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Chapter

Health Effect of Biomass Fuel Smoke

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Abstract

Almost half of the world population rely on solid (biomass fuel and coal) for cooking, heating and lightning purpose. The resultant exposure to fine particulate matter from household air pollution is the seventh-largest risk factor for global burden of disease causing between 2.6 and 3.8 million premature deaths per year. The health effect ranges from cardiovascular, respiratory, neurocognitive and reproductive health effect. The most important are cardiovascular and respiratory health effects; others are the risk of burns and cataract in the eyes. Biomass fuel is any living or recently living plant and animal-based material that is burned by humans as fuels, for example, wood, dried animal dung, charcoal, grass and other agricultural residues. Biomass fuels are at the low end of the energy ladder in terms of combustion efficiency and cleanliness. Incomplete combustion of biomass contributes majorly to household air pollution and ambient air pollution. A large number of health-damaging air pollutants are produced during the incomplete combustion of biomass. These include respirable particulate matter, carbon monoxide, nitrogen oxides, formaldehyde, benzene, 1, 3 butadiene, polycyclic aromatic hydrocarbons (PAHs), and many other toxic organic compounds. In this article, health effects of biomass fuel use will be described in details highlighting the most affected systems and organs of the body.

Keywords: health effects, biomass fuel, household air pollution, ambient air pollution, particulate matter

1. Introduction

Almost half of the world populations rely on biomass fuels (BMF) for cooking, heating and lightning purpose. Household air pollution (HAP) from incomplete combustion of BMF is now understood to be a major risk factor for adverse health outcomes [1]. According to the 2016 Global Burden of Disease Study (GBD), HAP is ranked as the single most significant environmental health risk factor globally. It accounted for 2.6 million deaths and 77.2 million disability-adjusted life years (DALYs) in year 2016 [2], with greater than 99% of death occurring in low and middle income countries [3]. The health effect ranges from cardiovascular, respiratory, neurocognitive and reproductive health effect. The most important one is cardiovascular and respiratory health effect [4–6].

1.1 Historical considerations

The association between high levels of air pollutants and adverse health outcomes has been known since the seventeenth century [7]. In the 17th century, Queen Elizabeth I forbid the burning of coal near the palace at Westminster due to the unpleasant nature of smoke, by 1661 in London, John Evelyn suggested that factories should be located far from residential area [7]. In the 19th century, clinicians started linking lung diseases to air pollution in England [8]. Also, smog incidents in Meuse Valley, Belgium in 1930, Donora, Pennsylvania in 1948, and London, UK in 1952 acutely affected the elderly and those with existing cardiac and respiratory diseases and it caused increased hospitalizations and deaths. As a result of the London smog, an estimated 4000 people died and over 100,000 people suffered adverse health effects [9, 10]. Earlier this year, millions of Australians have been reported to be affected by air pollution, especially in the southwestern part of Australia where fire killed about 20 people including 3 volunteered fire fighters from the country wildfire [11].

2. What are biomass fuels?

Biomass fuel (BMF) is any living or recently living plant or animal-based material that is burned by humans as fuels, for example wood, dried animal dung, charcoal, grass and agricultural residue such as straw and sticks, dried leaves, twigs and wild grass [12, 13]. Although biomass fuel is primarily used by women in developing countries for domestic cooking and it is also used in developed countries primarily for the purpose of heating at homes, for example, 5% of household surveyed in Australia used woodstoves for indoor heating. BMF may also be chosen for cooking in developing countries because of the flavor they impact during cooking processes e.g. barbecues, smoked meat and wood-fired pizza [14]. There is also occupational exposure to BMF in developing countries, such occur as fire fighters [15]. In addition, air pollution from BMF also result from planned forest fires for agricultural practices during autumn and spring, and bushfires from countries with substantial parks and bush lands such as Canada and the USA during summer [14].

Although, incomplete combustion of BMF from cooking and heating result mainly in household air pollution (HAP), it is also an important contributor to ambient (outdoor) air pollution (AAP), accounting for an estimated 10–30% of ambient fine particulate matter (PM) [16], this is particularly so in developing countries. However, exposure to biomass PM is increasing in developed countries mainly from domestic heating purposes, increasing wild fires, and which can substantially contribute to ambient PM concentrations, particularly in winter months [13–16].

2.1 Components of BMF

The air pollutants from burning of BMF is numerous and has been shown to consist of 200 different compounds. Some of the pollutants are PM, carbon monoxide, sulphur and nitrogen oxides; organic compound like formaldehyde, acrolein, etc. [17–19]. The exact chemical composition of biomass smoke is dependent upon the fuel type, the temperature of burning, whether an open fire or free radicals incinerator is used, and local conditions (e.g., wind speed, humidity, indoor or outdoor fires) [14].

Although there are many pollutants, it is PM that have received most of the attention in scientific literature, on the basis of robust epidemiological, clinical and toxicological association between PM, and respiratory and cardiovascular diseases [19, 20].

PM components of air pollution are mixtures of solid, liquid and mixed phased particles suspended in air. It consists of carbonaceous particles with associated adsorbed organic chemicals and reactive metals. Common components of PM include nitrates, sulfates, PAH, endotoxin, and metals such as iron, copper, nickel, zinc, and vanadium [17]. PM is heterogeneous and variations in the characteristics of particles (e.g. particle size, surface area, and composition) (e.g. PAH, metal, and endotoxin content) released from different emission sources can influence the biological response [21]. The composition of PM to air pollution is highly dependent on season, density of sources and the specific technologies employed as well as meteorology and topography. In middle and low income countries, homes using BMF with poor designs that do not have flues or hood to take smoke out of the living area are often affected by the adverse health effects of HAP due to lack of ventilations [12].

PM can be classified based on aerodynamic diameter; this determine the site of deposition [22–24]. PM with a diameter of 0.1 μm or less are termed ultrafine PM and are deposited in the alveoli. While diameter of 2.5 μm or less are termed fine PM.

PM which are light and can remain suspended in air for longer periods and they are deposited throughout the respiratory tract, particularly in small airways and alveoli [24, 25]. These particles can be inhaled deep into the lungs, and have been linked to oxidative stress and inflammation induced damage of the respiratory system [26]. Coarse PM has an aerodynamic diameter of 2.5–10 μm and are deposited in large airways [27, 28]. The concentration of PM can be as high as 100 times the recommended 24 hour concentration by the U.S. Environmental Protection Agency and the WHO [29]. Although much of the research has been on PM, other components of BMF contribute significantly to the damaging effect of the respiratory system [30].

3. Health effect of particulate air pollution

Several studies have reported association of PM with different respiratory diseases, cardiovascular diseases, cancers, reproductive, neurocognitive and metabolic diseases [18, 25, 31, 32]. In a meta-analysis by Atkinson and colleagues, every 10 μg/cm3 increase in PM2.5 concentration was associated with a 1.04% (95% CI 0.52%–1.56%) increase in all-cause mortality [33]. Using 85 studies from 12 low- and middle-income countries in a meta-analysis study, Newell et al. reported that a 0.47% (95% CI 0.34–0.61) increase for cardiovascular mortality and 0.57% (95% CI 0.28–0.86) increase for respiratory mortality for every 10 μg/cm^3 increase in PM2.5 concentration [34].

3.1 Mechanisms

One of the commonly cited mechanism for the relationship between air pollutants is oxidative stress. Both particulate and gaseous pollutants can produce oxidative stress and can act independently or synergistically together [26]. However, most of the research on mechanism has focused on PM, as discussed earlier. Also, while the toxicological and health effects of air pollutants from BMF and other sources may be informative, people are exposed to a toxic mixture of all the components and maybe challenging to extrapolate individual effect to the compound exposure of air pollutants.

Oxidative stress refers to the imbalance between the productions of reactive oxygen species (ROS) and the cells ability to detoxify reactive intermediates or to repair

cellular damage caused by ROS, examples of ROS are hydroxyl radicals and superoxide. When air pollution occurs, there is a dramatic increase in ROS level, resulting in significant damage to cellular components, including proteins, lipids, and DNA [35]. ROS can be derived from components of BMF smoke as well as the inflammatory cells recruited to the lungs [14]. Inflammatory response is produced through upregulation of pro-inflammatory cytokines such as tumour necrosis factor alpha, interleukin 6, and granulocyte colony stimulatory factor. Also, the immune cells are also recruited as well as upregulation of matrix metalloproteinases 2 and 9 and epithelial-mesenchymal transition [36]. In the airway and alveoli, this oxidative stress leads to alveolar macrophages activation and injury in the epithelial lining which in turn attracts inflammatory cells from the circulation [37]. In addition, PM in alveoli macrophages has been shown to modulate innate immune system and increased susceptibility to infection [38].

Lung inflammatory reaction can spill over into the systemic circulation and contribute to adverse effect in other organs [32]. PM exposure has been associated with systemic markers of oxidative stress which includes atherogenic precursors such as oxidized lipids, makers of hypercoagulability and thrombosis such as Von Willebrand factor and soluble CD 40 ligand, endothelial dysfunction, increased blood pressure and cardiac arrhythmias [31]. The systemic circulation could also be responsible for reproductive and intrauterine health effect [32].

Generation of ROS can also initiate free radical chain reactions which ultimately reach the nucleus and damage DNA leading to lung cancer and other cancers such as head and neck cancer. Additionally, gaseous pollutant present in air pollution such as nitrogen dioxide and volatile compound such as benzene can also lead to oxidative DNA damage. PAHs present in PM also form DNA adducts that has been implicated in carcinogenesis [39, 40].

4. Respiratory effects

There are compiling evidences associating exposure to solid fuel combustion products with respiratory diseases. Acute lower respiratory infection in children (ALRIs), chronic obstructive pulmonary disease (COPD) in women and lung cancer in women exposed to coal smoke are the three types of lung disease found to have strong evidence of association with exposure to solid fuel smoke: [12, 25].

4.1 Acute lower respiratory tract infection

The first report of indoor cooking smoke associated with childhood pneumonia and bronchiolitis was reported by Sofoluwe in Nigeria [41]. Acute lower respiratory tract infection (ALRI) is a leading contributor to the global burden of disease, it is also the commonest causes of morbidity and mortality particularly in children younger than five years. Almost all of this burden occurs in developing country where BMF is the primary source of household energy [12]. The relative risk for ALRIs for children exposed to BMF which include coal has been quantified in a number of studies [12]. In general, there is 2 to 3 times greater risk of developing ALRI in young children living in households exposed to solid fuel as compared to those not exposed [42].

The most recent meta-analysis by Smith and colleagues in 2014, using 23 observational studies and 1 randomized control trial, documented the pooled odds ratio of 1.78 (95% CI: 1.45–2.18) [18]. Although most of these studies are observational studies especially case control, and they used poor quality exposure measurement.

A recent study in Ethiopia, Adane and other investigators, recruited 5830 children less than 4 years old, and found that ALRI was linked with cow dung fuel use [AOR = 1.54 (95% CI: 1.02–2.330)], presence of extra indoors burning events

[AOR =2.19 (95% CI: 1.41–3.40)], child spending time near stove during cooking [AOR =1.41 (95% CI: 1.06–1.88)] and frequent cooking of meals [AOR =1.55 (95% CI: 1.13–2.13)] [43].

Although, there are many observational epidemiologic data that support an association between early childhood exposure to HAP and ALRI. Randomized control trials of interventions of improved cook stoves to reduce exposure to BMF smoke in order to prevent childhood ALRIs are emerging. A recent meta-analysis, of the six studies reporting child pneumonia outcomes, demonstrated no significant benefit in intention-to-treat analysis [44].

4.2 Tuberculosis

Tuberculosis is one of the leading causes of morbidity and mortality in the world, with a rate of 140 (95% CI: 915–1150) per 100,000 people. Exposure to HAP could impair the function of pulmonary alveolar macrophages and render the lungs prone to infections including tuberculosis [45]. There are conflicting results on the risk of tuberculosis and exposure to BMF. A meta-analysis by Lin et al. in 2014 reported that there were no association between BMF exposure and tuberculosis. With pooled odd ratio of 1.17 (95% CI: 0.83–1.65) for case–control study and 1.62 (95%CI 0.89–2.93) for cross sectional study [46]. Although, in Congo, Katoto reported that household air pollution is associated with chronic cough after completion of pulmonary tuberculosis treatment in adults [47].

4.3 COVID-19

The COVID-19 pandemic is still ongoing, there have been report of association of COVID-19 with AAP. Although, there are no available data specifically for HAP. Yao investigated the associations between PM concentrations and the case fatality rate of COVID-19 in 49 China cities, he found positive associations between PM pollution and COVID-19 in the 49 cities. Every increase in $PM_{2.5}$ and PM_{10} concentrations by 10 μg/m^3, raised the COVID-19 case fatality rate by 0.24% (0.01–0.48) and 0.26% (0.00–0.51) respectively [48]. Also, in Italy, Zoran and colleagues found positive correlations between confirmed COVID-19 daily new cases in Milan and air pollution with PM as follows, namely: with daily maxima PM_{10} (R^2 = 0.51), daily average surface air $PM_{2.5}$ (R^2 = 0.25), daily air quality index (R^2 = 4.35) [49]. In a USA study, after adjusting for factors such as population, weather, socioeconomic and behavioral variables, Wu and colleagues reported that increase of only 1 μg/m3 in $PM_{2.5}$ is associated with an 8% increase in the COVID-19 death rate (95% CI: 2%, 15%) [50]. However, air pollution has been documented to reduce during the lockdown period in different countries [51, 52], more epidemiological and experimental research are needed to estimate the impact of $PM_{2.5}$ on the incidence, severity and mortality of COVID-19.

4.4 Chronic obstructive pulmonary disease (COPD)

COPD is the fourth leading cause of mortality globally, causing more than 3 million death annually and over 80% of these deaths occur in low- and middle-income countries. It is also a substantial cause of economic and social burden [2]. It is characterised by persistent airflow limitation, associated with chronic inflammation of the airways and lungs in response to exposure to noxious particles and gases [53]. Previous systematic review has documented consistent association of biomass fuel with COPD. Kurmi et al. demonstrated that people exposed to BMF are at an increased risk of COPD compared to those that are not exposed [54]. Similarly, Hu et al. showed that people exposed to BMF smoke had increased odds ratio (OR) of

2.44 (95% CI, 1.9–3.33) for developing COPD, as compared to those that are not exposed, exposure to BMF was associated for developing COPD in Asian population, non-Asian population, in men and women [55].

In a recent meta-analysis by Sana et al., using 5 case–control studies and 19 cross sectional studies, they reported that biomass-exposed women were 1.38 times more likely to be diagnosed with COPD than non-exposed (OR 1.38, 95%Cl 1.28 to 1.57). The evidence is more for cross sectional studies than for case control studies but consistent in both rural and urban area [56]. HAP has been associated with increased risk of COPD exacerbation [57]. Effect of forest fire on emergency visit for COPD has been explored. Johnston et al. documented the odd ratio for COPD as 1.12 (95% CI: 1.02, 1.24) [58].

Intervention studies to prevent COPD from HAP is sparse, a prospective Chinese study show that substituting solid fuel with biogas for cooking and improving cooking ventilation were associated with a reduced decrease in FEV_1 and risk of COPD [59]. In a recent meta-analysis by Thakur et al., they documented that improved cook stoves were associated with a significant reduction in COPD among women: RR = 0.74 (95% CI 0.61 to 0.90). They also reported that ICS resulted in reductions in cough RR = 0.72 (95% CI 0.60 to 0.87), phlegm RR = 0.65 (95% CI 0.52 to 0.80), wheezing/breathing difficulty RR = 0.41 (95% CI 0.29 to 0.59) [60].

4.5 Asthma

Asthma is a non-communicable respiratory disease that is cause by chronic inflammation of the airways and results in wheezing, chest tightness, and cough. In 2015, approximately 400,000 people died of asthma worldwide [61]. In contrast to multiple studies on the risk of BMF smoke exposure and COPD, data are sparse on the risk of BMF and asthma. Although, there have been conflicting association or relationship between biomass exposure and asthma; evidences are emerging that biomass exposure may be linked with asthma risk, prevalence or incidence.

Barry et al. accessed 508 respondents and showed that individuals using wood or coal for cooking had increased odd of 2.3 (95% CI: 1.1–5.0) for reporting current asthma symptom [62]. Oluwole et al. also reported increased odds of asthma symptom in children who lived in household that used biomass had an adjusted OR of 1.33 (95% CI 1.05–1.6) for any of the asthma symptoms [63]. They further observed that biomass fuel use was associated with increased odds of severe symptoms of OR 2.39 (95% CI 1.16–4.84) [64]. Ayuk in their retrospective analysis of ISAAC phase III study found that open fire cooking was among the factors associated with asthma 1.28 (95% 1.06–1.51) [65].

However there are other studies that did not find an association between BMF and Asthma [66, 67]. Noonan et al. conducted a randomized control trial of air-filter intervention in asthmatic children exposed to BMF smoke, the intervention did not improve asthma quality of life. Although, there was an improvement in secondary measure of diurnal peak flow variability [68].

4.6 Other respiratory diseases

BMF exposure is also associated with interstitial lung disease referred to as 'hut lung' [12]. Hut lung disease is characterized by carbon disposition, dust macules, and mixed dust fibrosis, and it has been reported in cases primarily of women with chronic high-level exposure to indoor biomass smoke in developing countries [27]. In addition, bronchial anthracofibrosis has also been reported in elderly women who have worked long hours in poorly ventilated kitchen full of smoke due to incomplete combustion of BMF [69].

5. Cancers

5.1 Lung cancer

Lung cancer causes more death globally than any other cancer and it is the seventh leading cause of death globally [70], the International Agency for Research on Cancer concluded emissions from household coal combustion are a Group 1 carcinogen, while those from biomass were categorised as 2A due to epidemiologic limitations Although, smoking is the major risk factor for lung cancer worldwide, about 1.5% of lung cancer death are attributed to exposure to carcinogens from biomass fuel smoke annually [71].

In a meta-analysis to estimate the risk of lung cancer with the risk of BMF for cooking and heating, using 14 studies that were all case control, they found out that the risk of lung cancer with biomass for cooking and/or heating was OR 1.17 (95% CI 1.01 to 1.37). Although, more than 50% of the study did not report a clean reference category. When analyses restricted to studies with clean reference category, the evidence still remain the same for men and women [72]. A study published by Raspanti and colleagues, after the meta-analysis also reported OR: 1.77, (95% CI: 1.00–3.14), with the estimate more robust for non-smokers (P_{trend} = 0.01). Their study was a case control study of 606 lung cancer cases and 606 healthy controls matched on age (±5 years), gender, and geographical residence and adjusting for potential confounders such as tobacco use, tuberculosis status, Social economic status, age, gender, ethnicity, and exposure to second hand smoke. Conclusively, there was an increased risk of lung cancer among those who were exposed to HAPs [73].

5.2 Gastric cancer and esophageal cancer

Globally, gastric cancer is the fifth most common cancer and it is third among the causes of cancer mortality [74]. In a recent research carried out in Zambia, Kayamba et al. reported that there was an association between gastric cancer and reliance on BMF (OR, 3.5; 95% CI, 1.9 to 6.2; P < 0001) [40]. Okello and colleagues in a systematic review and meta-analysis using 16 case control studies, reported that the use of BMF was associated with increased risk of esophageal squamous cell carcinoma OR 3.02(95% CI 2.22, 4.11). Analysis by continent showed that Africa and Asia had the highest odds of esophageal squamous cell carcinoma [75].

5.3 Other cancers

Josyula and colleagues evaluated the relationship between HAP and other cancers apart from lung cancer in a meta-analysis, they found out that HAP was associated with cervical cancer neoplasia (OR = 6.46; 95% CI = 3.12–13.36); nasopharyngeal (OR = 1.80; 95% CI = 1.42–2.29); oral (OR = 2.44; 95% CI =1.87–3.19); laryngeal (OR = 2.35; 95% CI = 1.72–3.51) cancers and pharyngeal (OR = 3.56; 95% CI = 2.22–5.70). The association between HAP and upper aero-digestive cancers remained significant even when the analysis was restrained to only studies that controlled for smoking [76].

6. Cataract

Cataract is the clouding of the eye lens by preventing the passage of light and it is highly prevalent in developing countries. It is a leading cause of blindness

globally [77]. Several studies have shown an association between cataract and BMF. GBD Risk factors collaboration in 2015 reported that cataract is a global leading cause of blindness and it account for approximately 0.12% of all DALYS [77]. Smith et al. using seven studies from India and Nepal provided estimates for association between HAP and cataract, they reported a pooled OR of 2.64 (1.74, 3.50); the evidence for men was inconclusive. However, the estimate for women was OR 2.47 (1.61, 3.73) was deemed reliable [14].

A meta-analysis also reported in 2014, despite study heterogeneity, BMF use was associated with an increased risk of cataract with summary effect size of 2.12; 95% CI 1.61–2.80 [78]. Thakur et al. in a meta-analysis of 3 studies documented that improved cook stoves among women significantly reduce the presence of ocular symptoms RR =0.58, (95% CI 0.43–0.78) [60].

7. Cardiovascular, cerebrovascular and metabolic diseases

Cardiovascular disease is a leading cause of mortality worldwide and this is rapidly increasing in developing countries [31]. Although, there is a growing body of research linking HAP with sub-clinical indicators of cardiovascular disease risk including blood pressure, carotid atherosclerotic plaque, and arterial stiffness, epidemiological evidence linking BMF smoke and cardiovascular disease is limited [32]. According to a recent publication, it was reported that in middle- and low-income countries, household air pollution, along with other factors had stronger effects on cardiovascular disease or mortality compared to high income countries [79]. According to WHO, 12% of all death due to stroke can be attributable to the daily exposure to household air pollution arising from cooking with solid fuels and kerosene [80].

Fatmi and Coggon conducted a meta-analysis of 26 studies, 10 in south Asia, 4 in China, 2 in Turkey, 1 in Iran and 8 in Central and south American reported that the current balance of epidemiological evidence points to an increased risk of cardiovascular disease from HAP as a consequences of using solid and especially BMF for cooking and heating. Relative risks from long term exposure could be 2- to 4- fold [81]. In accordance with this study, using nationally representative and internationally comparable data, Arku et al. examined the association between solid fuel use and BP in 77,605 largely premenopausal women (aged 15–49) from ten resource-poor countries. They found that primary use of solid fuel was associated with 0.58 mmHg higher systolic BP (95% CI: 0.23, 0.93) as compared to primary use of clean fuel [82]. Ofoli et al. reported that use of BMF was associated with higher systolic blood pressure, more carotid intima media thickness (CIMT) and increased odds of pre-hypertension (OR: 1.67, 95% CI: 1.57–4.99, P = 0.035) [83].

In a cross sectional Chinese study involving more than 14,000 men and women aged 18 years or older showed that BMF and coal was associated with self-report of physician diagnosed ischemic heart disease with an adjusted OR 2.6 for ever versus never of solid fuel and significant trend across duration of use for stroke, hypertension and diabetes [32].

In addition, Right heart failure and pulmonary arterial hypertension were associated with HAP. Interestingly, women were almost two times more likely to present with pulmonary arterial hypertension (OR 1.72, 95% CI 1.17–2.55; p = 0.006), suggesting gender- related risk factors that may include HAP and HAP was also identified as a risk factor in women with isolated right heart failure in Kenya [84].

Most of the studies on metabolic diseases arose from ambient air studies [31, 32]. For example, a large study conducted in the United States reported that diabetes prevalence increases by 1% with each 10 $\mu g/m3$ $PM_{2.5}$ [85]. Since HAP has been

understudied with respect to cardiovascular disease most of our pathogenic hypothesis and more specific research is needed.

8. Reproductive and pregnancy health outcomes

There is limited but accruing evidence linking HAP exposure from solid fuel combustion with adverse maternal and perinatal outcomes. Also, epidemiologic evidence connecting AAP exposure with adverse pregnancy outcomes has been accumulating worldwide over the last two decades with several studies also attempting to summarize the available evidence [86].

In a meta-analysis by Amegah et al. including 19 studies, BMF use resulted in reduced birth weight, [86.4 g (95% CI: 55.5, 117.4)]. There was also an increased risk of low birth weight and stillbirth in those exposed to BMF, [OR (1.35, 95% CI: 1.23, 1.48)] and [OR (29, 95% CI: 1.18, 1.41)] respectively [86]. Interventional studies assessing HAP and low-birth weight are limited. However, Thompson et al. found an increase of 89 g in the birth weight of children of mothers using the intervention stoves (vs. open fires) (95% CI −27, 204), and reduced odds of a low birth weight child (OR 0.74, 95% CI 0.33–1.66) [87].

In a cross-sectional comparative study, Murkerjee and colleagues reported that there was positive association between BMF smoke exposure and menstrual irregularities such as irregular cycle (OR = 1.8, 95% CI 1.33–2.34), shortened menstrual cycle (OR = 5.1, 95% CI 3.62–9.21), spontaneous abortions (OR = 1.7, 95% CI 1.10–4.10), after controlling the potential confounders [88].

Agarwal and Yamamoto, using data from India's third National Family Health Survey (NFHS-3, 2005–2006) of 39,657 women aged 15–49 years who had a live birth in the previous 5 years, reported that women residing in houses using BMF had higher odd of reporting preeclampsia/eclampsia symptoms compared to those who reside in houses using cleaner fuels (OR = 2.21; 95%: 1.26–3.87; p = 0.006) [89]. This is further supported by a recent meta-analysis that showed maternal exposure to PM2.5 (per 10 $\mu g/m^3$ increment) is associated with elevated risk of preeclampsia (OR = 1.32, 95% CI 1.10 to 1.58%) especially in third trimester [90].

A recently published data from Ghana showed that using BMF was associated with adverse Apgar score at 5 min (aOR: 3.83, 95%CI: 1.44–10.11) and perinatal mortality (aOR: 7.6, 95%CI: 1.67–36.0) [91]. In sum, there are increasing evidences that BMF use are associated with menstrual irregularities, adverse pregnancy outcomes and perinatal morbidity and mortality.

9. Neurological health outcomes

There is a convergence of human, animal, and in vitro studies on the effects of air pollution on the brain, although most study has been associated with ambient PM [92]. Several neurological diseases have linked with exposure to air pollution ranging from neuro degenerative disease, to psychiatric disease, to neurodevelopmental and behavioural disorders in children.

Dementia denote memory loss and other cognitive abilities severe enough to interfere with daily life. Worse performance in tests evaluating episodic memory especially cognitive function tests had been associated with PM2.5 [93]. Other study has found associations between long-term exposure to PM2.5 among adults (>65 years) and neurodegenerative diseases such as Alzheimer's, and Parkinson's diseases [94].

Pre-natal maternal or child exposure to air pollutants during pregnancy, infancy or childhood (when the brain neocortex develops rapidly) has been related to delays in cognitive development in children [95, 96]. A metanalysis by Lam et al., involving 17 case–control, 4 ecological, 2 cohort studies, they documented that PM was associated with autism; summary odds ratios (ORs) of 1.07 (95% CI: 1.06, 1.08) per 10-μg/m^3 increase in PM_{10} exposure and 2.32 (95% CI: 2.15, 2.51) per 10-μg/m^3 increase in $PM_{2.5}$ exposure [97]. It has been postulated that air pollution induced oxidative stress can be related to dopaminergic neurotoxicity, and can cause depression and other neuropsychiatry disorder such as anxiety [93].

10. Conclusion

Exposure to air pollutants is one of the most important avoidable risks to health globally [98]. Air pollution has been termed the "silent killer" by the World Health Organization because its effects often go unnoticed or are not easily measured. Although the pulmonary and cardiac diseases have been the most studied adverse health, there are emerging evidence that air pollutants can damage any organ of the body and cause more ill-health. Chronic liver diseases [99], skin diseases [100], bone diseases [101] and autoimmune diseases [102] are increasingly been associated with air pollution. As data continues to increase on health effect of both HAP and AAP which are overlapping and contribute to each other, it is pertinent that all stakeholders; individuals, families, health care professionals, policy makers, governmental and non-governmental institutions work together to minimise the adverse health effects of the world's biggest environmental risk factor.

Acknowledgements

Abbreviations

AAP	Ambient air pollution
ALRI	Acute lower respiratory tract infection
AOR	Adjusted odd ratio
BMF	Biomass fuel smoke
HAP	Household air pollution
CI	Confidence interval
COPD	Chronic obstructive pulmonary disease
COVID-19	Corona virus disease 2019
DALYs	Disability-adjusted life years
DNA	Deoxyribonucleic acid
FEV1	Forced expiratory volume in 1 second
GBD	Global Burden of Disease Study
OR	Odd ratio
P	Significance value
PAHs	Polycyclic aromatic hydrocarbons
$PM_{2.5}$	Particulate matter with aerodynamic diameter 2.5 μm or less
PM_{10}	Particulate matter with aerodynamic diameter 2.5–10 μm

PM	Particulate matter
R^2	Coefficient of determination
ROS	Reactive oxygen species
WHO	World health organisation
CD	Cluster of differentiation

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