**ABSTRACT:** Over half the world’s population, most from developing countries, use solid fuel for domestic purposes and are exposed to very high concentrations of harmful air pollutants with potential health effects such as respiratory problems, cardiovascular, infant mortality and ocular problems. The evidence also suggests that, although the total percentage of people using solid fuel is coming down, the absolute number is currently increasing. Exposure to smoke from solid fuel burning increases the risk of chronic obstructive pulmonary diseases (COPD) and lung cancer in adults and acute lower respiratory tract infection/pneumonia in children. Despite heterogeneity among different studies the association between COPD and exposure to smoke produced by burning different types of solid fuel is consistent. However, there is strong evidence that while coal burning is a risk for lung cancer, exposure to other biomass fuel smoke is less so. There is some evidence that
reduction of smoke exposure using improve cook stoves reduces the risk of COPD and possibly acute lower respiratory infection in children so approaches to reduce biomass smoke exposure are likely to result in reductions in the global burden of respiratory disease.

**Keywords:** Indoor air pollution, lung diseases, solid fuel, biomass fuel
Introduction
The main activities that contribute significantly to indoor air pollution in low and middle income countries are the use of solid fuels for residential energy, active and passive smoking inside the house, oil mists and fumes from cooking, smoke from burning mosquito coils during the summer and incense sticks during religious rituals, and keeping pets and animals in the same dwelling where individuals live.

Globally, over 3 billion people, approximately half of the world’s population, rely on the use of biomass (wood, crop residues, twigs, shrubs, dried dung and charcoal) and coal, collectively known as solid fuels, to meet their basic domestic energy demands for cooking, lighting and heating [1, 2]. The proportion is even more staggering in rural parts of Africa, Central and South America, and Asia, being over 90% [3]. The majority of the solid fuels are burnt in inefficient traditional cookstoves located in places without adequate ventilation, although in Sub-Saharan Africa burning in the open environment is not uncommon [4, 5]. A large variety of harmful substances are released during combustion of solid fuels and remain in the indoor environment at very high levels for a number of hours after cooking and heating has stopped, because of the lack of adequate room ventilation. Most people spend approximately 90% of their time indoors, even more in the case of women, children, elderly and those with ill health. Cooking in developing countries is often done by women; and mothers tend to keep their young children, especially infants and toddlers, close by during cooking therefore placing women and children at risk of both acute and long term ill health from emissions of solid fuel combustion. The degree of risk is related to the levels of exposure which, in turn, are determined by a number of factors. For instance, kitchen type and the design of living areas in dwellings in developing countries can increase exposure to indoor air pollutants by several fold through natural (but inadequate) ventilation and lack of flues [6, 7]. Seasonality is also important as exposures to indoor air pollutants during the winter are several times higher than in the rest of the year as people spend more time gathering around fires to keep warm.
**Use of Solid Fuel as Household Energy**

Access to clean energy (e.g. electricity, natural gas) is low in developing countries\(^1\), but the situation is even worse in the least developed countries\(^1\) and in Sub-Saharan Africa\(^1\) (Figure 1), where 82% and 89% of the populations, respectively, rely primarily on solid fuels for domestic purposes compared to 56% in developing countries [8]. Variation also exists within these countries. In the least developed countries, as high as 97% of rural population have access only to solid fuels, compared to 73% among the urban dwellers. The difference is even more marked in Sub-Saharan Africa (95% vs. 58%) [8]. Most of the solid fuel users do not limit themselves to a single type of fuel but rather combinations of different types depending upon availability. Worldwide, wood is the most common biomass used, although coal is predominantly used in China while dried cow dung is used by a smaller fraction of the rural South Asian populations.

\(^1\) Based on the classification by the United Nations Development Programme. There are 140 developing countries, of which 50 are the least developed countries and 45 are Sub-Saharan Africa countries. Thirty-one countries belong to both least developed countries and Sub-Saharan Africa categories.
Figure 1: Total population using solid fuels in percentage (with permission from WHO)

**Trends in biomass fuel use**

Although the total population in absolute numbers primarily relying on the use of biomass as residential energy will increase from 2.68 billion in 2009 to about 2.77 billion in 2015, and probably remaining at that level until 2030 [9], the proportion of the total population from developing countries will decrease from 54% in 2009 to 51% in 2015, with further reductions to 44% in 2030. In China the population relying on biomass fuel is readily on the decline and is estimated to fall to 19% by 2030. It is anticipated that this trend will be followed by India after 2020 [9] such that by 2030 just 54% of the Indian population and 52% of other Asian countries will be using traditional biomass fuels. Increase in the price of kerosene and bottled gas in the developing countries is one of the main reasons for this slower transition to cleaner
fuels. However, the pace of decline in Sub-Saharan Africa will be much lower compared to other developing countries.

Factors preventing the use of clean fuel

The influence on the choice of fuel used is multifactorial, but cost and socio-economic status appear to be the main drivers (Figure 2). Less well-off households spend most of their income purchasing food and clothes and for medical expenses. Depending upon the availability of biomass fuels and the distance required to travel to acquire these fuels, those living in the least developed countries can spend on average 2-3 hours weekly in collecting biomass, leaving little or insufficient time for education [2] and work, thus making it very difficult for these families to improve their socio-economic status. In rural areas of developing countries, poor families often receive subsidy on clean fuels. However, many switch back to biomass when they cannot afford even the subsidised fuel, making them reliant on the cheaper but dirtier alternatives. The other important factor is the unavailability of clean fuels in rural areas, because of the lack of a sustainable supply-chain mechanism and/or the necessary infrastructure to deliver clean fuels. Consequently, clean fuel is not available at all or the demand for clean fuel cannot be met consistently, forcing rural dwellers to continue their dependence on biomass fuels.
Characteristics and toxicity of solid fuel use

Traditional stoves burning solid fuels have a very low energy conversion efficiency ranging from 12% to 25% depending upon the types of fuels (Figure 2). Approximately 8 to 10% of the solid fuels undergo partial combustion, often due to inadequate oxygen supply [10]. As a result, one of the main components of biomass smoke is carbon (5-20% of wood smoke as particulate mass) which is found in the particulate fraction of the smoke and is present across a range of particle sizes. Biomass smoke also contains more than 250 organic compounds, varying mainly by the type of fuel burnt and the combustion conditions [11]. Partial oxidation of organic matter generates high levels of carbon monoxide (CO), as well as hydrogen cyanide, ammonia, and nitrogen oxides. In addition, a large number of other toxic and carcinogenic compounds such as polycyclic aromatic hydrocarbons (PAHs) (e.g. benzo[a]pyrene), aldehydes, and free radicals [11] have been demonstrated in biomass smoke. While biomass fuels tend to have low levels of halogenated compounds, they may be
contaminated by chemicals such as pesticides, or mixed with plastics. Certain varieties of
coal, particularly in China, have particularly high fluoride or silica content [12].
Consequently, burning of these specific fuels may lead to production of toxic halogen
compounds, such as hydrogen chloride, phosgene, dioxin, chloromethane, bromomethane and
other halocarbons [13-15].
The toxicity of biomass smoke has been studied extensively in the laboratory. Rats exposed sub-
chronically to wood smoke at concentrations of 1 to 10 mg/m³ over weeks showed reduced CO-
diffusing capacity and increased airway resistance. There were also mild chronic inflammation and
squamous metaplasia in the larynx, alveolar macrophage hyperplasia and slight thickening of the
alveolar septae [16]. Wood smoke can cause greater levels of DNA damage in lymphocytes than
exposure to liquefied petroleum gas combustion products [17] and can both impair macrophage
function and be mutagenic [11].

Wood smoke condensates may damage the lens in rats, causing discolouration and opacities, probably
through oxidation by polycyclic aromatic compounds and metal ions [18] while chemicals such as
aldehydes and acrolein found in biomass smoke can cause eye irritation.

Biomass smoke is pro-oxidant and burning of biomass fuel may generate high redox active
components. In one study from India the oxidative potential of PM from cow dung cake smoke was
found to be increased using an \textit{in vitro} technique involving a synthetic model of the respiratory tract
lining fluid [19].

Inadequate combustion of biomass releases CO that binds with haemoglobin producing carboxy-
haemoglobin due to high affinity of haemoglobin to CO (200-250 times) compared to oxygen [2].
Potentially this can reduce oxygen transport to key organs and the developing foetus which may result
in low birth weight and perinatal death [20].

In human, acute exposure to sulphur dioxide, often released during biomass burning, can increase
bronchial reactivity in normal individuals and cause broncho-constriction in asthmatic individuals at
levels of around 100 parts per billion (ppb). Longer term exposure may increase susceptibility to viral
infections of the lung [21].
A few studies have looked at the toxicity of biomass smoke in exposed populations in developing countries. In chronically biomass smoke exposed Indian women, activation of circulating platelets, neutrophils and monocytes has been reported with high levels of leukocyte-platelet aggregates [22]. As a number of studies on ambient air pollution suggest that particulate pollutants increase fibrinogen levels, thus enhancing blood coagulation [23], it is plausible that biomass smoke exposure could be a risk factor for cardiovascular events. There have been very few controlled biomass smoke exposure studies [24] in human but there is a suggestion that exposure at levels of around 250 µg/m³ is associated with an increase in circulating Factor VIII and serum amyloid A, both of which confer an elevated cardiovascular risk.

The inherent toxicity of the smoke from biomass burning differs by the type of fuel, implying differential health risks conferred by different fuels. Airborne endotoxin concentrations in homes burning different types of biomass fuels in Nepal and Malawi [25] have been reported to be higher than those found in occupational settings [26, 27] and in the indoor environment in developed countries [28]. The median value of endotoxin (in EU/m³) was greatest in households burning maize crop residue (1609 EU/m³) followed by cow dung (365 EU/m³) and wood (113 EU/m³) all values being much greater than 40 EU/m³ [29], a health-based guidance limit recommended in the Netherlands for an 8-hr time weighted average occupational exposure.

**Improved cookstoves**

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. The most realistic alternative would be to reduce the exposure levels by switching to more efficient improved cookstoves. Major projects to produce and disseminate improved cooking stoves in the developing world have been initiated in the last decade. The aims were two-fold. First, by reducing the levels of indoor air pollutants, it was hoped that the health burden would be reduced. Second, by improving burning efficiency, fuel use could be economised to help slow
down deforestation and desertification. While there is no universal definition, generally speaking all improved cookstoves are characterised by a higher efficiency in thermal conversion, a higher heat transfer ratio, and a more complete combustion (and therefore a lower emission of smoke and other pollutants), compared with the “traditional” counterparts. Tests (water boiling, kitchen performance and controlled cooking) have been developed and recommended to monitor the performance and efficiency of the improved cookstoves. Improved cookstoves could be classified on the basis of (i) the types of fuel used (operable on one single or multiple fuel types), construction materials (made of a single or a combination of materials), portability (fixed or portable), and end use applications (mono-function for cooking only, or multi-function for cooking, room heating, etc.) [30]. The choice of stove should be customised for the target users, taking into account of local cooking requirements, and affordability and availability of fuels. Local availability of construction materials and maintenance are important keys to long term self-sustainable projects. In areas where improved cookstoves are not available, certain modification of the cooking environment and practices, such as improving ventilation, or even avoiding cooking indoors where possible could be helpful in reducing the smoke exposure. At present, approximately 27% of the total population using solid fuels (or 38% of the population in developing countries) have access to improved cookstoves - more than two-thirds (70%) in China, 9% in India and 4% in other South Asian countries. However, in Sub-Saharan Africa and the least developed countries, the figures were as low as 5.8% and 6.6%, respectively [3].

Exposure monitoring

Particulate matter (PM)
PM is classified on the basis of its aerodynamic diameter (AD) which is a function of particle size, mass and shape. Most of the studies where actual biomass smoke exposures have been measured (as opposed to a simple exposed/non-exposed classification) have reported exposures as PM$_{10}$ (with AD <10 µm), although more recently PM$_{2.5}$ (with AD <2.5 µm) has been used as a PM metric reflecting the likelihood that greater toxicity resides in the smaller size fraction. PM can be measured either by gravimetric techniques or by photometric devices. Gravimetric techniques give an aggregate of exposure concentrations over a period of time but photometric techniques are gaining popularity as they measure real time PM concentrations in a repeated manner which provides information on the variation of particle concentration over time and in relation to different activities. The down side of this technique is that measurements need to be calibrated against gravimetric results in the environment where exposure monitoring is to be done as the light scattering method often tends to overestimate PM concentrations [31].

Previous work has shown 24-hr indoor concentrations of PM$_{10}$ generated from solid fuels in different settings to be in the range 300-3000 µg/m$^3$ with peaks reaching as high as 20,000 µg/m$^3$ during cooking [4, 32-35]. In a wood-using community in Nepal, the 24-hr average PM$_{2.5}$ was found to be 680 µg/m$^3$ (range 616 -744 µg/m$^3$) [31] similar to other studies from Guatemala (22-hr average = 520 µg/m$^3$) [4] and China (24-hr average = 489 µg/m$^3$) [36]. All these reported concentrations are several fold higher than the WHO global ambient air quality guidelines, which recommend that the 24-hr and annual mean PM$_{10}$ concentration should not exceed 50 µg/m$^3$ and 20 µg/m$^3$ respectively with PM$_{2.5}$ not exceeding 25 µg/m$^3$ and 10 µg/m$^3$ respectively [37]. These result are several folds higher when compared to the results from the United Kingdom (12-34 µg/m$^3$) [38] for PM$_{10}$ and 35 µg/m$^3$ or less for total suspended dust in the United States[39].
Carbon monoxide (CO)

CO has been suggested as a cheaper but surrogate measure of indoor air pollution caused by burning biomass fuel. CO is measured by using either colour changing diffusion tubes or electro-chemical monitors. Diffusion tubes can only measure total exposure over time and the CO concentration is indicated by the stain inside the tubes when in contact with CO. More recently, small electro-chemical devices (e.g. HOBO CO loggers, EL-USB-CO loggers) have been used to measure CO concentrations. These devices are cheaper, smaller and require less power to operate over extended time periods.

Bruce et al. (2004) [40] reported 24-hour CO concentration in homes using an improved stove of just over 3 parts per million (ppm) compared to 12.4 ppm with open fires while unreported data from Nepal found broadly similar levels (18 ppm), but with substantial increases to around 200 ppm during cooking, while results from Guatemala (average 5.9 ppm) were similar [41]. The concentration of CO will depend upon the efficiency of fuel combustion and the moisture content of the fuel [42], wet wood fuel generating more smoke and thus more CO due to incomplete oxidation of the carbon content. WHO recommended time-weighted average guidelines for CO are 87 ppm (100 mg/m³) for 15 min, 52 ppm (60 mg/m³) for 30 min, 26 ppm (30 mg/m³) for 1 hr and 9 ppm (10 mg/m³) for 8 hr [43].

Health effects from exposure to solid fuel smoke

It is estimated that 1.9 million people die prematurely due to exposure to smoke from solid fuel burning [44]. According to the WHO, exposure to smoke from solid fuel burning is ranked as the top environmental risk factor worldwide being responsible for 3.3% of all mortality and 2.7% of all disability adjusted life years (DALYs) per year [45]. Long term exposure to solid fuel smoke is clearly associated with chronic obstructive pulmonary disease (COPD), increased risk of acute respiratory infections/pneumonia, lung cancer, tuberculosis and cataract [46-52]. The evidence is weaker for endpoints such as asthma, adverse
pregnancy outcomes, cancer of the upper aero-digestive tract, interstitial lung disease and ischaemic heart disease. More research both from animal and human studies are needed to establish the causal association between these health effects and exposure to biomass smoke. Some of the health effects associated with solid fuel smoke exposure are acute and include oxygen desaturation [53] and acute lower respiratory infection (ALRI)/pneumonia [54]. Here we discuss in detail the respiratory health effects associated with smoke from solid-fuel burning.

**Respiratory Health Effects**

The epidemiological data on biomass use coming from Asia (most of them from South Asia), Sub-Saharan Africa, Central and South America have provided substantial evidence to suggest that there is an association between exposure to biomass smoke and COPD in adults and ALRI/pneumonia in children.

**Chronic Obstructive Pulmonary Diseases (COPD)**

COPD, once regarded as a disease of developed countries, is now recognised as a common disease in developing countries. COPD is the fourth leading cause of all deaths, around 3 million people dying from the condition in 2004, of which 90% were from low and middle income countries [55]. While the main contributory factors to COPD are cigarette smoke and occupational causes, exposure to solid fuel smoke is a major contributory factor in developing countries. According to WHO estimates, approximately 700,000 out of the 2.7 million global deaths due to COPD could be attributable to indoor air pollution from solid fuels [56] particularly in women. However, the purported link between exposure to solid fuel smoke and COPD has often been based on surrogate measures of exposure and no studies have shown a relationship between direct measurement of biomass smoke exposure and the incidence or prevalence of COPD. Lower socio-economic status increases the risk of developing COPD although which component factors (e.g. poor housing, poor nutrition, low
income, no/poor education) are the most important in influencing COPD and to what extent is unclear. Nevertheless, one indicator of low socio-economic status, the use of solid fuel, has been suggested as a key causal factor [57-59]. Although the underlying mechanisms for the development of COPD among non-smokers exposed to biomass fuels are still unknown, some human challenge [60] and toxicological studies [19, 61] have reported that biomass burning produces chemicals with high oxidative potential and have implicated that oxidative stress and DNA damage might be one of the underlying mechanisms responsible for the pathogenesis of COPD [62, 63] in those exposed to biomass smoke. Rivera et al [64] compared the lung morphology in COPD secondary to cigarette and biomass smoke and reported that smokers with COPD had larger extent of emphysema and goblet cell metaplasia than women exposed to biomass smoke, but the latter presented more local scarring and pigment deposition in the lung parenchyma and more fibrosis in the small airways. In contrast, Moran-Mendoza et al [65] found that wood smoke exposed non-smoking women had histo-pathological findings (dyspnoea, airway obstruction, air trapping, increased airway resistance, pathological evidence of anthracosis, chronic bronchitis, centrilobular emphysema, bronchila squamous metaplasia and pulmonary hypertension) similar to smokers.

Studies on solid fuel use and COPD are often observational, small in sample size with insufficient statistical power to show a clear relationship, with relevant confounding factors often inadequately addressed. A further issue is the use of non-standard definitions of COPD or chronic bronchitis. Therefore, the findings across studies are open to some caution as the published estimates may be either under- or over-estimates of the true burden. A systematic review and meta-analysis [46] identified 23 studies, 10 reporting COPD based on both physician diagnosis and spirometrical definitions, 11 reporting chronic bronchitis based on
respiratory questionnaire data and 2 reporting both COPD and chronic bronchitis. The pooled effect estimate for lung function diagnosed COPD (OR = 2.96, 95% CI 2.01, 4.37) was greater than those diagnosed by doctor in hospital (OR = 2.29, 95% CI 0.70, 7.52) with a combined pooled effect estimate of 2.80 (95% CI 1.85, 4.23) for COPD (Figure 3). Similarly, the pooled effect estimate for chronic bronchitis (Figure 4) was 2.32 (95% CI 1.92, 2.80). The findings, published in 2010 [46] are similar to a recent meta-analysis [66] published for both chronic bronchitis (OR = 2.52, 95% CI 1.88, 3.38) and COPD (OR = 2.40, 95% CI 1.47, 3.93).

Figure 3: Forest plot showing risk of COPD in populations exposed to solid fuel smoke [46].
Figure 4: Forest plot showing risk of chronic bronchitis in populations exposed to solid fuel smoke [46].
It is likely that exposure to biomass smoke from an early age will have been important in retarding lung growth. In a study from Nepal where lung function was compared between a biomass smoke exposed population and a non-exposed population [67], the absolute values for various indices of lung function were significantly lower in both men and women in the biomass smoke exposed group, the difference being evident even in the youngest age group studied (16-25 years). This suggests an effect of biomass smoke exposure on lung growth in addition to any effect on rate of decline of lung function in later years. The prevalence of airflow obstruction (defined by forced expiratory volume in 1 s (FEV$_1$) / forced vital capacity (FVC) < 0.70) in the biomass smoke exposed group was almost doubled compared to non-biomass exposed (20% vs 11%).

Overall, there is good evidence that exposure to biomass smoke is associated with airflow obstruction and an at least doubling of the risk of COPD, the effect being detected by young adulthood.

**Asthma**

There is a wide variation in the prevalence of asthma worldwide [68]. Asthma has been less widely studied in the developing countries compared to the developed countries and understanding of the very different set of risk factors in these countries associated with its development, notably indoor environment and lifestyle, is limited. There is evidence that growing up in an agricultural environment is associated with a reduced risk of developing asthma, perhaps mediated by exposure to endotoxin [69] so it might be expected that exposure to biomass, largely occurring in rural communities, might not be associated with development of asthma per se but could be associated with exacerbations of existing asthma or with respiratory symptoms which might lead to a mistaken diagnosis of asthma. Some studies in developing countries have considered possible associations with biomass/solid fuel pollutant exposures [70-72]. Samuelsen et al [73] studied allergy adjuvant effect of particles from wood smoke and road traffic in laboratory animals and found that particles generated from wood burning had about the same capacity to enhance allergic sensitization as road traffic particles, but less
than diesel exhaust particles. Acute exposure to biomass smoke causes bronchial irritation, inflammation and increases bronchial reactivity possibly responsible for exacerbation of asthma [20].

**Table 1**: Studies of the relationship between biomass exposure and asthma prevalence.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Country</th>
<th>Fuel type</th>
<th>Sample size</th>
<th>Sample type</th>
<th>Diagnosis criteria</th>
<th>Effect size OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mohamed et al. 1995 [65]</td>
<td>Kenya</td>
<td>Biomass and Clean fuel</td>
<td>77 cases and 77 controls</td>
<td>Children aged 9-11 year</td>
<td>Adapted from IUATLD</td>
<td>2.5 (2.0, 6.4)</td>
</tr>
<tr>
<td>Azizi et al. 1995 [66]</td>
<td>Malaysia</td>
<td>Wood and kerosene</td>
<td>158 cases &amp; 201 control</td>
<td>1 month to 5 year old children</td>
<td>Hospital based medics diagnosed</td>
<td>1.4 (0.60, 3.60) (wood) and 0.9 (0.50, 1.60) (kerosene)</td>
</tr>
<tr>
<td>Melsom et al 2001 [64]</td>
<td>Nepal</td>
<td>Biomass and clean fuel</td>
<td>121 cases &amp; 126 control</td>
<td>11-17 year old</td>
<td>ISAAC criteria</td>
<td>2.2 (1.0, 4.5)</td>
</tr>
<tr>
<td>Mishra 2003 [68]</td>
<td>India</td>
<td>Biomass and clean fuel</td>
<td>38595</td>
<td>≥ 60 year old</td>
<td>Based on interviewee replying yes to Asthma questionnaire</td>
<td>1.59 (1.30, 1.94)</td>
</tr>
<tr>
<td>Schei et al 2004 [67]</td>
<td>Guatemala</td>
<td>Wood</td>
<td>1058</td>
<td>4-6 year old children</td>
<td>ISAAC criteria</td>
<td>1.8 (0.76, 4.19)</td>
</tr>
</tbody>
</table>

Published effect sizes of for asthma in relation to biomass exposure are presented in Table 1. All these studies adopted different techniques to determine asthma and none measured actual biomass exposure levels. While this limits the ability to compare the studies, all show positive associations between indoor air pollution and asthma at least in children.

A case-control study in Nepal assessed the home environment of school children in relation to asthma, using the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire [74] and found that passive smoking and use of biomass fuels was separately associated with an increased risk of asthma in boys only but keeping animals in home was associated with lower risk (OR=0.2, 95% CI 0.1, 0.5) of asthma. This finding is supported by a similar case-control study from Kenya [75] which found that dampness in the child’s sleeping area, indoor air pollution and presence of rugs or carpets in child’s bedroom were strongly associated with asthma (ORs 2.0 to 4.9) and by a study from Guatemala in relation to asthma symptoms in 4 to 6 year old children [68]. In contrast, a study from
Malaysia only found an association of asthma with environmental tobacco smoke and exposure to mosquito coil smoke [76].

A meta-analysis on the above four studies showed that exposure to indoor air pollution approximately doubles the risk of developing asthma in children (OR = 1.96, 95% CI 1.29, 2.99) but some caution needs to be taken in interpretation as the methodology was imperfect in some way in all studies.

Only one study has considered biomass smoke exposure and asthma in adults [68]. Using a simple approach to defining asthma (a positive response by the head of household to the question: “Does anyone listed suffer from asthma?”) the study found that people living in houses using biomass as a domestic fuel and being an active smoker had a significantly higher risk of asthma. Although this study found that asthma in elderly men and women (≥ 60 years) is associated with use of biomass fuel, the exposure was based on a crude yes/no response and there is a high possibility of misclassifying asthma.

In summary, the evidence possibly supports a role for biomass exposure being causally related to asthma in children but formal, well designed studies are needed to confirm this possibility. In particular methods should ensure as tight a diagnosis of asthma as possible (perhaps including measures of bronchial reactivity e.g. using mannitol challenge or exercise) to determine whether these findings simply reflect a pattern of asthma-like symptoms or true asthma.

**Acute respiratory infection (ARI)**

ARI can be divided into two types - upper respiratory tract infections (URI) and lower respiratory tract infections (LRI) which can in turn be categorised on clinical conditions, aetiology and markers of severity[59]. Though there are clinical and epidemiological criteria to separate URI from LRI, worldwide there are no uniformly accepted definitions used in epidemiological research. The lack of a uniformly accepted definition of ARI may cause bias.
that would contribute to the heterogeneity in reporting across different studies. The possible mechanism related to respiratory infection from acute exposure to particulate matter from biomass burning might be due to reduced mucociliary clearance and long term exposure increasing susceptibility to bacterial and viral lung infections [20].

LRI are major causes of morbidity and mortality leading to over four million deaths per year worldwide, approximately 69% of which occur in developing countries [77, 78]. After neonatal deaths, ARI (17%) is the second leading cause of deaths in children under 5 years and the fourth leading cause of death in the world where 7.4% women and 7.1% men die annually due to respiratory infections [78]. In low-income countries respiratory infection is the leading cause of deaths (11.2%, 2.94 million) [78].

Mortality due to respiratory infection is higher in the under 5 years and 60 years or above age groups. Young children exposed to solid fuel smoke have two to three times more risk of serious ARI than unexposed children [59]. Deaths due to respiratory diseases are highest in African countries followed by eastern Mediterranean and then south-east Asian countries [78] where most of the people are of low socio-economic status. More than 70% of the population in these areas use solid fuels for domestic purposes and respiratory deaths from respiratory tract infections could well be attributed to the high exposure from the burning of biomass/solid fuel. Exposure to air pollutants might increase the incidence of ARI by adversely affecting specific and non-specific defences of the respiratory tract against pathogens [79]. It is important to emphasise that death due to ARI is mainly associated with lower respiratory infection rather than upper respiratory infection.
A longitudinal study in rural Kenya studied ARI and acute lower respiratory tract infection (ALRI) in children under the age of 5 years [5, 80]. The study measured biomass exposure as PM$_{10}$ and found a dose-response relationship between PM$_{10}$ and the increase in ARI and ALRI frequency. The rates of ARI and ALRI were higher for exposures of PM$_{10}$ below 1000-2000 $\mu$g/m$^3$ but the rate of increase declined where exposure concentrations were above 2000 $\mu$g/m$^3$.

A longitudinal study (1984-1985) in Nepal of children under 2 years showed a possible relationship between ARI and average number of hours spent in the kitchen (OR = 2.2) reported by the mother [81]. A suggestion of a dose-response relationship was found in this study but the exposure assessment was not validated.

A case-control study from urban Nigeria did not find significant associations between age, nutritional status, environmental tobacco smoke (ETS) exposure, location of cooking and crowding with ALRI [82]. Mishra (2003) studied acute respiratory infection in pre-school children (under 5 years) in Zimbabwe and found that approximately 16% of the children had ARI [83] at the time of their survey. The study reported that, after adjusting for appropriate confounders, children in households using biomass were more than twice (OR = 2.2) as likely to suffer from ARI as children from households using clean fuel for domestic purposes. A one year cohort study carried out in 500 Gambian children under 5 years [84] reported that parental smoking appeared (non-significantly) to increase the risk of ALRI. However, the risk of ALRI was 6 times higher in girls than in boys, perhaps due to the fact that girls are carried on mother’s back more often than boys during cooking and hence are exposed more to biomass exposure.

A meta-analysis of 24 studies relating to pneumonia in children under 5 years exposed to smoke from solid fuels showed the exposure to solid fuel smoke approximately doubles the risk of pneumonia (OR
There is thus consistent evidence that biomass smoke exposure is associated with an increased risk of ARI/pneumonia in children.

**Tuberculosis (TB)**

There is inconsistent evidence that exposure to biomass smoke increases the risk of TB [86, 87]. The proposed mechanism is that biomass smoke compromises the respiratory system’s ability to resist infection by *M. tuberculosis* or to resist development of active TB in already infected persons [88]. There is enough evidence to support the belief that current and/or former smoking is associated with TB [87, 89-93] and some evidence to suggest that passive smoking is also a risk factor [94, 95] acting via a range of potential immune mechanisms. Similarly, biomass exposure interferes with the mucociliary clearance [96] and decreases several antibacterial properties of lung macrophages, such as adherence and phagocytic rate [97, 98] providing theoretical mechanistic reasons to support the possibility that biomass smoke might be a risk factor for TB.

The available data suggest (Table 2) that there might be a causal link between exposure to biomass smoke and either an increased risk of acquiring TB or progression of TB to clinical disease. There are very few studies that have explored this link and there is heterogeneity in design, measurement of outcome and the magnitude of risk estimates which need to be explored further to come to a firm conclusion. Most of the studies related to biomass use and TB suggest that prevalence of TB is higher in communities with poor sanitation and lower socio-economic status and these communities primarily uses solid fuel for domestic purposes. A meta-analysis on the 10 studies mentioned in Table 2 revealed a pooled effect estimate (OR) of 1.55 (95% CI 1.11, 2.18) suggesting that individuals exposed to solid fuel smoke are 55% more likely to get tuberculosis compared to a non-solid fuel
smoke exposed group. While there was significant heterogeneity among the studies ($I^2 = 70\%$, $p<0.001$), no significant publication bias was reported.

Table 2: Studies of tuberculosis infections in relation to biomass exposure

<table>
<thead>
<tr>
<th>Reference</th>
<th>Country</th>
<th>Fuel type</th>
<th>Sample size</th>
<th>Sample type</th>
<th>Effect size OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Case control studies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gninafon et al 2011</td>
<td>Benin</td>
<td>Solid fuel</td>
<td>200 cases and 400 controls</td>
<td>Age and sex matched community based controls</td>
<td>1.7 (1.1, 2.8)</td>
</tr>
<tr>
<td>Kan et al 2011</td>
<td>China</td>
<td>Solid fuel</td>
<td>202 cases and 404 controls</td>
<td>Age and sex matched community based controls</td>
<td>1.08 (0.62, 1.87) (NS)</td>
</tr>
<tr>
<td>Lakshmi et al 2010</td>
<td>India</td>
<td>Biomass</td>
<td>126 cases and 252 controls</td>
<td>Age matched community based controls</td>
<td>3.14 (1.15, 8.56) (p=0.02)</td>
</tr>
<tr>
<td>Kolappan et al 2009</td>
<td>India</td>
<td>Biomass</td>
<td>255 cases and 1275 controls</td>
<td>Age and sex matched community based controls</td>
<td>1.7 (1.0, 2.9) (p=0.04)</td>
</tr>
<tr>
<td>Pokhrel et al 2009[86]</td>
<td>Nepal</td>
<td>Biomass and Kerosene</td>
<td>125 cases and 250 controls</td>
<td>Age matched hospital based controls</td>
<td>1.21 (0.48, 3.05) (Biomass) 3.36 (1.01, 11.22) (Kerosene)</td>
</tr>
<tr>
<td>Shetty et al 2006[90]</td>
<td>India</td>
<td>Biomass</td>
<td>189 cases and 189 controls</td>
<td>Matched hospital based controls</td>
<td>0.90 (0.46, 1.76) (NS)</td>
</tr>
<tr>
<td>Crampin et al 2004[87]</td>
<td>Malawi</td>
<td>Biomass</td>
<td>598 cases and 992 controls</td>
<td>Community based controls</td>
<td>0.60 (0.3, 1.1) (NS)</td>
</tr>
<tr>
<td>Perez-Padilla 2001[99]</td>
<td>Mexico</td>
<td>Biomass (present/past)</td>
<td>288 cases and 545 controls</td>
<td>Hospital based controls</td>
<td>2.2 (1.1, 4.2)$^a$; 1.5 (1.0, 2.4)$^b$; 1.1 (0.6, 2.0)$^c$</td>
</tr>
</tbody>
</table>

|                  |         |               | Cross-sectional studies |                                                |                        |
| Mishra et al 1999[100] | India   | Biomass       | 260,162 persons screened | All aged $\geq 20$ in the sampling location | 2.58 (1.98, 3.37)      |
| Gupta et al 1997 | India   | Biomass       | 707                   | Adults                                       | 2.54 (1.07, 6.04)      |

$^a$ Present exposure to biomass; $^b$ Present or past exposure to biomass and $^c$ Past exposure to biomass

NS=Non-significant

**Lung cancer**

Lung cancer is one of the leading causes of death accounting for 1.3 million deaths annually worldwide [101]. While smoking is the major risk factor, as many as a quarter of cases are not attributable to tobacco use [102]. Lung cancer in never smokers is more common in women than men although there is considerable regional variation in the proportions of non-
smoking females with lung cancer; for instance, in East and South Asia, up to 83% of female lung cancer cases are never smokers, compared to 15% in the United States [102]. Emissions from combustion of solid fuels have been shown to have high concentrations of PAHs, benzo[a]pyrene and PM$_{2.5}$, which in turn have been associated with high lung cancer rates [102].

A meta-analysis of 28 studies relating to lung cancer exposed to solid fuel smoke showed a greater effect of coal smoke on lung cancer rates (OR = 1.82, 95% CI 1.60, 2.06) with biomass smoke, predominantly wood, (OR = 1.50, 95% CI 1.17, 1.94) and mixed biomass fuel smoke (OR = 1.13, 95% CI 0.52, 2.46) showing lesser effects. The higher risk of lung cancer in coal users was not surprising as combustion products obtained from in-home coal burning contain a range of PAHs classified as Group 1 carcinogens [103]. The general mechanism emerged from PAHs such as benzo[a]pyrene (BaP) is genotoxicity where BaP metabolises to electrophilic form that adducts to DNA. The International Agency for Research on Cancer (IARC) has classified combustion products from biomass (primarily wood) use as probably carcinogenic to humans (Group 2A) for lung cancer due to “limited evidence” in humans and experimental animals [103].

One methodological issue in such analyses is the use of appropriate comparison populations. The pooled effect size obtained from studies using population (OR = 1.83, 95% CI 1.51, 2.21) based controls (carrying 56% weight) were greater than those using hospital (OR = 1.63, 95% CI 1.34, 1.97) based controls (39% weight). This suggests the evidence of the carcinogenicity of biomass smoke is still not conclusive, supporting the IARC evaluation.

Cigarette smoking [104, 105] has been widely identified as the main contributory factor for lung cancer worldwide but no objective measurement of smoking was carried out in any of
the studies included here. However, all studies included in this review either adjusted for smoking or studied a population of non-smokers. While it is accepted that self-reported smoking history is the best that can be achieved when considering life-long smoking details, objective measurement of smoking, such as salivary cotinine, is becoming more easily usable in field studies and at least provides information on current smoking and will help reduce slightly exposure misclassification.

Almost 75% of the studies included in this meta-analysis did not adjust for ETS but studies dealing with coal smoke exposure and with ETS adjusted (OR = 1.47, 95% CI 1.13, 1.91) had lower pooled effect sizes compared to those where ETS was not adjusted for (OR = 1.74, 95% CI 1.60, 1.89). Only one study out of 8 related to biomass smoke exposure adjusted for ETS and had effect size higher than the other that were not adjusted for ETS. Thus, ambiguity regarding the combined effect of smoking, combustion products of solid fuel and ETS exposure still prevails and future studies need to address this issue. There is evidence from occupational studies that smoking and some occupational exposures (e.g. asbestos, PAHs) have a multiplicative rather than simply an additive effect on lung cancer risk [106, 107] and it is therefore possible that such a potentiating effect may be seen with respect to smoke from solid fuel burning, especially that from coal.

Most of the cooking in developing countries is done by women who are more likely to be exposed to indoor air pollution than men. The pooled effect size shows that the risk of lung cancer is greater in women (OR = 1.81, 95% CI 1.54, 2.12) compared to men (OR = 1.16, 95% CI 0.79, 1.69), similar to that reported in a limited earlier meta-analysis [108] for women only (OR = 1.83, 95% CI 0.62, 5.41).
There may be an effect on cell type as the pooled effect size for squamous cell carcinoma (OR = 3.58, 95% CI 1.58, 8.12) was greatest followed by adeno-carcinoma (OR = 2.33, 95% CI 1.72, 3.17) and unspecified lung cancer type (OR = 1.57, 95% CI 1.38, 1.80). Squamous cell lung cancer is more commonly associated with cigarette smoking [109] although reported series of lung cancers have recently shown an increase in the proportion of adeno-carcinomas which is thought not to be simply an issue of changes in classification/grading [110].

**Indoor air pollution from other sources and associated health effects**

There are number of other sources that contribute to the worsening of indoor air pollution in developing countries and may thus contribute to ill-health.

**Cooking oil fume**

**Cooking emissions**

Cooking is the treatment of food under heat. High temperature initiates volatilisation as well as a number of chemical reactions in the food ingredients, generally involving decomposition of lipids and amino acids [111]. The resulting emissions have been found to contain PM [112], volatile organic compounds (VOCs) [113] and other organic compounds including PAHs [114] and heterocyclic amines [115], some of which are potent mutagens and carcinogens. The types and levels of pollutants in cooking emissions are highly heterogeneous and depend on food ingredients [116-119]. For example, beef steak fried with margarine generated significantly higher levels of PM and aldehydes than when rapeseed oil, olive oil or soybean oil was used under the same cooking procedures [116]. On the other hand, Chiang and colleagues analysed emission samples in Taiwan and reported non-significant variations of levels of PAHs [118] and aromatic amines [119] using different
types of cooking oil. In a controlled environment, Fullana et al. reported higher levels of acetaldehyde and acrolein emissions from heated canola oil and olive oil and suggested this might be related to their difference in fatty acid composition, where canola oil contains a higher proportion of linolenic acid and a small proportion of oleic acid than that in olive oil [117]. The same report also provided evidence that the levels of pollutants are positively correlated with heating time and temperature [117]. Not surprisingly, different methods of cooking, such as frying (pan-, stir-, deep-frying), grilling, baking, can affect the levels of emission. For instance, when stir-frying meat the concentration of benzo[a]pyrene (2.64 µg/100 m³) can be 4 times as high as that when it was boiled (0.65 µg/100 m³) [120].

Chinese cooking appears to be of particular concern because the techniques involved generally require high temperature cooking with oil, such as in stir-frying and deep-frying. This is supported by a study in Taiwan, which found the annual rate of PAH emission was highest from Chinese restaurants compared with Western, fast food and Japanese restaurants, after taking into account of a number of factors including cooking oil consumption and cooking methods [121]. A study in Singapore compared the concentrations of PM$_{2.5}$ and PAHs in three ethnic food stalls and found the levels both pollutants to be highest in Malay, followed by Chinese and Indian stalls. The difference in the levels could be explained by the frying processes predominantly used at the Malay (deep-frying) and the Chinese (stir-frying) stalls, whereas simmering (at lower temperature) was mostly used at the Indian stall [122].

**Respiratory effects**

Compared to the wealth of knowledge on the respiratory effects of biomass and solid fuels, far fewer studies have been dedicated to cooking related emissions. Of those that did, the majority focused on lung cancer. A recent monograph from IARC identified 17 case-control
studies exploring the association between exposure to cooking emissions and lung cancer and all were conducted exclusively in the Chinese population [103]. Among these 17 studies, only four allowed the cooking-related effects to be distinguished from those related to fuels [123-126]. In a group of non-smoking women in Hong Kong, Yu et al. found an escalating dose-response relationship between cumulative exposure (frequency and duration, in dish-years) and lung cancer risk, with a relative risk of about 3 for 101-150 and 150-200 dish-years exposure, and over 8 for >200 dish-years (referent exposure being ≤50 dish-years) [124]. In the two studies from Shanghai, those women who stir-fried most frequently were 2.6 [126] and 2.3 times [123], respectively as likely to have lung cancer compared with those who performed least often. Similarly the study in Gansu reported a relative risk of 2.2 [125]. These findings might explain the observed high non-smoking lung cancer incidence in Chinese women, which could be attributed to their high cumulative exposure to cooking emissions. However, confounding by cooking fuel could not be ruled out because of the history of using biomass or solid fuels in the study populations in the latter three studies. In addition, recall and other forms of bias that are found in case-controls could be operating and contributed to the positive findings in some of these studies. Therefore, the causal relationship has not yet been totally confirmed.

There are few data on respiratory diseases other than lung cancer and none were derived from developing countries. A survey of 239 kitchen workers from 67 restaurants and found a four-fold and two-fold increase in risk of dyspnoea for females and males respectively compared with the controls [127]. However, the results could have been confounded by combustion products although relatively clean fuel (gas) was used in these restaurants. Assessing the possibility of acute responses, the lung function of 12 healthy volunteers were monitored over
a 24-hour period with and without exposure to cooking fumes for two and four hour but found no significant changes in spirometry [128].

**Burning of incense sticks**

Incense is regularly burnt in homes and offices for religious or ceremonial rituals and fragrance, particularly in developing countries. Incense is available in various forms including sticks, joss sticks, cones, coils, powder, rope, rocks/charcoal, and smudge bundles. The substances widely used to produce incense are resins (such as frankincense and myrrh), spices, aromatic wood and bark, herb seeds, roots, flowers, essential oils, and synthetic substitute chemicals used in the perfume industry [129]. Burning of incense releases different air pollutants such as PM, VOCs, carbonyl compounds, CO, nitrogen oxides, methane, non-methane hydrocarbons, organic carbon, elemental carbon and inorganic ions (chloride, nitrate, sulphate, sodium, ammonium and potassium ions) depending on the types of incense sticks and aroma used, the concentration being much higher during the peak burning time of incense sticks [129-131]. The types of compounds present in the smoke released after burning incense suggest that they can cause number of acute and chronic health impacts. Although number of studies have suggested that smoke released from burning can cause respiratory health effects [132, 133], lung cancer [134-136], dermatological allergic reactions and could be mutagenic and or genotoxic [137-139], the evidence is inconsistent, with some studies finding inverse relationships for lung cancer [140, 141] and COPD[142].

**Burning of mosquito coil**

Annually, 45 to 50 billion mosquito coils are used by approximately 2 billion people worldwide [143], particularly in rural and semi-rural communities of developing countries to prevent from mosquito bites. Liu *et al.* estimated that burning a mosquito coil can release mass concentration of PM$_{2.5}$ equivalent to burning of 75-137 cigarettes depending upon the
types of base material used to make the mosquito coil and release formaldehyde equivalent to 51 cigarettes [144]. The smoke released from burning mosquito coil contains some carcinogenic PAHs including benzo[a]pyrene, benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenzo[a,h]anthracene and indeno[1,2,3-cd]pyrene [144, 145]. People in developing countries tend to burn mosquito coils during the summer nights and are therefore regularly exposed to the smokes released for about 6-8 hours daily. Inhalation of the smoke has been reported to cause breathing difficulties, eye irritation, bronchial irritation, itching, cough and asthma [144, 146, 147].
Table 3: Research priorities on health effects/exposure/interventions of solid fuel smoke (adapted from [20])

<table>
<thead>
<tr>
<th>Research Priorities</th>
<th>Sub-groups</th>
<th>Types of studies</th>
</tr>
</thead>
</table>
| Health effects      | Basic studies | • Genetic susceptibility to various health effects  
|                     | Epidemiological studies | • Relationship between exposure and health outcomes  
|                     |                      | • Different health outcomes such as cervical cancer, visual impairment, lung growth in children, asthma in children  
|                     | Clinical | • Mechanisms of health outcomes related to solid fuel smoke  
|                     | Measurements | • Standardisation of cross-sectional and longitudinal monitoring of exposure  
|                     | Equipment | • Research and development on types of equipment such as cost, size, weight, power supply and resistance to extreme conditions for developing countries  
|                     | Improved cookstoves | • Types of stoves (multiple stoves, multiple fuel scenarios, multiple types of food cooked and different cooking practices)  
|                     | Social intervention | • Impact of massive educational programs on raising the awareness of health effects of exposure to biomass smoke  
|                     | Fuel types | • Research on inexpensive but cleaner fuel types or source of heat such as production of briquettes and charcoal from agricultural wastes  
|                     | Effects | • Impact of improved cookstoves programs on the health outcomes |
**Conclusion**

The available evidence suggests that despite heterogeneity among published studies there is sufficient evidence and consistency among published studies to conclude that exposure to solid fuel smoke is a risk factor to COPD in adults and pneumonia in children particularly those less than 5 years of age. Although the field has been hampered by methodological weaknesses, such as exposure not being measured directly and inadequate accounting of possible confounding factors, the overall data is sufficient to be sure that the effects size for COPD is around a three-fold risk for those exposed and around a two-fold risk for ALRI in children.

The available evidence also suggests that exposure to coal smoke is a risk factor to lung cancer whereas the evidence from biomass smoke exposure on lung cancer is not conclusive. As for asthma, there remains uncertainty as to whether biomass smoke does increase the risk of developing asthma in childhood and tighter methodological studies are needed to determine any true causal association. While there is limited information suggesting that deep pan frying, using incense and mosquito coil are risk factors to respiratory problems, these risk factors should be regarded as suggestive only at this stage and need to be explored further (Table 3).
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