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Household air pollution from biomass-burning cookstoves and metabolic syndrome, blood lipid concentrations, and waist circumference in Honduran women: a crosssectional study

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Authors' contributions

SR performed the data collection, analyzed the data and wrote the manuscript. BNY performed the data collection and wrote the manuscript. MLC designed the study and revised the manuscript. MLBC performed the data collection and revised the manuscript. AMB designed the study and revised the manuscript. RDB designed the study and revised the manuscript. TLN revised the manuscript. JV supported the data collection and revised the manuscript. SJR revised the manuscript. CL supported the data collection and revised the manuscript. NG supported the data analysis and revised the manuscript. KK revised the manuscript. SA supported data collection and revised the manuscript. ABOP supported data collection and revised the manuscript. JLP designed the study and revised the manuscript. All authors read and approved the final manuscript.

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Declarations

The study protocol was approved by the Institutional Review Board at Colorado State University.

Competing interests

The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. Two of the authors, Sebastian Africano of Trees, Water & People (TWP), and Anibal Benjamin Osorto of Asociación Hondureña para el Desarrollo (AHDESA), are members of the implementing non-governmental organizations that deploy the cookstove technology studied in this paper. Results of research like this are often shown as evidence of the effectiveness of this particular cookstove technology in TWP and AHDESA publications, including blogs, articles, and grant proposals, which may lead to future funding of these initiatives by individual and/or institutional supporters of the respective organizations.

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Abstract

Background: Household air pollution from cooking with solid fuels affects nearly 3 billion people worldwide and is responsible for an estimated 2.5 million premature deaths and 77 million disability-adjusted life years annually. Investigating the effect of household air pollution on indicators of cardiometabolic disease, such as metabolic syndrome, can help clarify the pathways between this widespread exposure and cardiovascular diseases, which are increasing in low- and middle-income countries.

Methods: Our cross-sectional study of 150 women in rural Honduras (76 with traditional stoves and 74 with cleaner-burning *Justa* stoves) explored the effect of household air pollution exposure on cardiovascular disease risk factors. Household air pollution was measured by stove type and 24-hour average kitchen and personal fine particulate matter [PM_{2.5}] mass and black carbon concentrations. Health endpoints included non-fasting total cholesterol, high-density lipoprotein, calculated low-density lipoprotein, triglycerides, waist circumference to indicate abdominal obesity, and presence of metabolic syndrome (defined by current modified international guidelines: waist circumference ≥ 80 cm plus any two of the following: triglycerides >200 mg/dL, HDL <50 mg/dL, systolic blood pressure ≥ 130 mmHg, diastolic blood pressure ≥ 85 mmHg, or glycated hemoglobin >5.6%).

Results: Forty percent of women met the criteria for metabolic syndrome. The prevalence ratio [PR] for metabolic syndrome (versus normal) per interquartile range increase in kitchen PM_{2.5} and kitchen black carbon was 1.16 (95% confidence interval [CI]: 1.01 – 1.34) per 312 µg/m³ increase in PM_{2.5}, and 1.07 (95% CI: 1.03 – 1.12) per 73 µg/m³ increase in black carbon. There is suggestive evidence of a stronger effect in women ≥ 40 years of age compared to women <40 (p-value for interaction = 0.12 for personal PM_{2.5}). There was no evidence of associations between all other exposure metrics and health endpoints.

Conclusions: The prevalence of metabolic syndrome among our study population was high compared to global estimates. We observed a suggestive effect between metabolic syndrome and exposure to household air pollution. These results for metabolic syndrome may be driven by specific syndrome components, such as blood pressure. Longitudinal research with repeated health and exposure measures is needed to better understand the link between household air pollution and indicators of cardiometabolic disease risk.

Keywords

indoor air pollution; cookstoves; cardiovascular disease; global health; Latin America

Introduction

More than 40% of the world's population, mostly in low- and middle-income countries, relies on solid fuels for daily cooking activities (1). Cooking and heating homes with solid fuels, such as wood, coal, charcoal, animal dung, and crop residues, on inefficient stoves is the main source of household air pollution, which is a leading environmental risk factor for the global burden of disease (2). In 2016, household air pollution was responsible for an

estimated 2.5 million premature deaths and 77 million disability-adjusted life years (3). Cardiovascular disease constitutes a substantial burden of mortality and disabilities from exposure to household air pollution from indoor solid fuel combustion (4). To better understand the magnitude of the disease burden from cooking and heating homes with solid fuels, there is a pressing need to investigate a wider range of health endpoints, including indicators of cardiometabolic disease risk that may be impacted by household air pollution (5).

Cardiovascular disease and its associated risk factors are increasing in prevalence in many low- and middle-income countries (6–8). Dyslipidemia (raised triglycerides or cholesterol, lowered HDL [high-density lipoprotein]) and metabolic syndrome increase the risk of cardiovascular disease and diabetes (9). Metabolic syndrome is a cluster of at least three specific medical conditions related to cardiometabolic disease that occur together, including elevated blood pressure, dyslipidemia, elevated fasting glucose, and abdominal obesity (10). The combination of these risk factors is linked to adverse cardiovascular events, as it is estimated that those with metabolic syndrome are twice as likely to die from and three times as likely to have a heart attack or stroke compared to people without the syndrome (9). While lifestyle, socioeconomic, and ethnic differences largely impact increasing rates of metabolic syndrome worldwide, environmental risk factors, such as air pollution, may also have important effects (11).

Investigating how air pollution relates to metabolic syndrome and its components could elucidate pathways between household air pollution exposure and cardiometabolic diseases. Evidence for an association between air pollution and metabolic syndrome or its components is limited and comes mostly from studies in higher income countries examining the effects of ambient air pollution (12). Positive associations between elevated levels of ambient air pollution (PM₁, PM_{2.5}, or PM₁₀, ozone) have been reported with total cholesterol (13–16), triglycerides (14, 16–19), and low-density lipoprotein (LDL) (14, 16, 19), but not all studies observed effects (20). Results on HDL have been inconsistent (14, 16, 20), and studies on household air pollution and metabolic syndrome are lacking.

We performed a cross-sectional study to examine the associations of 24-hour kitchen and personal air pollution measurements with blood lipid concentrations, waist circumference, and metabolic syndrome among women in rural Honduras using traditional and cleaner-burning *Justa* stove models (both use wood fuel). This is the first study to evaluate the association of quantitative concentrations of household air pollution with these cardiometabolic health outcomes, all of which are important risk factors for cardiovascular disease.

Materials and methods

Participant recruitment

We measured exposure to household air pollution and health endpoints in 150 women between February and April 2015 from 11 rural communities near the town of La Esperanza in western Honduras. A description of the study location and general population are also described elsewhere (21). We worked in conjunction with our Honduran partners and local

municipality and community leaders to design the recruitment strategy. We held local community meetings in villages surrounding La Esperanza and presented detailed information regarding the study to the community members. Researchers screened 500 households that attended the community meetings and expressed interest in the research project. We utilized convenience sampling to locate households who owned cleaner-burning *Justa* stoves and identified traditional stoveusing households living within one kilometer of a *Justa* stove home. In total, we visited 170 households from 9 February–30 April 2015. We recruited the female primary cook in each household to participate in the study. Enrollment criteria required that the primary cook be age 25–56 years, a nonsmoker, not pregnant, and must have owned a traditional cookstove or *Justa* cookstove for at least 4 months prior to the interview. Upon visitation, eighteen of the 170 households were excluded as they did not have a female primary cook who meet the eligibility criteria. Two women chose not to participate in the study; therefore, we enrolled a total of 150 participants. Women received an incentive consisting of food items worth US\$5. The study protocol was approved by the Colorado State University Institutional Review Board. Local leaders gave permission to work within their communities prior to data collection, and informed consent was obtained from all women.

Household Air Pollution

All kitchen and personal air pollution measurements were collected for 24 hours. For kitchen measurements, monitors were hung at a distance of 76–127 cm from the stove edge. For personal measurements, the women wore PM_{2.5} monitors with the inlet attached to a bag strap to measure the air near the breathing zone. The woman was instructed to place the bag next to her when she was bathing, sleeping or breastfeeding.

Particles less than 2.5 micrometers in aerodynamic diameter (PM_{2.5}) were collected on 37 mm Teflon-coated glass fiber filters (Fiberfilm™ T60A20, Pall Corporation, Port Washington NY, USA) using AirChek XR5000 pumps (SKC Inc., Eighty Four, PA, USA) and Triplex Cyclones (BGI by Mesa Labs, Butler NJ, USA) operating at 1.5 L/min. Filters were analyzed gravimetrically to determine PM_{2.5} mass.

Prior to and after each use, the pumps were calibrated to 1.5 L/min using a DryCal Dc-Lite primary flow meter (Bios International Corporation, Butler NJ, USA). Pump performance was considered adequate if the calibration flow rate did not deviate by more than 10% between pre/post measurements. The original sample size of 150 was reduced for the final air pollution models due to missing calibration data (i.e., malfunctioning flow meter at the beginning of the study); therefore, exposure could not be assessed in 41 houses (n=12 traditional stoves and n=29 *Justa* stoves). There were also two filters with missing post-sampling data, so final sample sizes for the pollution models are as follows: kitchen PM_{2.5} n=106; personal PM_{2.5} n=105; kitchen black carbon n=107; and personal black carbon n=106. One field blank and one measurement blank were collected every two weeks. Twenty-four hour time-weighted average PM_{2.5} concentrations were calculated by dividing the blank-corrected PM_{2.5} mass (post-sampling filter weight minus pre-sampling filter weight) by the volume of air sampled (average volumetric flow rate times sample duration). The concentration limit of detection (LOD; 25 µg/m³) was calculated by adding the mean

change in mass of the measurement blanks (29 µg; n=7) to three times the standard deviation (SD) of the measurement blanks (8 µg) and then converted to a concentration (22). Concentrations below the LOD (kitchen: n=7; personal: n=4) were substituted by LOD/(2).

PM_{2.5} black carbon concentrations were determined by the optical transmission of light through the air sampling filters using a transmissometer (model OT-21, Magee Scientific, USA). Transmission data were converted to mass concentrations based on published mass-absorption values for combustion aerosol and corrected for a filter loading artifact that leads to an underestimation of the black carbon concentration at high sample loading. The concentration LOD for black carbon was estimated to be 0.86 µg/m³ corresponding to three times the SD of 54 blank samples (additional blank filters were used from field sampling campaigns conducted within the same year to estimate the reference values for the transmissometer since presampling transmission data were not collected on sample filters). Values below the LOD (kitchen: n=3; personal: n=10) were substituted by LOD/(2). More detailed information on black carbon methods is available as supplementary material and has been previously described (23).

A dichotomous stove type variable (traditional vs. *Justa* stoves) was evaluated as an additional indicator of exposure. Both the traditional and *Justa* stove types were wood-burning stoves. Traditional stoves were self-built with a large open combustion chamber, either elevated or on the ground (Figure 1). *Justa* stoves had been previously installed by non-governmental organizations at least four months prior to data collection and included a rocket type (L-shaped) insulated combustion chamber, chimney, metal griddle, and a soot collector that allows for removal of excess ash from the chimney (21). All primary stoves were in an enclosed kitchen separated from the rest of the house or within the main living area.

Health Endpoints

Women were not required to fast prior to the health measurements. Lipids were measured with finger-stick blood samples using a CardioChek® PA Analyzer with Lipid Panel Test Strips (PTS Diagnostics, Indianapolis, USA) between 7 a.m. and 12 noon after the 24-hour exposure assessment period. The measuring ranges for total cholesterol, HDL and triglycerides on the test system are 100–400 mg/dL, 15–100 mg/dL, and 50–500 mg/dL, respectively. Total cholesterol values below the LOD (n=14) were replaced by LOD/(2). There were no HDL or triglyceride values below the LOD. LDL is estimated by the device by subtracting HDL and 1/5 triglycerides from total cholesterol. LDL is only calculated if total cholesterol is above the LOD and triglycerides are < 400 mg/dL.

Blood pressure was measured with a SphygmoCor XCEL device (AtCor Medical Pty Ltd, Sydney, Australia). The woman was asked to rest for at least 10 minutes in a seated position before the measurement. Three consecutive measurements were taken in a seated position with a one minute break in between measurements. For analysis, the average of the two last measurements was used. Glycated hemoglobin (HbA1c) was measured with the A1CNow +® system (PTS Diagnostics, Indianapolis, USA) using a finger stick sample of 5µl of blood, collected with BD Genie™ lancets (BD, Franklin Lakes, USA). Waist circumference was measured with a tape measure around the smallest circumference of the natural waist of

the woman or if this was not obvious at the upper border of the belly button. Results from this study population on the effects of household air pollution on HbA1c and blood pressure as individual health endpoints have been published (21, 23).

Metabolic syndrome was defined according to the International Diabetes Federation (9), modified for non-fasting triglycerides as recommended by Driver and colleagues (24). These guidelines are based on a consensus worldwide definition as the presence of abdominal obesity, plus any two additional risk factors for cardiovascular disease and type 2 diabetes mellitus (9). A woman was considered to have metabolic syndrome if the waist circumference was greater than or equal to 80 cm plus any two of the following: triglycerides >200 mg/dL, HDL <50 mg/dL, systolic blood pressure $\geq$ 130 mmHg, diastolic blood pressure $\geq$ 85 mmHg, or HbA1c $\geq$ 5.6%. In sensitivity analyses, we also evaluated metabolic syndrome as defined by the Joint Scientific Statement, which uses a slightly different definition as any three out of five cardiometabolic risk factors, as listed above (25). We calculated prevalence of metabolic syndrome for both definitions. Final models with associations with household air pollution explored both definitions; however, primary results reflect the International Diabetes Federation's definition due to greater comparability with existing literature.

Potential confounders and effect modifiers

Potential confounders and effect modifiers were assessed by additional anthropometric measurements and questionnaires on health, socioeconomic status, and cooking practices. Hip circumference was measured with a tape measure around the broadest part of the woman's hips. The waist-to-hip ratio was calculated by dividing the waist circumference by the hip circumference. Weight was measured with a scale. Height was taken with the woman standing barefoot with her heels against a wall by placing a level on her head and measuring from the ground up to the level. Body mass index (BMI) was calculated by dividing the weight in kilograms by the height in meters squared (kg/m^2).

We evaluated socioeconomic status through various indicators. The number of beds per person in the household was calculated by dividing the total number of beds by the number of people living in the house. Years of school completed was dichotomized as less than six years or greater or equal to six years, which represented a typical cutoff of education among our study population. The presence of electricity in the house was assessed (yes/no). A material-assets variable was defined by the sum total of the following items the household possessed: cars, bikes, motorbikes, televisions, radios, refrigerators, sewing machines, and electricity.

A dietary diversity score for the last 24 hours of food consumption was created by adding up the number of consumed food groups (26). A list of 19 individual food items was condensed into 10 food groups (cereals, pulses and nuts, roots, other vegetables, fruits, sugar/sweets, eggs, dairy, meat, and beverages). The dietary diversity score is sometimes used in place of socioeconomic status as it often varies according to socioeconomic status (27). Daily consumption of salt, sugar, and fat (vegetable shortening) were ascertained by showing the women commonly purchased quantities of the specific items and asking how often the

amounts were purchased. Daily consumption for each item was estimated by dividing the total daily household consumption (grams per item) by the number of household members.

Physical activity was assessed by asking women how many hours per day and how many days per week they perform the following lifestyle activities, which are culturally typical: cut wood, grind corn, wash clothes, milk the cow, work in the field, carry a heavy weight and walk normally outside the house. For each activity, the number of hours per week was estimated and multiplied by the corresponding metabolic equivalent (MET) from the updated 2011 compendium of physical activities (28). METs were not provided specifically for grinding corn; therefore, we categorized it as a general kitchen activity with moderate effort. We converted weekly hours of activity to MET-hours by multiplying the activity by the MET score. The MET values from all activities were summed to estimate weekly METs. Greater detail on this approach has been previously published for this study population (21).

A few additional variables were collected that could influence the exposure and/or health endpoints. Mean kitchen temperature was assessed with the EL-USB-2 Data Logger (Lascar Electronics, Erie, PA). Elevation of the house was measured using the cell phone app maps.me (My.com B.V. version 6.5.3). Lastly, women reported current use of any medications, caffeine and breakfast intake on the same day prior to the health measurements (yes/no), and the age when they began cooking to estimate total years of cooking (current age – age started cooking).

Data Analysis

Data were analyzed using Stata version 13.1 (StataCorp LP, College Station, TX) and R version 3.4.3 (R foundation for Statistical Computing, Vienna, Austria). Descriptive statistics were calculated for exposure and health outcomes, as well as for all potential confounders described above. Spearman correlation coefficients were calculated between exposure metrics to summarize the strength and directional associations between air pollutants.

To investigate the cross-sectional associations between exposures (stove type and 24-hour air pollution concentrations) and the continuous health endpoints of blood lipids and waist circumference, we used multivariable linear regression models. Continuous pollution values were natural log-transformed to satisfy the model assumptions. Univariable associations with the health endpoints were assessed for all potential confounders as identified in the previous section. Final co-variables were selected based on their strength of association in univariable regression analysis with each outcome of interest, further since several variables described similar characteristics, we chose the variable with the strongest effect from the following categories to include in final models: socioeconomic status (number of beds, years of school, electricity in the house, or material assets), diet (dietary diversity score, daily consumption of salt, sugar, or vegetable shortening), and, for lipids, anthropometry (waist circumference, waisthip ratio, or BMI). Final models for blood lipids (i.e., total cholesterol, HDL, calculated LDL, and triglycerides) were adjusted for age (continuous), material assets (continuous), daily consumption of vegetable shortening (continuous), physical activity (continuous), and BMI (continuous). Final models for waist circumference were adjusted for age (continuous), material assets (continuous), daily consumption of vegetable shortening

(continuous), and physical activity (continuous). Results of linear regression models are presented as adjusted means of the health endpoint per one unit increase in natural log-transformed 24-hour average PM_{2.5} and black carbon concentrations.

To investigate the cross-sectional association between exposures and dichotomous metabolic syndrome (normal vs. syndrome present), adjusted Poisson regression models were used to estimate prevalence ratios, as recommended by Barros and colleagues (29). Transformation of the pollution variables was not necessary for the Poisson regression models. Final models were determined by evaluating if there was any gain in precision by eliminating a potential confounder, while also considering the magnitude of its removal on the effect estimate of the exposure variable using a cutoff of 10%. That is, a potential confounder was eliminated if removing it from the model did not change the magnitude of the effect estimate of the exposure variable by more than 10% and the precision of the model was improved. Final Poisson regression models evaluating metabolic syndrome were adjusted for age (continuous), dietary diversity score (continuous) and years of school (binary: <6 years, ≥6 years). Results of Poisson regression models are presented per interquartile range (IQR) increase in 24-hour average pollution concentration or in relation to a reference category for categorical stove type exposure. Primary analyses evaluated metabolic syndrome as defined by the International Diabetes Federation (9); metabolic syndrome as defined by the Joint Scientific Statement (25) was explored in sensitivity analyses.

To evaluate possible heterogeneity of effects across subgroups (i.e., effect measure modification), dichotomous variables were created for age (<40 years vs. ≥40 years), blood pressure (normal: systolic <120 and diastolic <80 mmHg vs. borderline high/high blood pressure: systolic ≥120 and/or diastolic ≥80 mmHg), and diabetes status (normal: HbA1c <5.7% vs. prediabetes/diabetes: HbA1c ≥5.7%). For the continuous outcomes of blood lipid concentrations and waist circumference, additive interaction was assessed in separate linear regression models by including a product term created by multiplying natural log-transformed pollution or stove type by the dichotomous variable of age, blood pressure, or diabetes. In Poisson regression models evaluating the dichotomous outcome of metabolic syndrome, multiplicative interaction was assessed using terms created by multiplying the pollution or stove type variable with age. Information regarding interaction on the additive scale could be gleaned from the multiplicative interaction results (30).

To assess the robustness of the results, we conducted sensitivity analyses by removing the following women from the sample and re-running the final models: those with occasional exposure to secondhand smoke (n=5), BMI less than 18.5 (n=5), currently using medications related to the health outcomes (n=3), and PM_{2.5} pump flow that had decreased by more than 10% during the 24-hour measurement (kitchen: n=3, personal: n=5). We also re-ran final models with a fixed-term for community to account for potential similarities of respondents within the same communities. Metabolic syndrome was further assessed in sensitivity analyses using the definition by the Joint Scientific Statement (25). Lipid models were further evaluated by excluding BMI as a co-variable to assess for potential over-adjustment.

Results

In the present analysis, 150 Honduran women with an average age of 37.3 years (SD, 9.0; range, 25–56) were included. Half of the women owned a traditional stove (n=76), the other half (n=74) owned a cleaner burning *Justa* stove for a mean of 24 months (range, 4–120 months). Women had been cooking for an average of 26 years (SD, 9.9), and age was strongly correlated with years of cooking (Spearman correlation coefficient 0.96). Socioeconomic status, diet, physical activity and anthropometric measurements were similar for traditional stove owners and *Justa* stove owners (Table 1).

As shown in Table 1, average blood lipid levels among our total study population were as follows: total cholesterol 133 mg/dL (SD, 30; n=147), HDL 39 mg/dL (SD, 10; n=147) calculated LDL 59.2 mg/dL (SD, 19; n=121), and triglycerides 201 mg/dL (SD, 104; n=147). LDL could not be calculated for 26 women because their triglycerides levels were higher than 400 mg/dL or their total cholesterol was below the LOD. There were no substantial differences in blood lipid measures between traditional or *Justa* stove owners. Mean systolic blood pressure was slightly lower among *Justa* stove owners (116.8 mmHg, SD, 13.0) compared to traditional stove owners (120.6 mmHg, SD, 12.2), and diastolic blood pressure was nearly identical by stove type (Table 1). Average anthropometric measurements included waist circumference 83.8 cm (SD, 9.1), waist-to-hip ratio 0.88 (SD, 0.06), and BMI 25.8 (SD, 4.2), which were similar between traditional and *Justa* stove owners (Table 1). A total of 59 women (40%) met the criteria for having metabolic syndrome per International Diabetes Federation guidelines, 31 traditional stove owners and 28 *Justa* owners (Table 1). Per Joint Scientific Statement guidelines, 64 women (45%) had metabolic syndrome, 33 traditional stove owners and 31 *Justa* stove owners (Table 1). Three women (2%; two traditional stove owners and one *Justa* stove owner) were currently taking anti-hypertensive medications, and none reported current use of lipids or diabetes medications (Table 1).

Mean exposure to 24-hour kitchen fine particulate matter (PM_{2.5}) was 354 µg/m³ (SD, 373; median, 181; 25th to 75th percentile, 90 to 511; n=62) for traditional stove owners and 137 µg/m³ (SD, 194; median, 71; 25th to 75th percentile, 38 to 265; n=44) for *Justa* stove owners (Table 2). Personal 24-hour PM_{2.5} and black carbon measurements were similarly higher in traditional stove users compared to *Justa* stove users (Table 2). Although *Justa* stoves had lower average concentrations of PM_{2.5} and black carbon compared to traditional stoves, there was substantial overlap between the two groups (Figure 2). Air pollution metrics varied in how strongly they were correlated with one another. Specifically, Spearman correlation coefficients ranged from 0.67 (kitchen PM_{2.5} versus personal black carbon) to 0.89 (kitchen PM_{2.5} versus kitchen black carbon). Although the original sample size of 150 was reduced for the final air pollution models due to missing calibration data or post-sampling data, the population in the pollution models did not differ from the full study population. For example, in the kitchen PM_{2.5} analyses (n=106), mean (SD) age (37.0 [9.0] years) and material assets (2.1 [1.4] assets) did not differ from the full population (37.3 [9.0] years and 1.9 [1.3] assets).

Multivariable linear regression analyses of exposure metrics and continuous measures of blood lipids and waist circumference were consistent with a null association after adjusting for age (continuous), material assets (continuous), daily consumption of vegetable shortening (continuous), BMI (continuous) and physical activity (continuous). That is, all 95% CIs included zero for the adjusted mean differences in total cholesterol, HDL, calculated LDL, triglycerides, and waist circumference for the exposures of stove type, 24-hour kitchen and personal PM_{2.5}, and 24-hour kitchen and personal black carbon (Table 3).

Adjusted prevalence ratios for metabolic syndrome (according to the International Diabetes Federation guidelines) per interquartile range increase in 24-hour average pollution concentration were elevated for kitchen PM_{2.5} (PR per IQR increase= 1.16; 95% CI, 1.01, 1.34; n=103; IQR=312.0 µg/m³) and kitchen black carbon (PR per IQR increase= 1.07; 95% CI, 1.03, 1.12; n=104; IQR= 73.6 µg/m³) (Table 4). Adjusted prevalence ratios for personal pollution concentrations and stove type with metabolic syndrome were consistent with a null association (Table 4). We observed suggestive evidence of synergistic multiplicative interaction (and therefore synergistic additive interaction) between age and personal PM_{2.5}, kitchen black carbon, and personal black carbon on the outcome of metabolic syndrome (Table 5). For example, the association of personal PM_{2.5} and metabolic syndrome was suggestively stronger among women ≥40 years of age compared to women <40 years (p-value for multiplicative interaction in Poisson regression models = 0.12; Table 5).

We observed some evidence of additive interaction between exposure metrics and borderline high/high blood pressure on the effect of blood lipids (Supplementary materials). For example, there was evidence for a stronger association between 24-hour kitchen PM_{2.5} and kitchen black carbon with total cholesterol among women with borderline high/high blood pressure compared to those with normal blood pressure levels. The adjusted mean difference in total cholesterol was 6.95 mg/dL (95% CI, -0.19, 14.09) higher per one unit increase in natural log-transformed kitchen PM_{2.5} among 36 women with borderline high/high blood pressure status compared to no effect on total cholesterol among 69 women with normal status (p-value for additive interaction = 0.007) (Supplementary Table 12). Women with higher blood pressure and those defined as prediabetic or diabetic also had suggestive evidence of stronger associations between increased kitchen PM_{2.5} and higher levels of LDL (Supplementary Tables 14 and 19) compared to women without these health conditions. However, patterns from the effect modification models were inconsistent across other exposure metrics and health endpoints.

In the sensitivity analyses, results did not change after removing women occasionally exposed to secondhand smoke, those with a BMI less than 18.5, those currently taking anti-hypertensive medications, and those with a PM_{2.5} pump flow decrease by more than 10%. Including a term for community and excluding BMI from the lipid models did not influence the results (results not presented). Results using the Joint Scientific Statement definition of metabolic syndrome were nearly identical to the primary results presented above (results not presented).

Discussion

In our cross-sectional study among 150 women in Honduras using traditional and cleanerburning *Justa* stoves, we observed lower mean concentrations of all air pollutants among *Justa* users than traditional stove users. However, average 24-hour PM_{2.5} values for *Justa* stove owners were still above the World Health Organization's air quality guidelines for 24-hour concentrations (25 µg/m³) (31). Associations between household air pollution and blood lipids or waist circumference were consistent with a null association. However, we observed suggestive evidence for a higher prevalence of metabolic syndrome in women with increased 24-hour kitchen PM_{2.5} and black carbon concentrations. There was limited evidence that age and borderline high/high blood pressure may modify the effect of air pollution concentrations on metabolic syndrome and total cholesterol, respectively.

Levels of total cholesterol, HDL, and calculated LDL were low in our population. While low levels are desirable for total cholesterol and LDL, higher levels of HDL are beneficial (32). Triglycerides were high in our population, possibly due to non-fasting measurements (33). However, several large observational studies and reviews concluded that fasting is not required for lipid measurements (33–36). Non-fasting triglycerides levels have also been reported as being a better indicator of cardiovascular disease risk than fasting levels (36–39).

Our study was the first to evaluate the effect of household air pollution on metabolic syndrome. The majority of previous household air pollution research assessed separate components of metabolic syndrome, such as blood pressure (40–43), cardiovascular mortality from self-reported solid fuel use (4), or the associations between ambient PM₁₀ in high-income countries and metabolic syndrome (44). Given that we did not observe consistent associations between household air pollution and blood lipids or waist circumference, it is possible that household air pollution has stronger effects on some components of metabolic syndrome, such as high HbA1c and blood pressure (21, 23), rather than several components measured here or the syndrome as a whole (45). Previous work published from this Honduran sample found suggestive evidence linking household air pollution with the prevalence of prediabetes/diabetes based on HbA1c and elevated systolic and diastolic blood pressure (21, 23).

Evaluating metabolic syndrome is of particular interest in our setting as a recent study reported 34% prevalence of metabolic syndrome among women in urban Honduras in 2003–2006, similar to our study participants (40%) (46). This rate is high, considering that the same publication reported the prevalence of metabolic syndrome in Honduran males to be 12%, and the International Diabetes Federation estimates a prevalence of 20–25% for the world's population (9, 46). Our analyses provided suggestive evidence that the effects of household air pollutants on metabolic syndrome may be stronger among women ≥40 years of age compared to women <40 years. This observation supports the findings that age may act as an effect modifier for indicators of cardiovascular disease based on three studies that reported stronger effects of air pollution exposure on blood pressure in older age groups (21, 40, 41).

Blood lipids have not been evaluated in previous studies on household air pollution, and findings reported in the ambient air pollution literature have been inconsistent. For example, a Danish cross-sectional study reported a 0.78 (0.22, 1.34) mg/dL increase in total cholesterol per IQR increase in ambient PM_{2.5}, and a study from Taiwan reported positive associations between total cholesterol and PM₁₀ and PM_{2.5} (13, 15). The same Taiwanese study did not report any associations between air pollution and triglycerides or HDL, whereas a retrospective cohort study from Israel reported higher LDL and triglycerides as well as lower HDL in relation to higher concentrations of PM₁₀ and PM_{2.5} (15, 19). Several previous studies relied on measuring oxidized lipids as indicators of cardiovascular disease risk instead of nonoxidized lipids, as we did here. Lipid peroxidation can be induced by free radicals that are part of PM_{2.5} leading to reduced levels of regular blood lipids. Measuring oxidized and nonoxidized lipids in future studies may better inform the cardiovascular disease risk related to household air pollution.

In addition to a limited sample size, the following factors should be considered when interpreting our results. Selection bias may have occurred when recruiting the convenience sample; however, for our finding of a higher prevalence of metabolic syndrome in women with higher 24-hour kitchen PM_{2.5} and black carbon, it is unlikely that meeting attendance and eventual participation was influenced by these two exposure metrics and the health outcome, and not the other measures evaluated. Given the cross-sectional design, we were not able to establish temporality between exposure and the health endpoints. The definition of metabolic syndrome calls for data on fasting glucose, which was substituted with non-fasting HbA1c. However, this substitution should not be seen as an important limitation since a previous study among an Italian sample reported 91% agreement with this same substitution for the classification of metabolic syndrome (47). Since most of our observed associations were based on kitchen exposure measures, it is possible that measuring a single 24-hour personal exposure may not capture long-term exposure, as personal measurements can vary based on day-to-day activities. Although we adjusted for important confounders, residual confounding is still a possibility due to unmeasured or inaccurately measured factors. We may have also missed potential effect modifiers, such as genetic predisposition, pre-existing medical conditions that affect blood glucose levels, or hormonal imbalances. PM_{2.5} and black carbon may be surrogates for other health-damaging pollutants that we did not directly measure. Due to the high correlation between PM_{2.5} and black carbon, we did not assess two-pollutant models. Generalizability of our stove type results may be limited as other populations use different stove types and/or fuels. Lastly, repeating measurements over time for both exposure and health endpoints may increase reliability when longer-term associations between chronically experienced exposures and health impacts are of interest.

This study also had several strengths. All exposure and health metrics were directly measured without relying on proxies or self-report. The use of HbA1c rather than fasting glucose is a more stable indicator of long-term blood glucose levels over the past two to three months and is less affected by acute illness or infection (48). Furthermore, as the first study to present data on household air pollution and metabolic syndrome, this research is a critical step towards understanding the link with cardiometabolic disease risk among people with chronically high exposures.

Conclusions

The prevalence of metabolic syndrome in our study sample was twice as high as average estimates among adults worldwide, and suggestive effects were observed between metabolic syndrome and 24-hour kitchen PM_{2.5} and black carbon. There was suggestive evidence that cardiometabolic-related effects of household air pollution may be stronger among older women. There was no evidence of associations between other exposure metrics and metabolic syndrome or between exposures and blood lipids or waist circumference. However, borderline high/high blood pressure may modify the associations between household air pollution and total cholesterol. Longitudinal research with repeated measures of exposures and indicators of cardiometabolic disease risk are needed to continue clarifying the link between household air pollution and cardiovascular disease.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

BMI	body mass index
CI	confidence interval
HbA1c	glycated hemoglobin
HDL	high-density lipoprotein
IQR	interquartile range
LOD	limit of detection
LDL	low-density lipoprotein
MET	metabolic equivalent
PM_{2.5}	fine particulate matter
PR	prevalence ratio
SD	standard deviation

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Highlights:

- Household air pollution may adversely impact indicators of cardiometabolic disease risk
- Suggestive evidence links 24-hour household air pollution and metabolic syndrome
- Associations of cardiometabolic-related effects from household air pollution may potentially be stronger among susceptible sub-groups
- Longitudinal research with repeated exposure and health measures is needed

Figure 1:
Examples of typical traditional cookstoves (left and middle) and a *Justa* cookstove (right) among the study population.

Figure 2.

Distributions of 24-hour mean kitchen and personal PM_{2.5} and black carbon concentrations ($\mu\text{g}/\text{m}^3$) by stove type: 0=traditional stove, 1=Justa stove. The length of the box is the interquartile range, the top edge of the box is the 75th percentile and the bottom edge of the box is the 25th percentile. The diamond symbol inside the box is the group mean, and the horizontal line inside the box is the group median. The outlying circles represent outliers

Table 1:

Characteristics of Honduran study participants and households, for all women and by stove type (traditional and *Justa*)

	All participants		Traditional stove owners		<i>Justa</i> stove owners	
	N	Mean (SD); range or N (%)	N	Mean (SD); range or N (%)	N	Mean (SD); range or N (%)
Age (years)	150	37.3 (9.0); 25–56	76	38.5 (9.9); 25–56	74	36.1 (7.9); 25–56
Beds per person in household	149	0.5 (0.2); 0.2–1	76	0.5 (0.2); 0.2–1	73	0.5 (0.2); 0.3–1
>=6 years of school	148	78 (52.7%)	75	35 (46.7%)	73	43 (58.9%)
Electricity in the house	149	26 (17.5%)	76	12 (15.8%)	73	14 (19.2%)
Material assets [*]	149	1.9 (1.3); 0–9	76	1.9 (1.2); 0–5	73	1.9 (1.3); 0–9
Daily consumption of salt (grams)	150	8.5 (6.1); 1.1–40.0	76	8.9 (6.2); 1.2–33.3	74	8.2 (6.0); 1.1–40
Daily consumption of sugar (grams)	150	42.3 (25.0); 6.0–150.0	76	43.5 (26.1); 6.0–150.0	74	41.1 (24.0); 8.2–112.5
Daily consumption of vegetable shortening (grams)	150	13.0 (10.0); 2.2–56.8	76	14.7 (10.8); 2.9–56.8	74	11.2 (8.9); 2.2–45.4
Dietary diversity score [†]	150	6.0 (1.7); 2–10	76	6.1 (1.7); 3–10	74	6.0 (1.6); 2–10
Physical activity [‡]	150	212 (107); 31–543	76	216 (115); 31–543	74	207 (97); 46–445
24-hour average kitchen temperature (°Celsius)	146	21.3 (3.0); 12.5–27.2	73	21.8 (3.0); 12.9–27.2	73	20.9 (2.9); 12.5–26.9
Household elevation (meters)	149	1916 (107); 1729–2171	76	1894 (98); 1737–2152	73	1939 (112); 1729–2171
Had caffeine on measurement day	132	121 (91.7%)	67	60 (89.6%)	65	61 (93.9%)
Had breakfast on measurement day	132	117 (88.6%)	67	63 (94.0%)	65	54 (83.1%)
Years of cooking	150	25.8 (9.9); 7–50	76	26.9 (10.8); 7–49	74	24.7 (8.7); 9–50
Length of <i>Justa</i> stove ownership (months)					74	23.9 (17.4); 4–120
Waist circumference (cm)	150	83.3 (9.1); 64.8–111.8	76	83.8 (9.9); 64.8–111.8	74	82.9 (8.2); 67.3–104.1
Waist-to-hip ratio	150	0.88 (0.06); 0.74–1.09	76	0.89 (0.06); 0.74–1.09	74	0.87 (0.05); 0.77–0.99
BMI (kg/m ²)	150	25.8 (4.2); 17.1–37.5	76	25.6 (4.6); 17.1–37.5	74	26.0 (3.8); 18.2–33.6
Systolic blood pressure (mmHg)	150	118.7 (12.7); 91–160	76	120.6 (12.2); 97–158	74	116.8 (13.0); 91–160
Diastolic blood pressure (mmHg)	150	73.2 (8.7); 55–105	76	73.9 (8.5); 58–96	74	72.4 (8.9); 55–105
HbA1c(%)	142	5.5 (0.7); 4.1–13	72	5.5 (0.4); 4.7–6.5	70	5.6 (1.0); 4.1–13
Total Cholesterol (non-fasting); (mg/dL)	147	133 (30); 71–224	73	132 (30); 71–196	74	134 (30); 71–224
HDL (non-fasting); (mg/dL)	147	39 (10); 24–71	73	39 (10); 24–71	74	39 (9); 25–64
Calculated LDL (non-fasting); (mg/dL)	124	59.8 (20.4); 17–146	63	59.9 (21.4); 25–146	61	59.7 (19.6); 17–112
Triglycerides (nonfasting); (mg/dL)	147	201 (104); 55–500	73	201 (93); 55–500	74	201 (115); 58–500
Metabolic						
Syndrome International	148	59 (40%)	75	31 (41%)	73	28 (38%)

	All participants		Traditional stove owners		Justa stove owners	
	N	Mean (SD); range or N (%)	N	Mean (SD); range or N (%)	N	Mean (SD); range or N (%)
Diabetes Federation Definition Joint Scientific Statement Definition	143	64 (45%)	72	33 (46%)	71	31 (44%)
Current medication use for hypertension	150	3 (2%)	76	2 (3%)	74	1 (1%)

BMI: body mass index; HDL: high density lipoprotein; LDL: low density lipoprotein; SD: standard deviation

* Material assets were defined by the number of the following items the household possessed: cars, bikes, motorbikes, televisions, radios, refrigerators, sewing machines, electricity.

[†] Scale with values from 1–10 indicating number of food groups included in diet

[‡] Estimated weekly metabolic equivalents including the following self-reported activities: cut wood, grind corn (categorized as general kitchen activity with moderate effort), wash clothes, milk the cow, work in the field, carry a heavy weight and walk normally outside the house. For each activity the number of hours per week was calculated and multiplied with the corresponding metabolic equivalent (MET) from the Compendium of Physical Activities.

// Metabolic Syndrome as defined by International Diabetes Foundation, modified for non-fasting triglycerides: waist circumference ≥80cm plus any two of the following: (non-fasting) triglycerides >200mg/dL, HDL <50mg/dL, systolic blood pressure ≥130mmHg, diastolic blood pressure ≥85mmHg, or HbA1c >5.6%. Metabolic Syndrome as defined by the Joint Scientific Statement: three abnormal findings out of the following five risk factors- waist circumference ≥80cm, triglycerides ≥150mg/dL, HDL <50mg/dL, systolic blood pressure ≥130mmHg, diastolic blood pressure ≥85mmHg, or HbA1c >5.6%.

Table 2:

Air pollution data for the total population and by stove type among Honduran women using traditional and *Justa* stoves

	All participants		Traditional stove owners		<i>Justa</i> stove owners	
	N	Mean (SD); range Median; 25 th –75 th percentile	N	Mean (SD); range Median; 25 th –75 th percentile	N	Mean (SD); range Median; 25 th –75 th percentile
24-hour average kitchen PM _{2.5} (µg/m ³)	106	264 (328); 18–1654 132; 62–374	62	354 (373); 18–1654 181; 90– 511	44	137 (194); 18–1134 71; 38–265
24-hour average personal PM _{2.5} (µg/m ³)	105	101 (70); 18–346 79; 51– 135	62	125 (76); 18–346 114; 65–241	43	66 (38); 18–174 53; 39– 121
24-hour average kitchen black carbon (µg/m ³)	107	74 (148); 1–1172 21; 8– 82	63	104 (176); 1–1172 114; 44– 114	44	31 (80); 1–469 11; 4–19
24-hour average personal black carbon (µg/m ³)	106	17 (22); 1–123 7; 4–18	62	24 (26); 1–123 14; 6–32	44	7 (8); 1–47 4; 1–9

SD: standard deviation; PM_{2.5}: fine particulate matter

Adjusted mean difference in lipids and waist circumference in relation to pollution and stove type, Honduras, traditional and *Justa* stove users

Table 3:

	Total Cholesterol (mg/dL)		HDL (mg/dL)		Calculated LDL (mg/dL)		Triglycerides (mg/dL)		Waist circumference (cm)	
	N	Adjusted mean difference (95% CI)	N	Adjusted mean difference (95% CI)	N	Adjusted mean difference (95% CI)	N	Adjusted mean difference (95% CI)	N	Adjusted mean difference (95% CI)
Kitchen PM _{2.5} (µg/m ³)*	105	0.4 (-4.5-5.3)	105	0.4 (-1.2-2.1)	89	-0.2 (-3.8-3.3)	105	1.1 (-16.2-18.4)	106	1.25 (-0.23-2.73)
Personal PM _{2.5} (µg/m ³)*	104	-7.1 (-15.4-1.2)	104	1.0 (-1.8-3.8)	88	-5.9 (-11.8-0.0)	104	-2.6 (-32.1-26.9)	105	0.50 (-1.98-2.99)
Kitchen black carbon (µg/m ³)*	106	-0.5 (-4.0-3.0)	106	0.3 (-0.9-1.4)	90	0.3 (-2.3-2.9)	106	-4.8 (-17.1-7.6)	107	0.74 (-0.33-1.81)
Personal black carbon (µg/m ³)*	105	-1.0 (-5.2-3.3)	105	1.5 (0.1-2.9)	89	-1.2 (-4.3-1.9)	105	-8.2 (-23.0-6.7)	106	0.33 (-0.96-1.62)
Stove type [†]										
<i>Justa</i>	73	ref	73	ref	60	ref	73	ref	73	ref
Traditional	73	-3.5 (-13.1-6.0)	73	-0.8 (-3.9-2.4)	63	-1.6 (-9.0-5.8)	73	-1.9 (-35.3-31.5)	76	0.58 (-2.31-3.47)

Adjusted for age (continuous), material assets (continuous), BMI (continuous), daily consumption of vegetable shortening (continuous), physical activity (continuous)

* Adjusted mean difference in lipid or waist circumference per log increase in pollution

[†] Adjusted mean difference in lipid or waist circumference vs reference

BMI: body mass index; CI: confidence interval; HDL: high density lipoprotein; LDL: low density lipoprotein; PM_{2.5}: fine particulate matter

Table 4:

Adjusted prevalence ratios for the association of prevalent metabolic syndrome with air pollution and stove type, Honduras, traditional and *Justa* stove users

	Metabolic Syndrome (metabolic syndrome vs. normal)			
			Poisson regression	
	N	IQR	PR per IQR increase or vs reference	95% CI
Kitchen PM ₂₅ (µg/m ³)	103	312	1.16	1.01–1.34
Personal PM ₂₅ (µg/m ³)	103	84	1.05	0.79–1.39
Kitchen black carbon (µg/m ³)	104	73	1.07	1.03–1.12
Personal black carbon (µg/m ³)	103	14	1.04	0.91–1.18
Stove type	146			
<i>Justa</i>			ref	
Traditional			1.08	0.72–1.61

Adjusted for age (continuous), dietary diversity score (continuous), years of school (<6 years, ≥6 years)

CI: confidence interval; HbA1c: glycated hemoglobin; IQR: interquartile range; PM_{2.5}: fine particulate matter; PR:prevalence ratio

Table 5:

Adjusted prevalence ratios for the association of prevalent metabolic syndrome with multiplicative interaction terms between age and pollution measures and stove type categories

				Poisson regression		
		IQR	N	PR per IQR increase or vs. reference	95% CI	p-value for interaction
Kitchen PM ₂₅ (µg/m ³)		312	103			0.81
	age<40		67	1.19	0.89–1.58	
	Age 40		36	1.14	0.97–1.34	
Personal PM ₂₅ (µg/m ³)		84	103			0.12
	age<40		68	0.82	0.49–1.37	
	age 40		35	1.33	0.96–1.84	
Kitchen black carbon (µg/m ³)		72	104			0.31
	age<40		68	1.07	1.01–1.14	
	age 40		36	1.12	1.04–1.21	
Personal black carbon (µg/m ³)		13	103			0.29
	age<40		68	0.94	0.66–1.33	
	age 40		35	1.14	1.01–1.30	
Stove type: Traditional vs <i>Justa</i> (ref)			146			0.73
	age<40		92	1.03	0.58–1.84	
	age 40		54	1.19	0.68–2.06	

Adjusted for age (continuous), dietary diversity score (continuous), years of school (<6 years, ≥6 years)

CI: confidence interval; IQR: interquartile range; PM_{2.5}: fine particulate matter; PR: prevalence ratio