All these studies adopted different techniques to determine asthma prevalence and none measured actual biomass exposure levels. A meta-analysis of four studies showed that exposure to indoor air pollution approximately doubles the risk of developing asthma in children (OR = 1.96, 95% C.I. 1.29, 2.99)¹. On the other hand, another meta-analysis failed to show a significant association either in children or in women (Table 4)⁵⁵.

Asthma severity—Exposure to solid fuel smoke may enhance disease severity among asthmatics. In a small cohort study of children in a boarding house of a metropolitan school in New Zealand, peak levels of air pollution from wood burning were associated with small but statistically significant effects on FEV₁ diurnal variability, morning values of FEV₁, and night time peak expiratory flow rate values in the (doctor-diagnosed) asthmatic students⁹³.

Mechanisms—Several mechanisms are hypothesized for the development of asthma due to exposure to solid fuel smoke. High indoor air levels of nitrogen dioxide and sulfur dioxide have been associated with asthma⁹⁴. In the presence of air pollutants, pollen grains become agglomerated with airborne particles⁹⁵. These agglomerated pollens then release higher levels of eicosanoid-like substances that increase serum IgE, eosinophils and neutrophils⁹⁶. Samuelson *et al* found that particles generated from wood burning had about the same capacity to enhance allergic sensitization as road traffic particles in laboratory animals⁹⁷. Apart from this, biomass smoke contains high levels of endotoxin (especially animal dung smoke) and volatile organic compounds that are risk factors for asthma⁹⁸. Once inhaled, endotoxin stimulates an amplifying series of endotoxin–protein and protein–protein interactions, leading to lung inflammation and oxidative stress, which may contribute to the development of asthma.

II. G. Interstitial Lung Disease (Hut Lung)

Hut lung, an interstitial lung disease characterized by carbon deposition, dust macules, and mixed dust fibrosis, has been reported in case series primarily of women with chronic high-level exposure to indoor biomass smoke in developing countries^{99–102,103}. This disease has however also been described from North Carolina, United States, where it was attributed to a malfunctioning indoor wood-burning heater¹⁰⁴.

Although there are currently no longitudinal studies describing the natural history of hut lung, this disease is best described in a case series by Sandoval *et al* of 30 rural Mexican nonsmoking women in their 60s¹⁰⁰. These women had a mean exposure to biomass emissions of 400 hour-years. Their main symptoms and signs included dyspnea (100%); cough (93%); cyanosis (63%) and inspiratory crackles on auscultation (70%). Arterial blood gas demonstrated hypoxia with a mean PaO₂ of 45 mm Hg and PaCO₂ of 36 mm Hg. Pulmonary function testing usually showed obstruction or mixed obstruction-restriction pattern (although other case series have also described normal lung function⁹⁹ or a restrictive pattern¹⁰³). Abnormal chest radiographs were found in 90% of subjects, with 2–3 mm reticulonodular opacities the most common findings, followed by cardiomegaly. The opacities closely resembled radiographic categories 2p, 2q, 3p, and 3q for rounded opacities and 2s, 2t, 3s, and 3t for irregular opacities seen in pneumoconiosis¹⁰⁵. The vast majority had evidence of cor pulmonale with electrocardiographic evidence of right ventricular hypertrophy in 77% of subjects, and a mean pulmonary artery pressure of 46 mm Hg by right heart catheterization. Bronchoscopy revealed grossly visible anthracotic plaques, usually seen at the bifurcations of lobar bronchi. Histopathologic examination of transbronchial biopsy specimens showed thickened basement membranes with diffuse

deposition of fine anthracotic particles. Open lung biopsy specimens confirmed the presence of diffuse anthracosis and areas of interstitial fibrosis. Other case series have described the fibrosis as bronchiolocentric in distribution ¹⁰³. Although the treatment of this disease is not known, improvement with systemic corticosteroids followed by inhaled corticosteroids has been reported ¹⁰³

III. Health effects of indoor combustion of cleaner fuels

Kerosene

There may also be respiratory effects associated with the indoor combustion of kerosene, a common cooking fuel in many parts of the world, for which emissions and exposures are intermediate between those for solid and for gaseous fuels (Smith 1987), but on which few studies of health effects seem to have been conducted. In one such study, indoor kerosene stoves were associated with low lung function in a cross-sectional evaluation of 7–12 year old Malaysian children ⁶⁹. In another cross-sectional study of Indian women, kerosene users had less symptoms than biomass fuel users (13% vs. 23%) but more than LPG users (8%) ⁴⁴. A similar trend was noted for lung function in that study ⁴⁴.

Gas and electricity

Studies on the respiratory health effects of exposure to emissions from the two major sources of energy used for cooking in developed countries, gas and electricity, have yielded inconsistent results with small effect sizes ¹⁰⁶. Thus, fuels that are highest on the energy ladder do not have the same magnitude of health effects as smoke from solid fuels.

IV. Limitations of the existing literature

There is a need to address the following critical knowledge gaps:

1. Lack of actual exposure measurements in most studies adds to misclassification bias. Exposure assessment in most studies is by self-report and direct measurement of solid fuel smoke exposure is performed in few studies only. Therefore, exposure-response information is also inadequately established in the literature.
2. Few longitudinal studies have been reported. As a result, the precise effect of solid fuel smoke exposures on longitudinal outcomes (such as decline in lung function or lung growth) or on incidence of chronic diseases is not well understood.
3. Few interventional studies have been conducted. As a result, the quantification of the effect of improved stoves on longitudinal outcomes or incidence of chronic diseases cannot be accurately assessed.
4. Small sample size. Studies of small sample size often have insufficient statistical power to reveal possible cause and effect relationships.
5. Inadequate outcome assessment adds to misclassification bias. An example is the use of non-standard definitions of emphysema or chronic bronchitis. Similarly, the diagnosis of asthma is troublesome since objective measures of bronchial hyperreactivity have not been used and, therefore, it is not possible to determine whether solid fuel smoke is associated with a pattern of asthma-like symptoms or true asthma. The lack of a uniformly accepted definition of acute respiratory tract infections may similarly cause misclassification bias that may contribute to the heterogeneity in reporting across different studies.
6. Lack of adjustment for key covariates. Socioeconomic status is an important confounder in these studies since both solid fuel use and diseases such as tuberculosis and COPD are more prevalent in poorer populations. Cigarette

smoking is another important confounder but no objective measurement of smoking was carried out in most studies.

7. Inadequate understanding of the mechanistic bases for these associations and of the role of genetics and epigenetics in affecting individual susceptibility are additional limitations of this field.
8. Solid fuel smoke as a risk factor for COPD and lung cancer is not well-characterized in developed countries. This is due to insufficient research on the adverse respiratory effects of relatively lower levels of exposure that are often considered 'harmless'; the overwhelming interest in cigarette smoking as the major risk factor; the mistaken perception among people in these countries that wood (being natural) is benign; and the inadequate awareness that wood smoke may have major respiratory effects when combined with cigarette smoking.

V. Summary

Smoke from indoor solid fuel combustion for cooking or heating purposes is associated with multiple acute and chronic respiratory conditions. Although indoor solid fuel smoke is likely a greater problem in developing countries, wood burning populations in developed countries may also be at risk for these conditions, especially when these exposures are combined with cigarette smoking. Studies suggest that lower solid fuel smoke exposure may be associated with lower risk for select respiratory outcomes. It is currently unclear if prevention of chronic disease can be achieved by reducing solid fuel exposure and how much reduction in exposure is required to achieve a useful benefit. It is important to increase awareness about the health effects of solid fuel smoke inhalation among physicians and patients and promote preventive initiatives through education, research, and policy change.

Key

ALRI	acute lower respiratory tract infection
AURI	acute upper respiratory tract infection
CC-16	Clara cell protein 16
COPD	chronic obstructive pulmonary disease
DNA	deoxyribonucleic acid
EPA	Environmental Protection Agency
FEV₁	forced expiratory volume in one second
FVC	forced vital capacity
IARC	International Agency for Research on Cancer
LPG	liquefied petroleum gas
MMP	matrix metalloproteinase
PM	particulate matter
WHO	World Health Organization

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Figure 1.

The Energy Ladder. Fuels lower in the energy ladder are less efficient and produce more pollution, but are less expensive. Conversely, fuels higher in the energy ladder are more efficient and produce less pollution, but are more expensive.

Figure 2.
Global Use of indoor solid fuels in 2010, as reported in percent, by the World Health Organization (WHO). Reproduced with permission from the WHO ¹⁰⁷.

Figure 3.

Key differences in solid fuel exposure between developing and developed countries. The picture in the right in the panel was reprinted with permission from Elsevier (The Lancet, 2009; 374:733–743¹⁰⁸).

Figure 4.

Traditional open fire used for cooking (panel A) and the locally developed and constructed chimney woodstove, the *plancha* (panel B) in Guatemala. The chimney woodstove has a thick metal heating surface for cooking tortillas and holes with removable concentric rings for pots, a firebrick combustion chamber with baffling, a concrete and brick body, tile surfaces around the cooking area, dirt and pumice stone insulation, a metal fuel door, and a metal chimney with damper. Infants and toddlers are highly exposed to combustion smoke as they are carried on their mother's back while she cooks, a common cultural practice in Guatemala and other regions. Picture reprinted with permission from Elsevier (The Lancet, 2011; 378:1717–1726) ⁶.

Table 1

List of indoor fuels

Solid fuels

Agricultural Crop Residues

Animal Dung

Wood

Charcoal

Coal

Liquid fuels

Kerosene

Ethanol & Methanol

Liquefied Petroleum Gas

Gas Fuels

Natural Gas

Methane Gas

Electricity

Table 2**Mechanisms by which solid fuel smoke may affect respiratory outcomes**

Neutrophilic inflammation
Macrophage phagocytic dysfunction and surface adherence
Increased MMP activity (pro-MMP-2; pro-MMP-9, and MMP-9)
Greater MMP gene expression (MMP-2 and MMP-12)
Pulmonary surfactant deactivation
Reduced bacterial clearance
Reduced mucociliary clearance
Upregulated arginase activity
Greater oxidative stress
Increased apoptosis
DNA damage
Break in integrity of the pulmonary air-blood barrier.

Key - MMP: Matrix metalloproteinase; DNA: deoxyribonucleic acid

Table 3

Diseases associated with smoke from solid fuel use

<u>Strongly associated respiratory outcomes</u>
COPD- emphysema/chronic bronchitis
Acute respiratory tract infections/pneumonia
Lung cancer (coal smoke)
<u>Weakly associated respiratory outcomes</u>
Asthma
Tuberculosis
Interstitial lung disease
<u>Associated non-pulmonary outcomes</u>
Cataract
Pregnancy-related complications
Nasopharyngeal cancer
Ischemic heart disease
Cor pulmonale

Table 4

Overall pooled odds-ratios for associations between respiratory outcomes and biomass fuel exposure, compared with other fuel type exposures among children and women. Data obtained from meta-analysis of 25 studies by Po et al. published in 2011 ⁵⁵.

Respiratory Outcome	Number of studies included in meta-analysis	Strength of association
Acute respiratory tract infection in children ^{**}	8 [*]	3.53 (1.93, 6.43)
Asthma in children	4	0.50 (0.12, 1.98)
Asthma in women	5	1.34 (0.93, 1.93)
Chronic bronchitis in women	6	2.52 (1.88, 3.38)
COPD in women	6	2.40 (1.47, 3.93)

^{*} All studies included in the meta-analysis are from developing countries except one from the United States that examined acute respiratory infections in American Indian children ⁷³.

^{**} Acute respiratory tract infection in this meta-analysis included both upper and lower respiratory tract infections.