



Exposure to biomass smoke as a cause for airway disease in women and children

Rahul Kodgule and Sundeep Salvi

Purpose of review

An estimated 3 billion people (about half the world's population) burn biomass fuel (wood, crop residues, animal dung and coal) for cooking and heating purposes exposing a large population, especially women and children, to high levels of indoor air pollution. Biomass smoke comprises gaseous air pollutants as well as particulate matter air pollutants, which have significant harmful effects.

Recent findings

Exposure to biomass smoke is a major contributor to morbidity and mortality. Children, women and the elderly are most affected. Apart from poor lung growth seen in growing children, the risk of developing respiratory tract infections (both upper as well as lower) is greatly increased in children living in homes using biomass. Women who spend many hours cooking food in poorly ventilated homes develop chronic obstructive lung disease (COPD), asthma, respiratory tract infections, including tuberculosis and lung cancer. It has been argued that exposure to biomass fuel smoke is a bigger risk factor for COPD than tobacco smoking.

Summary

Physicians need to be aware about the harmful effects of biomass smoke exposure and ensure early diagnosis and appropriate management to reduce the disease burden. More research needs to be done to study health effects due to biomass smoke exposure better. Reducing the exposure to biomass smoke through proper home ventilation, home design and, if possible, change of biomass to cleaner fuels is strongly recommended in order to reduce biomass smoke-induced mortality and morbidity.

Keywords

asthma, biomass smoke, chronic obstructive lung disease, lung cancer

INTRODUCTION

About 1.5 million years ago, our ancestors *Homo erectus* learnt to light fire from wood to keep wild animals away. Soon humans started using fire for cooking and heating purposes using biological sources of fuel such as wood, crop residues, animal dung, dried twigs and grass and coal, commonly called biomass [1]. Today, approximately 50% of the population of the world and 90% of rural households, especially those in the developing countries, continue to rely on biomass fuel and coal as their main domestic source of energy [2]. Regions most abundantly using biomass fuel are South-east Asia and Africa (Fig. 1), and rural homes in South America and Middle-Eastern countries also use significant amounts of biomass for cooking purposes. Being cheap and easily accessible biomass fuels are popular especially amongst the poor. Because of increasing costs of cleaner fuels many homes in developed countries are also shifting to solid fuels mainly to

keep their homes warm [3]. Currently more than 3 billion people in the world use biomass fuel and coal for cooking or heating purposes, and it is projected that the overall use of solid fuels will keep on rising in the near future. The most commonly used biomass fuels are wood, crop residues and animal dung (Fig. 2), although some countries, such as China and many African countries, use coal on a large scale.

Chest Research Foundation, Marigold Complex, Kalyaninagar, Pune, India

Correspondence to Dr Sundeep Salvi, MD, DNB, PhD, FCCP, Director, Chest Research Foundation, Marigold Complex, Kalyaninagar, Pune 411014, India. Tel: +91 20 27035361; fax: +91 20 27035371; e-mail: ssalvi@crfindia.com

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KEY POINTS

- Fifty percent of households worldwide use biomass fuel for cooking and heating purposes, exposing 3 million people to high levels of indoor air pollution.
- Homes burning biomass fuel have very high levels of indoor air pollutants like particulate matter, carbon monoxide, sulfur dioxide, nitrogen dioxide and polyaromatic hydrocarbons.
- Exposure to biomass smoke is associated with inadequately developed lungs at birth; acute respiratory infections and poor lung growth in childhood; and faster decline in lung function, COPD and lung cancer in adult life.
- Patients with biomass smoke-related COPD typically present with breathlessness and have bronchial wall thickening, diffuse emphysema, mild interstitial fibrosis and pulmonary artery hypertension.
- Exposure to biomass smoke is associated with higher levels of inflammatory cells and mediators, oxidative stress and DNA damage in the lungs, and these levels are comparable to those found in tobacco smokers.

BIOMASS SMOKE AS AN INDOOR AIR POLLUTANT

Biomass fuels are inefficient as they not only produce less heat but also burn incompletely to release various pollutants in the air. The choice of fuel is largely determined by the cost and accessibility. Crop residues are least fuel-efficient and the fuel efficiency improves in order as one would switch to animal dung, wood, kerosene, coal, liquefied petroleum gas (LPG) and electricity. Burning of biomass fuel produces more than 200 identified chemical and compound groups, over 90% of which are in the inhalable size range [4]. These pollutants can be broadly categorized into gaseous air pollutants, such as carbon monoxide, sulfur dioxide (SO₂), nitrogen dioxide (NO₂) and particulate matter air pollutants such as those less than 2.5 µm (PM_{2.5}) and those less than 10 µm (PM₁₀) in aerodynamic diameter, polyaromatic hydrocarbons (PAHs), chlorinated dioxins, arsenic, lead, fluorine and vanadium. Particles less than 10 µm in diameter can be easily inhaled and reach the deeper portions of the lung and are therefore considered to be most damaging to the lungs. Burning solid fuels produces very high levels of PM₁₀ with average 24-h levels ranging between 300 and 3000 micrograms per

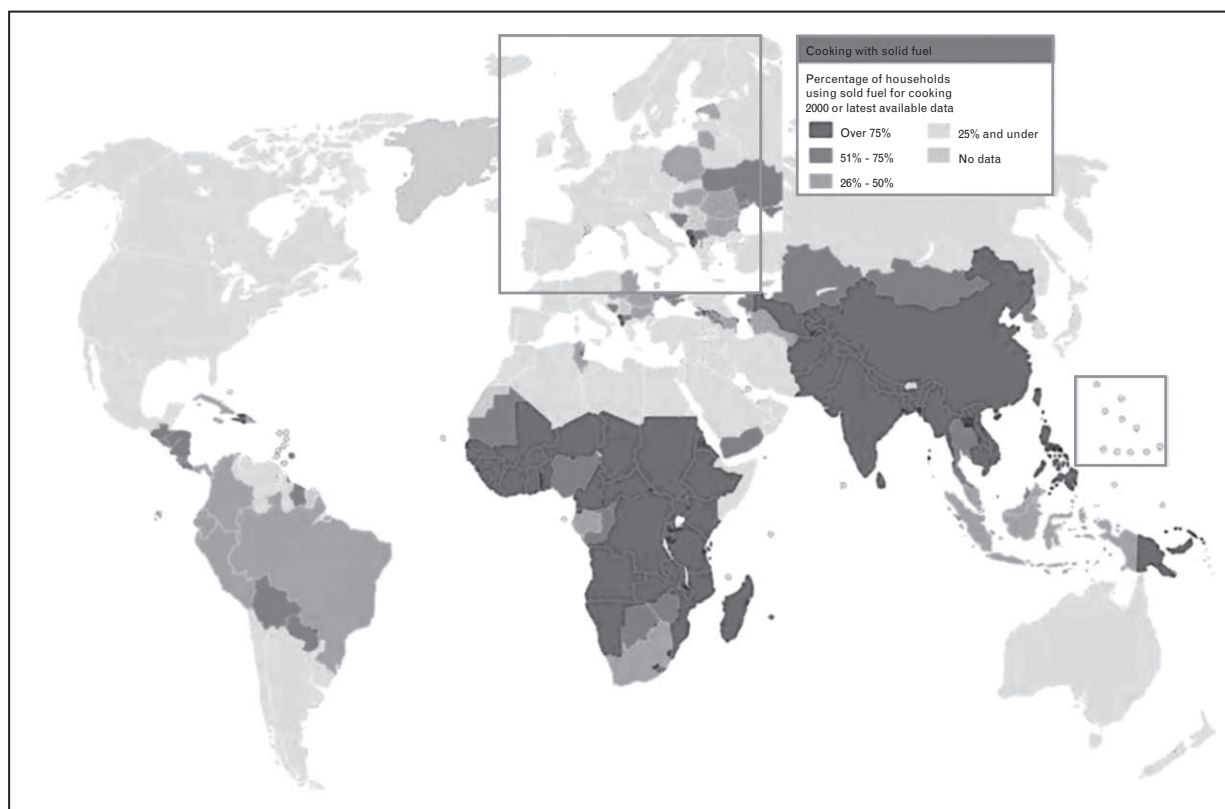


FIGURE 1. Worldwide use of biomass fuel for cooking (Modified from: Children's environmental health. Part 2: global environmental issues, WHO 2008). Reproduced with permission from www.who.int/ceh/publications/en/map09b.jpg.

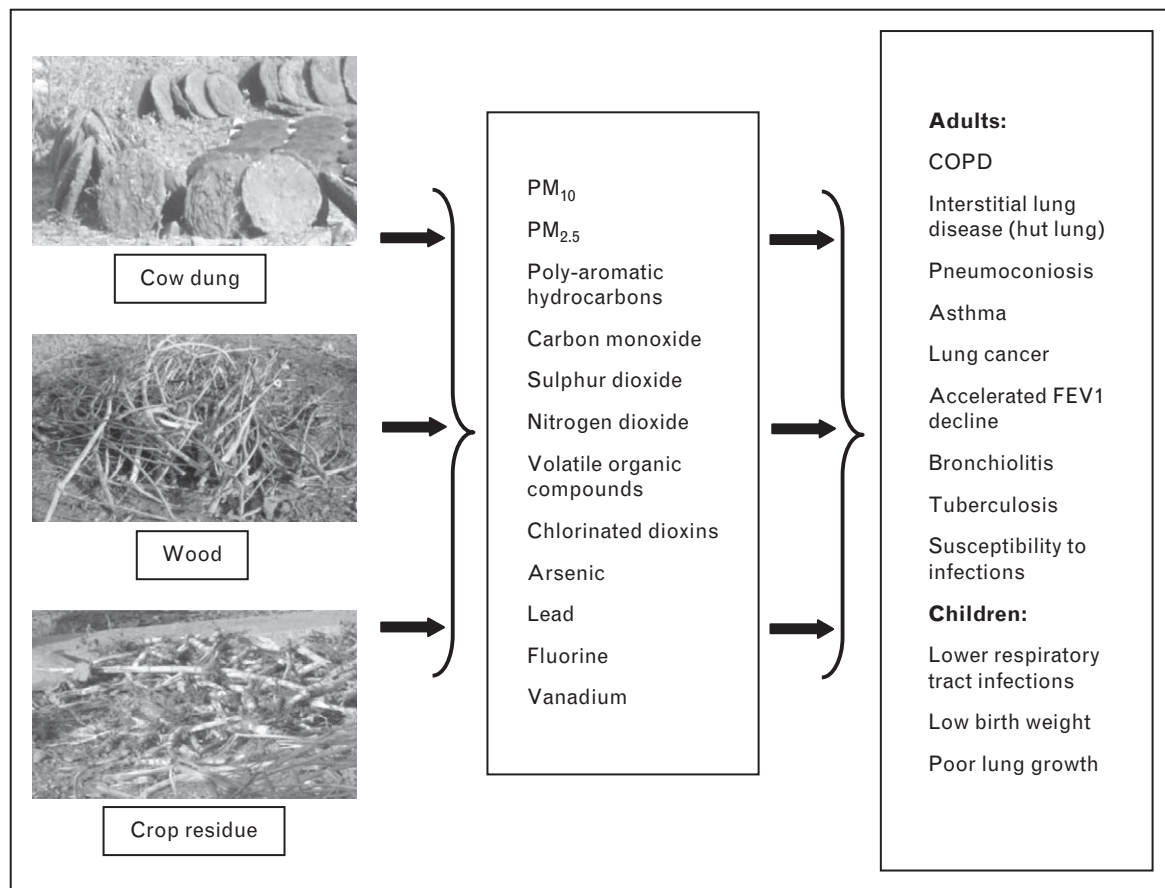


FIGURE 2. Different types of biomass fuels, their composition and health effects.

cubic metre ($\mu\text{g}/\text{m}^3$) and the peak levels during cooking may reach levels as high as $10\,000\ \mu\text{g}/\text{m}^3$ (Fig. 3). The peak levels form up to half of total exposure in women who are involved in cooking. These levels are several-fold higher than the United States' Environmental Protection Agency (US-EPA) standard for annual mean outdoor PM₁₀ levels in outdoor air levels, which are $50\ \mu\text{g}/\text{m}^3$ [5]. We have recently reported that rural homes in India that burn biomass fuel show median levels of PM_{2.5} that are more than two-fold higher than homes that use liquefied petroleum gas (256 vs. $109\ \mu\text{g}/\text{m}^3$) [6]. The highest concentrations of particulate matter are generated by burning animal dung, followed, in order, by wood and kerosene [7]. Biomass smoke also contains a substantial number of particles of size less than $2.5\ \mu\text{m}$ (PM_{2.5}). These can penetrate deeper into the lung and even enter into the systemic circulation and thereby have more damaging effects in other organs of the body.

The pollutants released during biomass burning mainly include 17 pollutants designated as priority pollutants by the US-EPA which contain up to 14 carcinogenic compounds, 6 cilia-toxic and mucous coagulating agents and 4 co-carcinogenic or cancer-promoting agents [8]. The majority of these

pollutants are known to be toxic or irritant to the respiratory system, including PM₁₀, carbon monoxide, nitrogen and sulfur oxides (NO₂, SO₂),

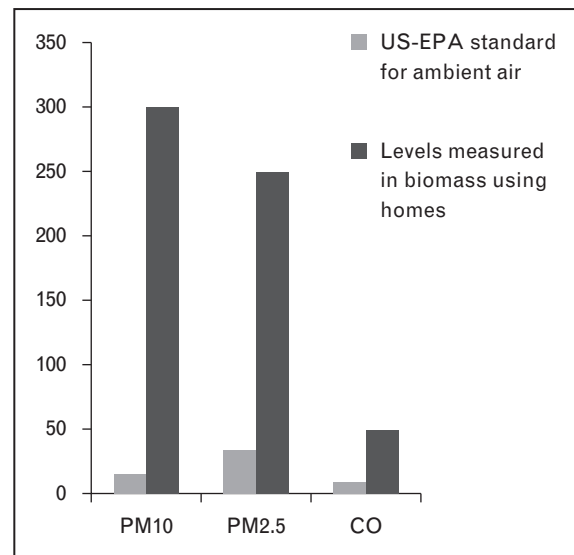


FIGURE 3. Comparison of US-EPA standard for ambient air and levels measured air in homes exposed to biomass smoke PM₁₀, PM_{2.5} and CO. Levels expressed as $\text{mcg}/\text{m}^3 \times 10$ for PM₁₀, mcg/m^3 for PM_{2.5} and ppm for CO.

aldehydes (e.g. formaldehyde), polycyclic aromatic hydrocarbons (e.g. benzopyrene), volatile organic compounds (VOCs), chlorinated dioxins and free radicals [4]. Apart from sulfur oxides coal fuels may emit other toxic elements, including arsenic, lead and fluorine [9]. At least 12 PAHs considered to be potentially mutagenic and/or carcinogenic have been identified in biomass smoke [10].

SUSCEPTIBLE POPULATION AND FACTORS AFFECTING EXPOSURE

Individuals most affected by biomass smoke exposure are women who cook food and children who are often carried on their mother's back while she is cooking or are in close vicinity to the stove [3]. Women are typically exposed to the peak levels of pollution for 3–7 h each day while cooking and spend 70% of their time in the polluted indoor atmosphere [11]. On an average, women spend more than 60 000 h during her lifetime cooking near a biomass stove, during which period she inhales a total volume of over 25 million liters of polluted air. Balakrishnan *et al.* [12] found that women cooking with biomass fuel indoors are exposed to higher levels of particulate matter as compared with those cooking outdoors. Whereas presence of partition to the kitchen did not make any difference to the overall exposure of the cook, it significantly reduced exposure to other members in the house [12]. About one-third of rural and one-fourth of urban households in countries like India live without a separate kitchen, indicating that a large number of people are indeed exposed to high levels of indoor air pollution [13]. Children, sick and the elderly people largely remain indoors, and therefore inhale a greater volume of biomass air pollutants. We have recently observed that duration of cooking using biomass fuel was associated with a greater risk of respiratory morbidity (unpublished observation). We also found that presence of an additional window in the kitchen was associated with lower adverse respiratory effects (unpublished observation) indicating that kitchen ventilation is an important determinant of adverse health effects and may have a role for intervention.

HEALTH EFFECTS DUE TO EXPOSURE TO BIOMASS SMOKE

Amongst all the air pollutants emitted from biomass, the one that has shown the maximum amount of adverse health effects is particulate matter. A $50 \mu\text{g}/\text{m}^3$ increase in 24-h PM_{10} has been associated with a 2.5–5% increase in premature nonaccident-related mortality [14]. Even a $10 \mu\text{g}/$

m^3 increase in PM_{10} has been estimated to increase respiratory disease mortality by 3.4% and total mortality by 1% [15]. In the year 2000, solid fuel use accounted for 2.6% of global ill health [9]. The World Health Organization (WHO) has attributed 1.5 million premature deaths every year to indoor air pollution (IAP) from the burning of solid fuels. More than half of these deaths occur in children less than 5 years of age and the rest occur mostly in women [5]. According to the WHO, indoor air pollution is one of the top 10 preventable risk factors contributing to the global burden of disease [4].

Exposure to biomass smoke has been associated with several diseases of the lung including chronic obstructive lung disease (COPD), asthma, bronchiolitis, lung cancer, interstitial lung disease, faster decline in lung function and risk of developing pulmonary tuberculosis, in adults, and respiratory tract infections (RTIs) and poor lung growth in children. In this review we discuss biomass smoke-related airway diseases with a major focus on COPD.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

COPD may probably be the most important and detrimental effect of exposure to biomass smoke.

Epidemiological evidence

Several studies have shown that exposure to biomass smoke is an independent risk factor for the development of COPD, especially amongst women [16]. The PREPOCOL [17] study on COPD prevalence, carried out in Columbia, revealed that exposure to wood smoke for more than 10 years was a significant risk factor for COPD as defined by a postbronchodilator ratio of Forced Expiratory Volume in one second to Forced Vital Capacity (FEV_1/FVC) less than 0.7. Perez-Padilla *et al.* [18] reported that wood smoke exposure for greater than 200 h-years increased the risk of chronic bronchitis by around 15 times and that this was strongly associated with exposure–response effects. We have recently reported that exposure to biomass smoke is strongly associated with COPD in a rural population as well as in an urban slum population in India [19,20]. In a meta-analysis of 15 studies, Guoping *et al.* [21] concluded that people exposed to biomass smoke had a 2.4-fold increased risk [odds ratio (OR) = 2.44, 95% confidence interval (CI) 1.9–3.33] of having COPD compared with those not exposed. This association has been substantiated by another recent meta-analysis which has shown similar risks (OR = 2.40, 95% CI 1.47–3.93) [22]. It is interesting to note that this

risk is similar to that seen in smokers who develop COPD (OR=2.5), and greater than those who develop COPD due to passive smoking or those who have α_1 -antitrypsin deficiency. Compared with 1.1 billion smokers worldwide, there are 3 billion people who are exposed to high levels of indoor air pollution largely due to the burning of biomass fuel. We have therefore argued that exposure to biomass smoke is probably the biggest risk factor for COPD globally [3].

Histopathological changes

Histopathologically biomass-related COPD is characterized by presence of anthracosis and fibrosis along with typical changes of broncho-vascular inflammation and emphysema that are also commonly seen in tobacco smoking-related COPD. This typical picture seen in people living in African huts was earlier described as 'Hut Lung disease' as it could not be categorized histopathologically into any other disease and was found to be mainly due to a combination of biomass fuel smoke and inhalation of fine sand dust due to grinding of maize and wheat on soft stone. Similar observations have also been made in a rural population in India [23–25]. Higher levels of peribronchial inflammation, alveolar wall destruction, emphysematous changes, respiratory epithelial cell proliferation, parenchymal infiltration and fibrosis, nodular aggregation and perivascular inflammation have been observed in animals experimentally exposed to biomass smoke [26,27].

Clinical presentation

Biomass-related COPD patients typically present as middle or old-aged women complaining of breathlessness rather than cough with or without expectoration. They are more likely to be diagnosed as

asthma because of absence of smoking history and lack of awareness amongst physicians about non-smoking COPD. These patients are more susceptible to develop infective respiratory tract exacerbations, because of both biomass smoke exposure (discussed later) and poor living conditions. Pulmonary hypertension has been reported to be more commonly found in biomass smoke-exposed individuals [11].

Physiological and radiological changes

Patients having COPD associated with biomass smoke have airway obstruction on spirometry, increased airway resistance on impulse oscillometry (R_5 , R_{20}) and decreased airway conductance on body plethysmography (sGaw). They also have significant hyperinflation as suggested by an increased Residual Volume to Total Lung Capacity ratio (RV/TLC) and reduced diffusion capacity as suggested by reduced absolute Diffusion Capacity of the Lung using carbon monoxide (DLCO) and DLCO/alveolar ventilation (VA) values (Fig. 4) [28].

High-resolution computed tomography (HRCT) of biomass smoke-related COPD patients usually shows presence of diffuse emphysema, bronchial wall thickening, thickening of interlobular septae, increased bronchovascular arborization, nodular opacities, ground-glass appearance, and pleural thickening [29–32].

Airway inflammation

The pathogenetic mechanisms behind development of COPD due to exposure to biomass smoke are inadequately understood but are likely to be quite similar to those due to tobacco smoking-related COPD. There is consistent evidence of higher levels of airway inflammation and oxidative stress in individuals exposed to biomass smoke. Recently, we

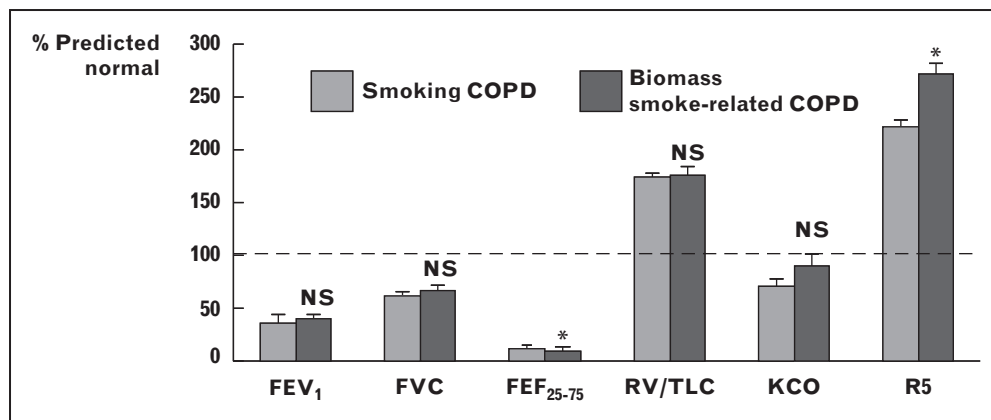


FIGURE 4. Comparison of lung function parameters between tobacco-smoking COPD and biomass-smoke exposure associated COPD [28].

compared 15 biomass smoke-exposed COPD patients with 25 tobacco-smoking related COPD patients and 11 age-matched healthy nonsmoker, nonexposed controls and found significantly higher levels of inflammatory cells (neutrophils, eosinophils) and mediators [IL-8, Matrix Metalloproteinase (MMP)-9 and 8-isoprostane] in the induced sputum samples of biomass smoke-exposed COPD patients compared with healthy individuals. Interestingly, these levels were similar to those found in tobacco smoke-related COPD (Fig. 5) [33]. Mondal *et al.* [34] found higher counts of neutrophils, eosinophils, monocytes, mast cells and alveolar macrophages in sputum of women exposed to biomass smoke. The biomass-exposed women were also found to have significantly higher levels of reactive oxygen species (ROS) generation which correlated positively with years of cooking and significantly lower levels of the antioxidant, superoxide dismutase (SOD) in the sputum homogenate [34].

Apart from finding high sputum neutrophil count in never-smoking biomass-exposed women, Banerjee *et al.* [35] found higher plasma levels of pro-inflammatory cytokines tumor necrosis factor- α (TNF- α), IL-6, IL-12 and an almost two-fold increase in the levels of the neutrophil chemoattractant, IL-8. They also found higher levels of ROS and lower levels of SOD in biomass smoke-exposed women [35]. Higher oxidative stress has also been reported in women exposed to biomass smoke [36]. Montano *et al.* [37] reported high activities of MMP-2, 9 and 12 in the broncho-alveolar lavage fluid of COPD patients exposed to wood smoke than healthy nonexposed individuals. Interestingly, activities of MMP-2 and MMP-9 in these individuals were higher than tobacco smoke-associated COPD patients.

Exposure to both biomass smoke and cigarette smoke has been shown to be more deleterious than

either of the two alone as reflected by higher levels of symptoms, histopathological changes, inflammatory cells and oxidative stress in this subgroup of COPD patients [26].

ASTHMA

The recent dramatic increase in asthma has been associated with industrial development and rapid rise in urban air pollution suggesting a possible role of air pollutants in development of asthma [38,39]. Although several epidemiological studies have shown an association between ambient air pollution and asthma [40,41], there are only a few studies that have investigated the association between exposure to biomass smoke and asthma. Using the International Study of Asthma and Allergies in Children (ISAAC) questionnaire amongst residents of Guatemala, Schei *et al.* [42] reported that children exposed to open biomass fire for cooking had a 3.4-fold higher prevalence of asthma symptoms compared with those using the improved stove, plancha (adjusted OR = 3.4, 95% CI 1.3–8.5).

Several mechanisms may be hypothesized for the development of asthma due to exposure to biomass smoke. High nitrogen dioxide (NO₂) levels in indoor air have been associated with asthma and enhanced allergic response [43]. It is suggested that in the presence of air pollutants pollen grains undergo changes in shape and become agglomerated with airborne particles [44,45]. These pollens then release higher levels of eicosanoid-like substances that increase serum IgE, eosinophils and neutrophils [46]. Apart from this, biomass smoke contains high levels of endotoxin (especially dung smoke) and VOCs. Once inhaled, endotoxin stimulates an amplifying series of endotoxin–protein and protein–protein interactions, leading to lung

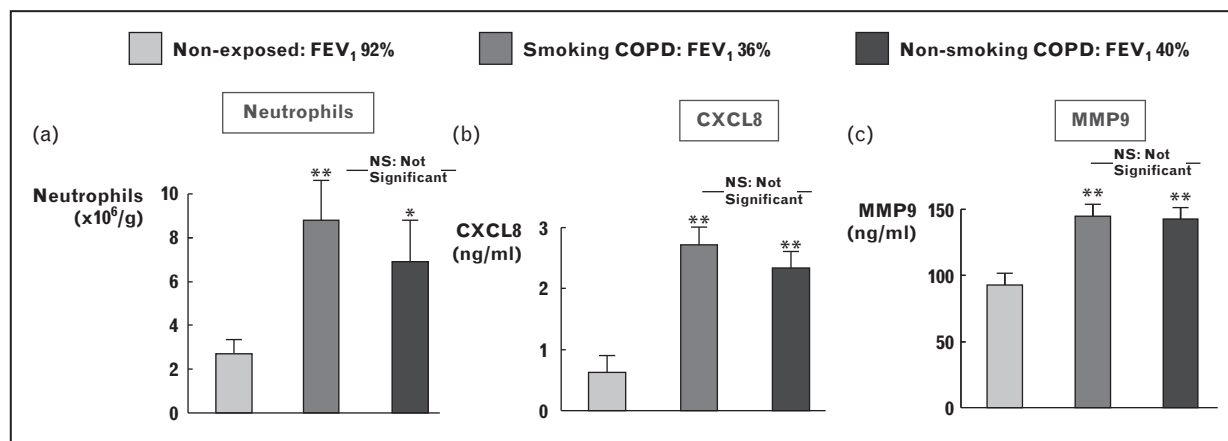


FIGURE 5. Comparison between sputum cellular and mediator inflammatory profiles between tobacco-smoking COPD and biomass smoke-associated COPD [33]. NS, not significant.

inflammation and oxidative stress that may contribute to the development of asthma. Studies have shown significant associations between household endotoxin concentrations and diagnosed asthma, asthma medication use and severity of asthma symptoms [47]. VOCs such as benzene, ethylbenzene and toluene which are present in biomass smoke are also significant risk factors for development of asthma amongst those chronically exposed to biomass smoke. Exposure to solid fuel smoke may also act as an asthma trigger. In rural areas of China, heating with charcoal stoves has been shown to be a trigger for asthma attacks [48].

LUNG CANCER AND DNA DAMAGE DUE TO BIOMASS SMOKE EXPOSURE

Several studies have shown that exposure to biomass smoke is a risk factor for the development of lung cancer. Behera and Balamugesh [49] found a significant association between exposure to biomass fuel and lung cancer on multivariate logistic regression analysis with OR of 3.59 (95% CI 1.07–11.97) after adjusting for smoking and passive smoking. Similar observations have also been made in China and Mexico [11]. The most common malignancy associated with biomass smoke exposure is lung adenocarcinoma. Mondal *et al.* [34^{*}] reported significant DNA damage in 85 non-smoking biomass-exposed women compared with 76 nonbiomass-exposed women, with the most harmful effects seen in women burning animal dung cake. It is suggested that DNA strand breaks are caused by higher levels of oxidative stress. This genotoxicity could be majorly responsible for development of lung cancer in developing countries and may be driven in great part by polyaromatic hydrocarbons, which are substrates for detoxification by GSTM1 [50].

BIOMASS SMOKE EXPOSURE AND ACUTE RESPIRATORY INFECTIONS IN CHILDHOOD

Children living in homes using biomass fuel for cooking have been shown to have a significantly increased risk of developing acute lower respiratory tract infections (ALRIs). ALRIs remain the single most important cause of death globally in children under 5 years and account for around 2 million deaths annually in this age group [11]. Half of these deaths are attributable to indoor air pollution [5]. In a multivariate analysis, infant mortality was found to be significantly associated with the proportion of biomass fuel used, after controlling for potential confounders [51].

Evidence from 13 studies in developing countries indicates that young children living in solid-fuel using households have two to three times increased risk of serious acute respiratory tract infections (ARIs) than unexposed children after adjustment for potential confounders including socioeconomic status [52]. ALRIs have been related to biomass combustion in children living in Nepal, Argentina, Nigeria, Tanzania, Gambia, Brazil and India [48]. A recent study [8] also characterized the exposure–response relationship between biomass smoke exposure and ARI in children of rural Kenyan households.

Several pathogenic mechanisms behind susceptibility to infections amongst those exposed to biomass smoke have been suggested. Since most particulate matter in biomass fuel is less than 2 μm in diameter, it is possible that such particulate matter may reach the deepest portions of the respiratory tract and alter defense mechanisms. Acute exposure to particulate matter causes bronchial irritation, inflammation and reduced mucociliary clearance, resulting in increased residence time of inhaled particles, including microorganisms [51]. Cow dung smoke has been shown to have high PAHs and resulted in deranged macrophages, damage to bronchiolar epithelium and edema in female wistar rats, thus making lungs susceptible to various respiratory infections [53]. Exposure to PAHs, especially benzo[a]pyrene, which is found in large quantities in biomass smoke, can increase bronchial reactivity and susceptibility to bacterial and viral infections [54]. Apart from this, important components of innate immunity such as surfactant proteins A and D have been shown to be a target for air pollutants [55].

BIOMASS SMOKE EXPOSURE AND LOW BIRTH WEIGHT AND POOR LUNG GROWTH IN CHILDREN

It is thought that the effects of biomass smoke can begin as early as *in utero*. Sreeramareddy *et al.* [56] reported that children born in households using high polluting fuels were 73 g lighter than those born in households using low polluting fuels. This association was significant even after controlling for other known confounding factors (adjusted OR = 1.21, 95% CI 1.06–1.32) [56]. A study [51] in rural Guatemala showed that babies born to women using biomass fuel for cooking were 63 g lighter.

Mean carbon monoxide concentrations in homes using biomass for cooking, are in the range of 2–50 ppm, reaching a peak of 500 ppm during cooking (levels much higher than the US-EPA, 8-h carbon monoxide standard of <9 ppm on average),

have been incriminated as a cause for low birth weight [51]. Carbon monoxide binds to hemoglobin in preference to oxygen and thus reduces oxygen delivery to key organs, consequently hampering the growth of developing fetus.

Low weight at birth has further been associated with poor lung function in adult life. Barker *et al.* reported that men with lower birth weight had lower FEV₁ adjusted for age and height and had higher standardized mortality rate for COPD. This was substantiated by a study [57] on Indian men and women in which mean FEV₁ fell by 90 ml with each pound decrease in birth weight in men and by 60 ml in women, independent of smoking. In another study [58], birth weight was found to positively correlate with spirometric and plethysmographic (TLC and Functional Residual Capacity) lung function and with lung diffusing capacity even after adjusting for exposure to cigarette smoke *in utero* and during childhood. Thus exposure to biomass smoke in mother's womb may have its deleterious effects that might last lifelong.

GENETIC SUSCEPTIBILITY

Like tobacco smoking, not all individuals exposed to biomass smoke develop disease, especially COPD, suggesting a role for genetic susceptibility in these individuals. Polymorphisms of alpha-1 antitrypsin deficiency associated gene (SERPINA-1), oxidative stress-related genes (GSTM1, GSTP1) and several other genes (SERPINA-2, genes for MMP-9,12, TNFβ1, TNF-α, etc.) have been associated with genetic susceptibility to COPD and other diseases, majorly in the Western world and sometimes in developing countries too [59]. It can be postulated that similar genetic susceptibility might play a significant role in biomass smoke-induced airway diseases, although, there is very little evidence currently available.

Biomass-related lung cancer has been shown to be associated with GSTT1 null and GSTM1 null genotype and significant DNA damage [50]. Polymorphisms in oxidative stress genes GSTM1 and GSTP1 have been suggested to lead to susceptibility to development of asthma in individuals exposed to air pollution and such asthma patients are likely to respond to antioxidant therapy [39[■]].

CONCLUSION

The causality of biomass smoke in COPD, asthma, tuberculosis and respiratory tract infections needs to be further explored through epidemiological and mechanistic studies. The burden of COPD in Asia is currently greater than that in Western countries.

Bronchitis and asthma have already been recorded as leading causes of death in rural India (males: 9.6%, females: 7.7% of all deaths in rural India). On top of it, the rate of tobacco smoking is rising rapidly in developing countries, the total number of biomass users is projected to rise and life expectancy is also improving, threatening an unprecedented rise in the burden of COPD in future. That time has come to take aggressive steps in recognizing biomass smoke as a social hazard and urgently take necessary measures to replace it preferably with cleaner fuel or at least reduce exposure with improved stoves and/or improved ventilation.

Acknowledgements

None.

Conflicts of interest

The authors do not have any conflict of interest to declare.

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- of special interest
- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 000–000).

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