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RESEARCH ARTICLE

Impact of exposure to biomass on the vascular function of Senegalese women

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ABSTRACT

Background: In rural Senegal, biomass fuels are the main and often the only source of domestic energy for cooking. Their combustion is a source of particles and many other chemical contaminants that could alter cardiovascular function. **Aims and Objectives:** The aim of our study was to assess the effects of exposure to biomass fumes on vascular function in non-smoking women in rural Senegal. **Materials and Methods:** We conducted a cross-sectional 8-month study of 64 women (32 exposed to biomass smoke and 32 unexposed controls) who were active or housewives and involved in cooking. The subjects were aged 33.56 ± 9.34 years and 30.22 ± 6 years, respectively. All the women received a questionnaire with questions on sociodemographic characteristics, habitat characteristics, cooking habits, and biomass exposure conditions. Flow-mediated brachial arterial vasodilation flow-mediated dilatation (FMD) expressed as a percentage of the diameter was measured basally at rest and post-compression at 5, 30, 60, 90, and 120 s and 10 min after deflation. **Results:** All participants were non-smokers. 12.5% of subjects used gas in addition to biomass. Seniority was 18. 90 ± 10 years, with a mean daily biomass exposure of 4 ± 1 h. The analysis of cardiovascular parameters and blood sugar found no significant difference between the two groups. Kinetic FMD comparisons between the two groups showed significant differences at points 60 and 90 s 10 min post-occlusion. A negative correlation was found between FMD at 90 s after deflation and the duration of exposure in exposed women ($r = 0.44$ $P < 0.05$). **Conclusion:** Our results showed that exposure to biomass is implicated in the appearance of vascular dysfunction in non-smoking women.

KEY WORDS: Biomass; Cow Dung; Wood; Vascular Dysfunction; Flow-mediated Dilatation

INTRODUCTION

Modernization in developed countries has been accompanied by a shift in fuels and from biomass to petroleum derivatives and electricity. In developing countries, although there are cleaner and more sophisticated fuels, primary biofuels are used on a large scale. One-third of people living in rural communities in developing countries use biomass as their sole source of energy.[1,2] In Senegal, women living in a rural environment use fuels from biomass: Cow dung, mainly from domestic animals, especially the cow. Air pollution from the combustion of biomass is considered an important risk factor for respiratory human health since it is responsible for about 1.6 million premature deaths worldwide.[3] Raw materials of these fuels are burnt in open fires and inefficient stoves. As a result of incomplete combustion, small suspended particles of <2.5

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μm emerge in the domestic environment particulate matter2.5 (PM2.5) and/or <10 μm (PM10).[4] Such combustions form complex and very heterogeneous mixtures with many other pollutants such as polycyclic organic compounds and gases such as carbon monoxide and formaldehyde, a major source of air pollution inside homes.[5,6] Chronic exposure to fine particles could contribute to the onset of atherosclerosis, high blood pressure and type 2 diabetes[7] and is associated with increased risk of adverse cardiovascular outcomes[8-11] resulting in reduced life expectancy.[12] The objective of our study is to evaluate the effects of exposure to biomass fumes on vascular function in rural non-smoking women in Senegal by a comparative study.

MATERIALS AND METHODS

Participants

The protocol of the study was approved by the Ethics Committee of the University of Thies. We conducted a cross-sectional study conducted over 8 months between July 2017 and February 2018 among 64 premenopausal African women, non-smokers (32 exposed to biomass smoke and 32 unexposed controls), and living in the villages of the region of Thies. These were housewives, implicated in cooking. The exposed group consisted of women exposed to biomass smoke. These subjects used cow dung as the main source of domestic energy for cooking. During the 3-month rainy season, these women used wood instead of dried dung. The control group included 32 women not exposed to biomass inhalation products. They were from the same village as the women exposed but reside in urban areas due to their professional activities. There was no polluting industry around the study area. Oral consent was obtained from all participants. All the women had an anonymous questionnaire that included sociodemographic, habitat, cooking habits, and biomass exposure conditions: Number of hours of exposure per day, number of years of exposure, and persisting exposure at the time of the survey or not.

Exclusion criteria were pregnancy, active and passive smoking, and chronic diseases such as diabetes and high blood pressure.

Anthropometric, Biochemical, and Cardiovascular Parameters

Body weight and height were measured, and body mass index (BMI) was calculated dividing weight in kilograms by height in meters squared (kg/m^2). Systolic and diastolic blood pressure (DBP) was manually measured in the left arm using a manual sphygmomanometer in standardized position and after 30 min of rest. Biochemical parameters such as plasma lipids total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL), cholesterol, and glycemia were evaluated using standard enzymatic methods.

FMD

Flow-mediated dilatation (FMD) of the brachial artery was measured by the same experienced cardiologist and by ultrasound according to the guidelines described by Corretti *et al*.[13] Brachial artery ultrasonography was performed after resting in the supine position for 15 min and the fasting state. All participants refrained from drinking beverages containing caffeine or alcohol for 12 h before the examination and were also advised not to take antihypertensive or vasodilator drugs the day of examination. Patients were examined in a quiet and temperature-controlled room (25°C). The right arm was extended and immobilized with an angle of ˜60° from the trunk of the body. A 10-MHZ linear transducer connected to an ultrasound device (sonoline G50; siemens) was placed on the brachial artery at 1–2 cm proximal to the elbow joint. After scanning the baseline artery diameter, the cuff was rapidly inflated to 50 mm Hg above systolic blood pressure (SBP) and kept for 5 min. By rapid deflation of the cuff, reactive hyperemia was induced and scanning was performed at 5, 30, 60, 90, and 120 s and 10 min after cuff deflation to obtain the FMD, expressed in percentage of the baseline diameter (%FMD). Due to limited technical (software) resources, we were not able to capture the diameter continuously. FMD was measured in duplicate for each patient with at least 1h in resting condition between the two measurements, and the mean of the two FMD values was calculated. A difference of <10% between the two measurements was considered as acceptable. The cardiologist who performed FMD was blinded to the diagnostic of the patient.

Statistical Analysis

Data collection was made from individual forms of collection and input using Excel 2013. The statistical analysis was performed using the STATA software version 11.0. The results were expressed in mean standard deviation and percentage. Pearson correlation test was used in looking for links between parameters. The threshold of significance was set at $P < 0.05$.

RESULTS

Our results showed that there is no significant statistical difference between the two groups regarding age and BMI [Table 1]. The average age of subjects exposed and unexposed to biomass smoke was 33.56 ± 9 years and 30.22 ± 6 years, respectively. In the exposed group, the average duration of exposure to biomass was 18 years with extremes ranging from 4 to 33 years. The biomass most frequently used by the exposed population in culinary activities was dominated by the combination of cow dung and wood 87.5%. The wood is being used only during the rainy season. In contrast, 12% of the subjects used butane gas in addition to these two solid fuels.

The type of kitchen used was for the most part of the closed type absolutely without a chimney or without window [Figure 1].

Table 1: Characteristics of the population

Characteristics	Exposed women *n*=32	Women checks *n*=32	*P*-value
Age (years)	33.56±9	30.22±6	0.08
BMI (kg/m²)	21.37±7.79	23.05±4.08	0.39
Duration of exposure to biomass (years)	18.90±10	NA	*P*<0.05
Number of hours of exposure per day	4±1	NA	*P*<0.05
Cow dung+wood	87.5	NA	*P*<0.05
Cow dung+wood+gas	12.5	NA	*P*<0.05

BMI: Body mass index, NA: Not applicable

Table 2: Cardiovascular and biochemical parameters of subjects exposed to biomass and checks women

Parameters	Exposed women *n*=32	Women checks *n*=32	*P*-value
SBP (mmHg)	126.40±19.90	117.72±7.97	0.06
DBP (mmHg)	80.77±7.98	74.15±6.8	0.31
Glycaemia (g/l)	1,87±1.19	0.91±0.31	0.07
Total cholesterol (g/l)	1.62±0.41	1.75±0.33	0.24
LDL cholesterol (g/l)	1.24±0.39	1.31±0.41	0.07
HDL cholesterol (g/l)	0.54±0.16	0.80±0.65	0.20
Triglycerides (g/l)	0.51±0.24	0.47±0.16	1.00

DBP: Diastolic blood pressure, SBP: Systolic blood pressure, LDL: Low density lipoprotein, HDL: High density lipoprotein

There was no difference between the two groups of lipid profile total cholesterol, triglycerides, HDL, and LDL cholesterol [Table 2]. The same observation was made for the plasma glucose and cardiovascular parameters no difference for systolic and diastolic pressures [Table 2].

At rest, with basic conditions we noted in Figure 2 slight vasodilatation in subjects exposed to biomass compared to the controls. However, the kinetics of FMD in the different times (5 s, 30 s, 60 s, 90 s, and 120 s and 10 min after deflation) between the two groups showed a significant decrease in vasodilation in subjects exposed to biomass compared to controls at different points, at 60 s (P = 0.007) and 90 s (P = 0.005), and 10 min (P = 0.003) after deflation. A negative correlation [Figure 3] was found between FMD at 90 s after deflation and the duration of exposure in exposed women (r = 0.44 P < 0.05).

DISCUSSION

Biomass combustion produces relatively high amounts of suspended PM. The dramatic increase in suspended PM results from the production, by incomplete combustion of a mixture of soot and hydrocarbons which are creosote and tar. Almost all of these particles are <3 μm in diameter and can be considered inhalable that is to say capable to penetrate deeply the lungs.

Chronic exposure to biomass causes vascular/endothelial dysfunction. Our results show globally that the women

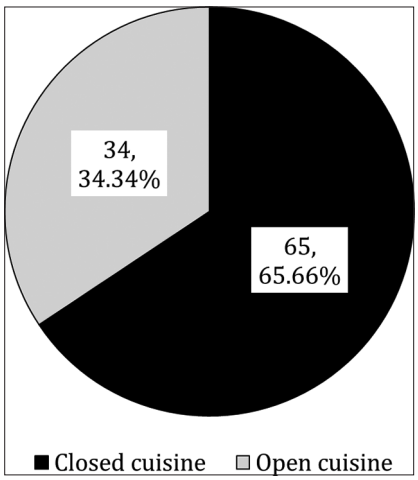

Figure 1: Type of cuisine

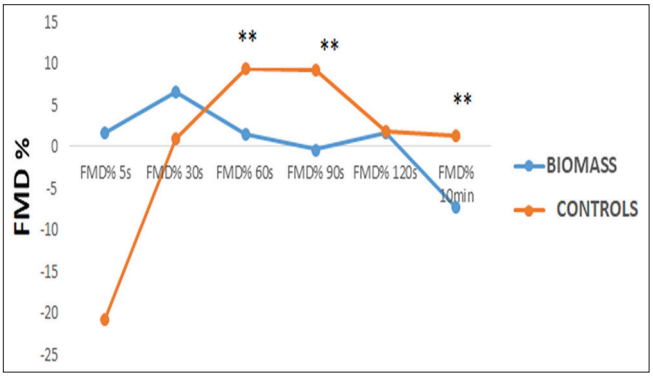

Figure 2: Comparison of the kinetics of the flow-mediated dilatation of the two groups

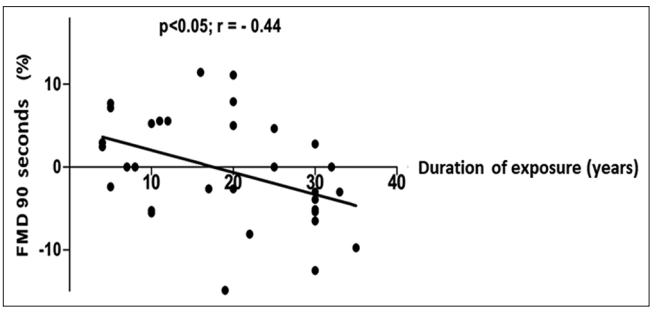

Figure 3: Correlation between flow-mediated dilatation 90s and the duration of exposure

exposed to this biomass have abnormal FMD kinetics compared to those that have not been exposed. Furthermore, our results suggest that the vascular dysfunction observed in these women is associated primarily to exposure to biomasses, which has the capacity to carry on their surfaces, toxic chemicals in certain cases (benzopyrenes) and by their small size, allowing them to cross the capillary-alveolar membrane and to be found in the blood.

The deleterious effects of chronic exposure to biomasses on the cardiovascular system are widely suspected in the literature.[14] In fact, studies show that long-term exposition to biomass is associated with a number cardiovascular risk factors such as high blood pressure and diabetes.[7] This leads to the development of arteriosclerosis[8,15] and an increase in the prevalence of cardiovascular morbidity and mortality.[10,11] The cardiovascular profile between the two groups was statistically similar (SBP 126.40 ± 19.90 vs. 117.72 ± 7.97 mm Hg and [DBP] 80.77 ± 7.9 vs. 74.15 ± 6.8 mm Hg for exposed and female controls, respectively). For blood glucose, no significant difference was found (1.87 ± 1.15 g/l and 0.91 ± 0.31 g/l for exposed and female controls, respectively). Our results point in the same direction as those of Buturak *et al*.[16] who evaluated endothelium-dependent vasodilation in individuals exposed to biofuels, their results showed significant decrease vasodilation compared to controls.

The precise mechanism by which endothelium-dependent vasodilation is reduced in individuals who have long-term exposure to biomass smoke is unknown,[16] although several studies have been conducted to elucidate the possible mechanism of cardiovascular damage caused by air pollutants. Impairment of endothelial function may result from chronic exposure to toxic PM, hydrocarbons, oxygenated organic compounds, free radicals,[8,17] carbon monoxide,[18] and oxidative stress.[19,20] Vascular oxidative stress is closely related to cardiovascular diseases.[21,22] Circulating levels of oxidative stress markers such as malondialdehyde,[19] the protein carbonyl[20] increases in patients exposed to biomass smoke.[19,20] However, the mechanism by which particles induce oxidative stress remains undefined. Rongson *et al*.[23] have shown that ultrafine particles (diameter <0.1 μm PFU) stimulate superoxide production by endothelial cells in part, mediated by the activation of a kinase of the mitogen-activated protein kinase family which leads to the production of reactive oxygen species cellular in cells treated with stress stimuli. Superoxide radicals, produced as a result of oxidative stress, combine with nitric oxide (NO) to form peroxynitrite, reducing the bioavailability of NO. In vascular smooth muscle cells, superoxide inhibits the activity of enzymes such as guanylyl cyclase[24] and cGMP dependent kinase protein,[25] thereby reducing endothelium-induced and NO-dependent vasodilatation. Several *in vitro* studies of cultured cells showed that wood smoke increased the expression and production of pro-inflammatory cytokines.[26,27] These results are in agreement with those of Barregard *et al*.[28] who reported a modest increase in pro-inflammatory mediators in subjects exposed to wood smoke. Dutta *et al*.[29] observed an increase of systemic inflammation in women who were cooking with biomass fuels, as well as a positive association between inflammatory markers and environmental levels of PM10 and PM2.5. In fact, exposure to pollution of air by fine PM2.5 particles could lead to the release of pro-inflammatory cytokines such as interleukin-6 or tumor necrosis factor α from alveolar macrophages[30] or peritoneal.[31] This systemic inflammation due to the inhalation of fine PM2.5 particles alone could contribute to pro-atherogenic changes in the vascular wall.[32]

CONCLUSION

Exposure to biomass leads to atmospheric pollution probably related to fumes from the combustion of biomass. This phenomenon could lead to vascular dysfunction which constitutes a major cardiovascular risk factor. These results suggest the need to implement programs that will reduce this risk of biomass exposure in rural Senegal. Finally, despite the limitations of our study, such as sample size, this study can be considered as a starting point for further prospective studies including larger numbers of individuals and long-term follow-up to elucidate the possible links between chronic exposure to biomass smoke and vascular function.

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