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Effects of a Cookstove Intervention on Cardiac Structure, Cardiac Function, and Blood Pressure in Western Kenya

To the Editor:

Exposure to household air pollution (HAP) is responsible for 2.9 million to 4.3 million deaths annually.¹ The burden of HAP is greatest in sub-Saharan Africa, where the majority of homes rely on traditional cooking practices.² Of the numerous potentially toxic environmental pollutants in HAP, most of the literature has focused on carbon monoxide (CO)

and fine particulate matter <2.5 μ m in diameter (PM_{2.5}). There is a paucity of direct evidence linking HAP and cardiovascular disease outcomes or intermediate surrogate markers in low- and middle-income countries. Few studies, including some of our own work,³ have measured the association between HAP and cardiac structure or function by echocardiography, but the results have been mixed.⁴⁻⁶ To address some of these gaps in knowledge, we recently introduced a cookstove that reduces household levels of CO and PM_{2.5}, measured blood pressure (BP), and performed echocardiography to assess its impact on cardiac structure and function.

This was a longitudinal nested cohort study of women who participated in a parent cookstove intervention study. The methods for this study have been previously published³ and are described here briefly. The cookstove intervention study used a locally improved cookstove, the Eldoboma cookstove. Key design features include containment of fuel in an enclosed oven and chimney shielding outside the kitchen. In independent testing by the Centre for Research in Energy and Energy Conservation (Kampala, Uganda), the Eldoboma stove exceeded goals for overall emissions and indoor emissions. Participants were recruited before the cookstove intervention, and a series of study measures were obtained before an Eldoboma stove was issued to each of them. Additional study data were collected before and at 1 and 6 months after the cookstove intervention.

A sample size of 40 participants was determined to be able to detect a 5 mm Hg minimum detectable difference (14% change) in right ventricular (RV) systolic pressure after the cookstove intervention on the basis of previous literature,⁴ with a two-sided α level of 0.05 and 80% power. Inclusion criteria were being a woman \geq 18 years of age who reported spending at least 4 hours per day in the kitchen. Active smokers were excluded. Data were collected between December 2013 and November 2014. The study protocol was approved by the human subjects committees at Duke University, Stanford University, and Moi University.

HAP measures included CO measured in parts per million using the EasyLog USB CO Monitor (Lascar Electronics, Eerie, PA) and PM_{2.5} measured in micrograms per square meter using the Personal Data Ram 1000AN (Thermo Fisher Scientific, Waltham, MA) suspended from the ceiling in the center of the kitchen. Devices were zeroed and calibrated before each measurement. Nephelometric PM_{2.5} measurements were adjusted for relative humidity using the methods of Klasen *et al.*⁷ Median values of air pollution levels in the kitchen across \geq 20 hours of recording time were used as proxies for individual participant exposure specifically from their home cookstoves.

A certified nurse collected all physical measurements, including BP, height, and weight, in the home. Echocardiography was performed in the home or at a nearby health facility by a trained technologist using a standard protocol. The echocardiographic imaging protocol included two-dimensional, color, and spectral Doppler and M-mode imaging from traditional imaging windows using a Philips CX-50 machine (Philips Healthcare, Bothell, WA). Images were analyzed using the Philips Xcelera cardiology image information management system (Philips Healthcare). All measures were calculated from an average of three consecutive cardiac cycles. Two cardiologists and one cardiac sonographer performed and verified all echocardiographic measures. Table 1 lists the echocardiographic measurements that were assessed. Right atrial pressure was assessed by inferior vena cava dynamics. The images were transferred to the Duke Cardiovascular Diagnostic Unit for analysis of myocardial strain using TomTec software (TomTec Imaging Systems, Unterschleissheim, Germany). Two-dimensional strain derived from speckle-tracking imaging was also used to measure RV and left ventricular (LV) function. RV global



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Characteristic	Baseline (n = 44)	1-mo follow-up (<i>n</i> = 43)	6-mo follow-up (<i>n</i> = 42)	P_{trend}
Systolic BP, mm Hg	123 ± 8	116 ± 8	115 ± 7	<.001
Diastolic BP (mm Hg)	80 ± 5	74 ± 5	74 ± 5	.005
Right atrial minor axis (cm)	4.0 ± 0.6	3.7 ± 0.6	4.0 ± 0.6	.780
RV basal diameter (cm)	$\textbf{3.0} \pm \textbf{0.7}$	3.5 ± 0.5	3.5 ± 0.5	.010
Tricuspid annular plane excursion (cm)	2.4 ± 0.5	2.3 ± 0.4	2.3 ± 0.4	.180
Pulmonary arterial diastolic pressure (mm Hg)	7.4 ± 3.6	7.5 ± 3.9	7.6 ± 3.1	.895
Right ventricular systolic pressure (mm Hg)	26.4 ± 7.0	21.8 ± 7.2	26.3 ± 7.2	.344
Mean pulmonary arterial pressure (mm Hg)	14.1 ± 4.5	12.2 ± 4.4	13.9 ± 3.0	.833
LV ejection fraction (%)	62 ± 6.0	59 ± 4.7	58 ± 6.9	.001
LV GLS (%)	-21.1 ± 3.3	-20.7 ± 2.2	-20.7 ± 2.5	.592
RV GLS (%)	-22.5 ± 4.6	-23.4 ± 2.8	-22.4 ± 4.0	.394

Table 1	BP and echocardiogra	phic characteristics	of the study sa	ample over time or	n follow-up

Data are expressed as mean \pm SD.

longitudinal strain (GLS) assessment was performed in the apical fourchamber view using the RV free wall and septum. GLS of the left ventricle was averaged from the results of the segmental peak systolic strains of apical four-, three-, and two-chamber views.

The mean age of the 44 female participants was 34.5 ± 6.4 years at baseline, as described in our publication of baseline parameters in this cohort.³ A robust linear mixed-effects model⁸ accounting for repeated measures demonstrated that the cookstove intervention was associated with a decrease in 24-hour CO levels from a median of 13.8 ppm (interquartile range IIQRI, 7.0–27.8 ppm) at baseline to 7.0 ppm (IQR, 0.0–15.5 ppm) at 1 month following the cookstove intervention. Similarly, the 24-hour median PM_{2.5} level decreased from 57.0 μ g/m³ (IQR, 32.0–84.3 μ g/m³) at baseline to 23.9 μ g/m³ (IQR, 8.0–62.7 μ g/m³) at 1 month following the stove intervention, before rebounding to 30.0 μ g/m³ (IQR, 5.4–77.7 μ g/m³) at 6 months after stove intervention. Level of education, age, and body mass index did not have appreciable effects on the within-person longitudinal change in HAP level following the intervention.

Similar models, adjusted for age, body mass index, and level of education, showed that most echocardiographic parameters remained within the clinical range of normal at each time point (Table 1). Mean RV systolic pressure decreased from 26.4 ± 7.0 mm Hg at baseline to 21.8 ± 7.2 mm Hg at 1 month postintervention; however, this decrease was not sustained at 6 months postintervention. There were no statistically significant changes in RV or LV GLS. Mean LV ejection fraction decreased from baseline to 6 months but remained in the normal range, irrespective of baseline CO level. Systolic BP decreased significantly from 123.2 mm Hg (95% CI, 118.9– 127.5 mm Hg) before the intervention to 115.0 mm Hg (95% CI, 110.7–119.2 mm Hg) 6 months after the cookstove intervention (P < .001). Similar results were observed for diastolic BP (6 mm Hg decrease), pulse pressure (2 mm Hg decrease), and mean arterial pressure (6 mm Hg decrease).

We evaluated whether the longitudinal changes in BP and echocardiographic parameters were associated with the longitudinal change in HAP levels. An IQR decrease in log-transformed CO was associated with small changes in LV ejection fraction (-1.1% [-1.8% to -0.04%] per IQR ppm decrease) and LV GLS (0.31% [0.02% to 0.59%] per IQR ppm decrease). Longitudinal changes in CO and PM_{2.5} were associated with a 1.8 mm Hg (95% Cl, -3.1 to -0.6 mm Hg; P = .006) and a 2.7 mm Hg (95% Cl, -4.3 to -1.0 mm Hg; P = .002) lower systolic BP, respectively. There were no statistically significant associations between per IQR longitudinal change in $PM_{2.5}$ and change any of the echocardiographic parameters.

We found that cookstove replacement was associated with a pronounced and statistically significant decrease in household CO, more so than $PM_{2.5}$. Of the echocardiographic parameters, we found an unexpected small decrease in LV ejection fraction 6 months after the stove intervention, which remained in the normal clinical range. Concurrently, both systolic and diastolic BP were significantly reduced after cookstove replacement, with a stepwise decrement corresponding to decreases in CO level.

Studies that have investigated the association between HAP and cardiac structure or function by echocardiography have shown inconsistent findings. Most have used echocardiography at one point in time.⁴⁻⁶ Few studies have investigated longitudinal echocardiographic parameters in association with a cookstove intervention, which distinguishes this study from prior research. Our main findings support the observed lack of an association between cookstove intervention and any meaningful changes in left or RV structure or function,⁵ at least within 6 months following the intervention. Although there was a statistically significant decrease in RV diameter and LV ejection fraction following stove intervention, these changes were small and did not cross abnormal clinical thresholds. Given significantly different study designs and research questions, it is difficult to directly compare our longitudinal results with prior cross-sectional research. Moreover, important differences in study populations' age, sex, and absence of significant echocardiographic abnormalities at baseline, which could partially account for the disparate results.

Longitudinal changes in BP following stove replacement were statistically significantly associated with longitudinal changes in both CO and $PM_{2.5}$, although without complete concordance between these two measures of HAP. Our results confirm findings from prior research showing that cookstove interventions can reduce BP in women up to 6 months postintervention.⁹

We acknowledge some limitations in generalizing our findings, including our sample size and population, which included young women without significant cardiac disease at baseline and unknown exposure to secondhand smoke. We did not include a comparator arm and therefore lack a nonintervention control group with which to compare our findings and rule out, for example, other time-varying changes that might have influenced BP. It is also possible that the null effect we observed on cardiac structure and function was due to insufficient lowering of HAP levels or insufficient follow-up time. An intervention that lowered HAP levels further or a longer period of postintervention observation may have shown different results.

Given the significant morbidity and mortality associated with HAP exposure and dearth of outcome data, more investigation into additional cardiovascular surrogate outcomes remains a high priority. The lack of an association between HAP reduction and these specific echocardiographic markers could suggest that alternative echocardiographic measures should be considered in future studies in this field. Future studies may also focus on those with preexisting, or at high risk for, cardiopulmonary disease to detect changes in echocardiographic measures over time.

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