Public Health Focus

Targeting Household Air Pollution for Curbing the Cardiovascular Disease Burden: A Health Priority in Sub-Saharan Africa

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Household air pollution (HAP) is a major public health problem, particularly in sub-Saharan Africa where most of the populations still rely on solid fuels for cooking, heating, and lighting. This narrative review highlights the direct and indirect evidence of the important role of HAP in cardiovascular disease, especially in sub-Saharan African countries where highest rates of major cardiovascular disease and death are observed, and thus provides ample reason for promotion of preventive interventions to reduce HAP exposures in the region. There is an urgent need for efficient strategies to educate populations on the health issues associated with this health hazard, to provide affordable clean cooking energy for poor people and to promote improved household ventilation. High-quality data on household energy practices and patterns of HAP and related health issues are still needed for efficient policy making in this region. *J Clin Hypertens (Greenwich).* 2015;17:825–829.

Household air pollution (HAP) is a term used to describe health and environmental impacts of pollutants emanating from incomplete combustion of solid fuels burnt on open fires or traditional cookstoves within poorly ventilated homes.[1–3] Solid fuels include wood, animal dung, charcoal, crop wastes, and coal. Burning of these fuels can generate a number of chemicals, including gases (eg, carbon monoxide, nitrogen oxides, sulfuroxides), suspended liquid or solid particles, polyaromatic hydrocarbons (eg, benzene), heavy metals (eg, mercury), and other organic compounds.[2–4] The amount and type of pollutant released in ambient air greatly depends on the type of fuel used. Along with their health impact, released pollutants can significantly contribute to climate change.[3] The main unhealthy compounds are suspended particle matters (PM) of different diameters: <10 μm (PM_{10}), <2.5 μm ($PM_{2.5}$), and <0.1 μm ($PM_{0.1}$).[2,3] Despite a decline in the global proportion of households primarily using solid fuels for cooking, heating, and lighting, the absolute number of people relying on these fuels for these purposes has stabilized at around 2.7 to 2.8 billion over the past 3 decades owing to population growth.[1] According to the World Health Organization (WHO), 4.3 million premature deaths were attributable to HAP in 2012, mainly from respiratory and cardiovascular disease (CVD) and cancers.[5] HAP is responsible for nearly 5% of the global disease burden (expressed as disability-adjusted life-years), making it the single most important environmental risk factor globally.[6]

Increasing awareness of the adverse health effects of HAP, along with climate change and the growing demand for energy, have driven a global interest to reduce the domestic use of solid fuels. Initiatives such as the US Environmental Protection Agency's Partnership for Clean Indoor Air, the Global Alliance for Clean Cookstoves, the Initiative for Sustainable Energy for All in 2011, and hundreds of motivated nonprofit organizations have been launched to design and implement innovative and regionally specific solutions to provide clean cooking energy for poor people, to reduce the effect of deforestation and climate change, and ultimately reduce the burden of disease, especially diseases related to HAP.[7]

HAP AND CVD: HOW DO THEY CONNECT?

So far, only a few studies have investigated a direct link between HAP and cardiovascular outcomes (Table).[8–10] The most convincing is the large-scale population-based study that involved 14,068 adults in China. It found that household use of solid fuels was independently associated with self-reported coronary heart disease (odds ratio [OR], 2.58; 95% CI, 1.53–4.32), hypertension (OR, 1.70; 95% CI, 1.40–2.07), and diabetes (OR, 2.48; 95% CI, 1.59–3.86). Additionally, being in the highest tertile of the duration of solid-fuel exposure compared with the lowest tertile was associated with a history of stroke (OR, 1.87; 95% CI, 1.03–3.38).[8] Furthermore, a matched case-control study in Pakistan investigated the risk of acute coronary syndrome among rural women according to their use of solid fuel. The study found that after adjusting for confounders, the

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TABLE. Studies Investigating a Link Between Household Air Pollution and Cardiovascular Disease

Author (Year of Publication), Location	Population	Study Design	Exposure Variable	Cardiovascular Outcome	Key Findings	Comments
Lee et al (2012), China[8]	14,068 adults (54% women). Age ≥18 y, mean (SD) 49 (17) y. 19% hypertensive	Cross-sectional and retrospective analyses of random selection from census track data	Ever used solid fuel (coal or biomass) and duration of use	Coronary heart disease and stroke	Ever used solid fuel in home associated with an OR of 2.58 (95% CI, 1.53–4.32) increased odds of coronary heart disease. Compared with individuals in the lowest tertile of the duration of solid-fuel exposure, those in the highest tertile of the duration of solid-fuel exposure had a 1.87 increased odds of stroke (95% CI, 1.03–3.38)	Age, sex, smoking habits, secondhand smoking, level of education, body mass index, waist circumference
Fatmi et al (2014), Pakistan[9]	73 rural women presenting to the hospital with an acute coronary syndrome (mean age [SD] 56.3 [14.7] y) and 73 matched controls (mean age [SD] 55.2 [14.9] y) admitted to the hospital for other reasons	Matched case-control study	Use of solid fuel or use of natural gas, duration of use	Acute coronary syndrome	Current use of solid fuel was strongly associated with acute coronary syndrome (OR, 4.8; 95% CI, 1.5–14.8), and risk was lowest in women who had last used solid fuel more than 15 years earlier. The population-attributable fraction for acute coronary syndrome in relation to current use of solid fuel was 49.0% (95% CI, 41.3%–57.4%)	Adjusted for body mass index, educational status, type of kitchen, type of house, and use of ghee and meat
Alam et al (2012), Bangladesh[10]	22,337 rural adults (53.5% women). Age ≥18 y. 155,669 person-years of observation.	Retrospective cohort study. Comparison of cause-specific mortality (data obtained through verbal autopsy) between groups (solid-fuel vs gas-supplied households) over a 10-y period	Use of natural gas or solid fuel	Adult cardiovascular mortality over a 10-y period	Mortality caused by cardiovascular event was 5.1 and 4.8 per 1000 person-years in people from the solid-fuel and gas-supplied households, respectively. The incident rate ratio was 1.07 (95% CI, 0.82–1.41)	Household solid-fuel use was associated with a nonsignificant increased risk of cardiovascular mortality

Abbreviations: CI, confidence interval; OR, odds ratio; SD, standard deviation.

current use of solid fuel (wood, cow dung, crop residue, or coal) was strongly associated with acute coronary syndrome (OR, 4.8; 95% CI, 1.5–14.8) and that the risk was lowest in women who had last used solid fuel more than 15 years earlier. The population-attributable fraction for acute coronary syndrome in relation to current use of solid fuel was 49.0% (95% CI, 41.3%–57.4%).[9] Moreover, a retrospective cohort study in Bangladesh assessed adult cardiopulmonary mortality over a 10-year period in relation to household fuel type as a surrogate for exposure to indoor air pollution.[10] Cause-specific mortality data were obtained through verbal autopsy as part of a permanent surveillance. Mortality caused by cardiovascular events was 5.1 and 4.8 per 1000 person-years in people from the solid-fuel and gas-supplied households, respectively, and the incident rate ratio was 1.07 (95% CI, 0.82–1.41). Household solid-fuel use was therefore associated with a nonsignificant increased risk of cardiovascular mortality in this study.

Overall, whether the association between HAP and CVD in the before mentioned studies conducted in China and Pakistan is causal has yet to be confirmed. By contrast, there is consistent evidence that ambient outdoor air pollution and particularly PM are independent risk factors for all-cause CVD and cardiovascular mortality.[11,12] Similarly, active and secondhand smoking increase the occurrence of CVD as confirmed by smoking cessation strategies and implementation of public smoking bans across different geographical areas.[13]

HAP is a major source of ambient outdoor and indoor air pollution,[11] especially in impoverished societies, resulting in higher levels of $PM_{2.5}$ in those areas where it is widely spread.[1,14] Still, household use of solid fuels and secondhand smoke (reflecting one's smoking habits at home[13]) are both markers of indoor air pollution.[9] Moreover, ambient outdoor air pollution, secondhand smoke, and HAP share several common aspects. Indeed, each of the pollutant mixtures contains PM and toxic gases.[2] As these components are the triggers of CVD resulting from outdoor air pollution and secondhand smoke,[2,11,13] and given the important contribution of HAP to outdoor air pollution together with the association of secondhand smoke and HAP in indoor air pollution, it is likely that HAP is also causally associated with CVD. In this light, the hypothesis of HAP as an important cardiovascular risk factor is based on interpolation from risk functions for other forms of combustion-generated air pollution (ambient outdoor air pollution, secondhand smoke, and smoking habits at home),[6,15] the similarities in pollutant characteristics and exposure conditions, and the evidence of a generalized dose-response relationship between particulate air pollution and CVD.[2,12,16] In fact, ambient outdoor air pollution and secondhand smoke have been associated with various cardiovascular endpoints including autonomic[11,17,18] and endothelial dysfunctions,[11,19–21] heart rate abnormalities,[11,18] increased right heart pressure,[11,22] left and right ventricular hypertrophy,[11,22,23] heart failure,[11,24,25] coronary heart,[11,26–28] and cerebrovascular diseases.[29,30] These are all consequences of three main biological mechanisms induced by fine particles almost completely inhaled in the naso-oropharangeal tract[2,8–12,14–31]: (1) systemic inflammation and oxidative stress with release of proinflammatory markers, free radicals, and vasoactive molecules such as endothelin; (2) autonomic nervous system imbalance with the predominant role of the sympathetic nervous system over the parasympathetic nervous system; and (3) the penetration of PM or particle constituents (metals, organic compounds) into blood circulation.[11] Indeed, conduction disorders and cardiac arrhythmias can be generated by autonomic nervous system imbalance because of heart rate variability and by impaired myocardial blood flow and perfusion resulting from PM exposure.[11,12,17,18] Pulmonary hypertension may occur as a result of increased pulmonary vessels constriction caused by systemic inflammation and oxidative stress or to autonomic nervous system imbalance or to both.[11,14,22] Ventricular dysfunction reflected by heart failure has been shown to be caused by decrease in ventricular filling secondary to increased pulmonary resistance in healthy anesthetized rats after short-term inhalation of airborne PM-related environmentally persistent free radicals.[14] Ventricular hypertrophy itself emanating from myocardial fibrosis and ventricular remodeling could be attributed to local ventricular inflammation since PM exposure has been associated with systemic inflammatory and oxidative stress markers in the left ventricle.[31] Endothelial dysfunction, the initial step of atherosclerosis,[11] likely results from systemic inflammation as suggested by the association of both post-exposure $PM_{2.5}$ mass and tumor necrosis factor α (TNF-α) with the degree of endothelial dysfunction in one study.[31] Owing to its prothrombotic and systemic inflammatory effects, $PM_{2.5}$ exposure is associated with the progression of atherosclerosis, which is an inflammatory disease.[11,32] Coronary heart disease and cerebrovascular diseases can be explained by the increased frequency of atherothrombosis.[11,13] Individuals with preexisting coronary artery disease, ie, presenting with decreased coronary reserve and oxygen supply, are particularly susceptible to myocardial infarction[11,12,31] following PM exposure as a consequence of reduced ventricular filling and thus cardiac output. More so, PM inhalation is associated with increased hospital admissions and death from myocardial infarction[11,31] as a result of concomitant decrease of cardiac output/coronary flow and oxidative stress. Likewise, systemic inflammation and rapid progression of atherosclerosis might explain the excess stroke-related mortality associated with air pollution.[29] Despite the high CVD burden attributed to ambient outdoor air pollution, the relationship between HAP and CVD has been less studied probably because air pollution has less been regarded as a

putative trigger of CVD in low- and middle-income regions such as sub-Saharan Africa, which are more concerned by the adverse health effects of HAP.[14]

HAP AND CVD IN SUB-SAHARAN AFRICA: WHAT DO WE KNOW AND WHAT IS TO BE DONE?

Unlike in other parts of the world where the proportion of households mainly relying on solid fuels has declined over the past three decades, it increased up to 77% in sub-Saharan Africa. The absolute number of people still exposed to smoke from burning of solid fuels almost doubled from 333 million to 646 million. Thus, sub-Saharan Africa is the region with the highest burden of HAP.[1] The distribution of solid-fuel utilization in households varies widely across and within countries, with proportions up to 100% in rural areas.[3] Moreover, indoor pollutants' concentrations are far above the WHO recommended levels. For instance, the mean 24-hour PM_{10} concentration in the indoor air of developing countries is 15 to 150 times greater than the recommended 20 $\mu g/m^3$.[3] As a result, HAP is the most important environmental risk factor for disease in the region.[6]

More than 80% of worldwide CVD deaths occur in sub-Saharan Africa along with other low- and middle-income countries (LMICs).[33] Sub-Saharan Africa is experiencing a growing epidemic of hypertension and diabetes mellitus, both of which are CVD triggers.[33,34] However, traditional cardiovascular risk factors seem insufficient to explain the huge burden of CVD in LMICs. Indeed, a recent study revealed that although the risk-factor burden (using the INTERHEART risk score) was lowest in low-income countries, the rates of major CVD and death were substantially higher in low-income countries than in high-income countries.[35] This discrepancy seemed unexplained by the prevalence of traditional cardiovascular risk factors such as hypertension and diabetes, suggesting that traditional cardiovascular risk scores might underestimate cardiovascular risk among populations in sub-Saharan Africa alongside other LMICs. Consequently, nontraditional risk factors such as socioeconomic status,[36] infectious diseases,[37] and HAP should be considered while assessing cardiovascular risk among sub-Saharan African populations.

Given that sub-Saharan Africa already experiences the heaviest burden of disease attributable to CVD,[33] and that this region is also the most exposed to HAP,[1] an important impact of HAP on CVD in sub-Saharan African populations might constantly and more rapidly increase death rates if nothing is done to tackle the situation. Despite the lack of data to adequately inform policies on the burden of CVD attributable to HAP in sub-Saharan Africa, there is an urgent need to strengthen the fight against this scourge. That fight requires self-consciousness of the problem and collaboration between African leaders including health authorities, researchers, politicians, energy suppliers, and leaders from civil societies' movements. Experience from other parts of the world such as Asia or South America that are already overcoming this problem should be capitalized by sub-Saharan African countries.[38] Strategies should include intensive public education emphasizing on the dangers of HAP; promotion of and increase in the access to cleaner, safer, and more efficient household energy fuels such as liquefied petroleum gas, natural gas, biogas, solar energy, and electricity; and improvement of household ventilation.[38] For this purpose, the WHO has recently issued new recommendations on indoor air quality pertaining to household fuel combustion. These recommendations aim to help governments and their partners provide access to cleaner technologies that reduce indoor pollution and lower disease burden.[39]

Research is another crucial point in the fight against HAP in sub-Saharan Africa. There remains important knowledge gaps on the amplitude and characteristics of HAP and its health impacts in the region that need to be filled to provide evidence-based prevention strategies.[36,37] Household energy practices and ventilation that influences the intensity and composition of HAP exposure need to be deeply investigated in the region. Large and well-designed studies will help to assess personal exposure, to define dose-response relationships, and to better identify associated health risks and their pathogenesis. Such data are critical to determine cookstove interventions that can efficiently reduce exposure and consequently improve health outcomes. Ongoing large, randomized cookstove intervention trials with high-quality, quantitative exposure monitoring assessments in Ghana (NCT01335490) and Malawi are intended to provide these important data. However, their results might not be representative of the whole of Africa, pointing out the importance to expand such research projects to the whole region.

CONCLUSIONS

HAP is a major public health problem worldwide and particularly in sub-Saharan Africa. There is indirect evidence suggesting that HAP may be an important cardiovascular risk factor among populations in sub-Saharan African countries alongside populations from other LMICs. HAP should therefore be a major target for curbing the growth of CVD in these countries. To tackle the burden of HAP, populations need to be educated on health issues associated with this health hazard, clean fuels and lower emission solid-fuel stoves should be promoted over solid fuels, and household ventilation improved. High-quality data on the health issues related to HAP should be gathered as a prelude to efficient policy-making. Particularly, there is a critical need for strong direct evidence linking HAP with cardiovascular events.

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