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Effects of a Liquefied Petroleum Gas Stove Intervention on Blood Pressure in Older Adult Women after 24 months

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2015

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Abstract

Effects of a Liquefied Petroleum Gas Stove Intervention on Blood Pressure in Older Adult Women after 24 months

By Brendan Gray

Background: Globally, solid fuels are frequently burned inside homes for cooking or heating leading to excessive household air pollution (HAP) exposure. HAP is a risk factor for elevated blood pressure. In a randomized controlled trial involving older adult women (OAW) from three low- and middle- income countries, we hypothesized that an 18-month liquified petroleum gas (LPG) stove intervention would result in lower systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure (PP), and mean arterial pressure (MAP) in participants compared to those using solid fuel cookstoves, at 6 months after intervention completion.

Methods: This study is part of the Household Air Pollution Intervention Network (HAPIN) trial. Nonpregnant women aged 40-79 were recruited across three resource-limited settings in India, Rwanda, and Guatemala and randomized into either the LPG intervention group or the control group. The intervention lasted 18 months. Each participant had blood pressure measurements taken at baseline and 6 months after intervention completion (24-month follow up). An intention-to-treat analysis (ITT) using a linear regression model captured the difference in average SBP, DBP, PP, and MAP due to the intervention. Subgroup ITT effect analysis was performed to identify potential effect modifiers.

Results: At 6 months after intervention completion, there was no statistically significant difference in SBP, DBP, PP, or MAP levels between the control (n=82) and intervention (n=76) groups. The subgroup analysis revealed that the intervention might reduce SBP for participants with baseline SBP less than or equal to 120 (-8.76 mmHg, CI: -14.81, -2.71). However, the intervention was associated with higher SBP among participants with an SBP greater than 120 (15.407, CI: 9.62, 21.19) a DBP greater than 80 (15.537, CI: 8.25, 22.82), an age greater than or equal to 50 (6.988, CI: 0.34, 13.63), or a BMI greater than or equal to 25 (10.01, CI: 3.22, 16.79).

Conclusion: In an LPG stove intervention in an LMIC setting, there was no difference in blood pressure in OAW between the intervention group and the control group at 6 months after the 18-month intervention. The intervention, however, may have an impact based on specific subgroups.

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Background

For more than 2 billion people worldwide, particularly in low- and middle-income countries (LMICs), solid fuels (e.g. wood, coal, charcoal, dung, and crop waste) are routinely used for cooking and heating.[1] The burning of these fuels indoors exposes families to household air pollution (HAP), which is composed of high levels of fine particulate matter (PM), carbon monoxide, and black carbon, among other pollutants.[1]

Chronic exposure to HAP is one of the many preventable risk factors (e.g. outdoor air pollution, tobacco use, unhealthy diet, physical inactivity, and harmful alcohol consumption) that is associated with an increased risk of non-communicable diseases (NCDs).[2] These include hypertension, chronic obstructive pulmonary disease, ischemic heart disease, stroke, and lung cancer, among others.[2] The prevalence of these risk factors have contributed to an increase in NCD burden in LMICs, where 7 out of 10 deaths are the result of NCDs.[3] Notably, in 2014 the World Health Organization (WHO) reported that HAP was "responsible for the world's largest single environmentally-related disease burden."[4] Furthermore, it has been estimated that 3.2 million people die prematurely from illnesses associated with HAP exposure.[2] Exposure to HAP differs across socio-demographic development, where countries with high Socio-demographic index (SDI) have had decreasing HAP exposure, while countries with low SDI continue to use solid fuels residentially, thus driving increased HAP exposure.[5, 6]

As noted above, HAP is associated with NCDs such as hypertension. Hypertension, or high blood pressure, occurs when the pressure exerted by the blood on the arterial vessel walls is persistently elevated. Physiologically, blood pressure is influenced primarily by cardiac output, the resistance of vessel walls, the autonomic nervous system, and the kidneys.[7] Interference with

any one of these components can lead to elevated (or decreased) blood pressure.[7] Clinically, hypertension is often defined as a measured systolic blood pressure (SBP) ≥140 mmHg and/or a diastolic blood pressure (DBP) ≥90 mmHg on two different days.[8] More recently, the American Heart Association has defined high blood pressure as a measured SBP ≥130 mmHg and/or a DBP pressure ≥80 mmHg.[9]

High blood pressure is a well-recognized risk factor for multiple NCDs, notably cardiovascular disease, stroke, and kidney disease.[8] However, it should be noted that there is a continuous, incremental risk of these diseases across increased levels of both SBP and DBP, without clear evidence of a specific threshold.[7] Nevertheless, elevations in SBP and DBP – as well as other blood pressure components such as pulse pressure (PP) and mean arterial pressure (MAP) – are independent risk factors for NCDs.[10-12] Elevated SBP is an important predictor for adverse cardiovascular events, stroke, and kidney function decline.[10-12] Increased DBP is also independently considered a predictor of stroke but there is still inconclusive evidence on its utility as a predictor of cardiovascular disease.[11, 13] High PP, the difference between SBP and DBP (normal = 40 mmHg), has been shown to be a predictor of worsening kidney function and cardiovascular health.[10, 14] Lastly, increased cumulative MAP, which is defined as the mean blood pressure throughout the cardiac cycle, is an independent risk factor of ischemic stroke in patients with hypertension.[15]

Elevated blood pressure can result from numerous causes, some modifiable and non-modifiable.[16] Age is the most important non-modifiable cause, since blood vessel walls become stiffer as adults get older.[16] Socioeconomic status of a particular region is another important non-modifiable factor that has contributed to the prevalence of hypertension. From 1990-2019, the number of people with hypertension worldwide has doubled, with the largest increase occurring

in LMICs, where over 1 billion people in those regions currently have hypertension.[17] The prevalence of hypertension has decreased in high-income countries (HIC), which has been driven by access to blood pressure treatments and resources that support healthy lifestyle modifications necessary for normal blood pressure.[18]

Modifiable risk factors, such as smoking, obesity, high sodium intake, and air pollution also cause elevated blood pressure.[16] Expanding on the physiologic impact of air pollution, specifically, the inhaled PM causes oxidative stress, lung inflammation, and autonomic imbalance, which leads to blood vessel dysfunction and systemic inflammatory response, ultimately resulting in increased blood vessel resistance and elevated blood pressure.[19] Given the significant prevalence of HAP exposure in LMICs compared to their HIC counterparts, HAP may be playing an important role in the overall burden of hypertension in LMICs.

There have been many studies that have reported the association between HAP exposure and elevated blood pressures.[20-24] For example, a study utilizing data from the Demographic and Health Surveys from 12 LMICs found that cooking with solid fuels was associated with increases in blood pressure and odds of hypertension in pre-menopausal women.[25] Similarly, in China an investigation from the prospective urban and rural epidemiology (PURE) study found that cleaner fuel types was associated with lower blood pressure and prevalence of hypertension.[26] These two studies reflect the potential relationship between HAP and hypertension, but these were both observational and cross-sectional studies that did not capture longitudinal exposure to HAP. Another study performed a meta-analysis looking at the effect of improved biomass cookstoves in LMICs in the reduction of air pollutants (i.e. carbon monoxide and particulate matter) and mean SBP and DBP in non-pregnant adults.[27] The study concluded that the greatest reduction of blood pressure was associated with the use of improved cookstoves

with chimney feature, however only 4 studies were used for this analysis and other clean fuel alternatives were not included in the analysis.[27]

Notably, several studies have investigated the relationship between HAP and blood pressure specifically in non-pregnant adult women.[20, 28, 29] This is an important demographic considering that women in many LMICs are commonly responsible for collecting and burning solid fuels for cooking.[30-32] A recent study in Uganda and Ethiopia revealed that adult women disproportionately spend more time cooking and experience a much higher exposure to PM from HAP compared to men in the same age group.[33] Since women are at a higher risk of excess HAP exposure, it is important to consider the potential relationship between HAP and high blood pressure in adult non-pregnant women. Blood pressure and risk of hypertension increases with age for both men and women. But post-menopausal women (on average >51 years old) are at higher risk of developing hypertension because of decreasing estrogen levels contributing to increased blood vessel stiffness.[34] Given this physiologic disposition, excess HAP exposure may contribute to elevated blood pressure and hypertension in older adult women (OAW).

A study conducted in rural China captured this relationship, where for women >50 years old, a 1-log-$\mu g/m^3$ increase in PM exposure was associated with 4.1 mmHg higher SBP (95% CI, 1.5 to 6.6) and 1.8 mmHg higher DBP (95% CI, 0.4 to 3.2).[35] Another cross-sectional study in Honduras looking at HAP exposure and blood pressure for women using traditional stoves versus a cleaner Justa stove found that a unit increase in natural log-transformed kitchen $PM_{2.5}$ concentration was associated with an SBP increase of 5.2 mmHg in women >40 years old.[20] These studies provide important insight on the impact of HAP on blood pressure in OAW, and reveal the potential impact of a HAP-reducing intervention on lowering blood pressure.[20] However, more longitudinal interventional data is required to understand the potential solutions

for HAP reduction and the implications for blood pressure in OAW.

Among the WHO's Sustainable Development Goals (SDG) for 2030 is the pledge for clean household energy through universal access to affordable, reliable and modern energy services (e.g. electricity, biogas, liquified petroleum gas, and solar fuels).[36] Despite this call, however, several challenges are slowing its attainment, particularly in LMICs.[37, 38] As alluded to in the SDG, affordability of cleaner fuels is a significant barrier, primarily for lower income households.[39] Poorer households have difficulty affording and maintaining cleaner fuel options, especially when there are no options for financial assistance, such as government subsidies.[39] Additionally, cultural and traditional values have served as another barrier to the use of cleaner fuel options.[40] For example, families note the importance of preparing their local dishes to the same taste, using the same cooking utensils, and cooking for large gatherings when considering the adoption of an improved cookstove.[40] Interventions to improve access to clean household energy, and ultimately reduce HAP exposure, must consider multiple cultural, social, economic, and government factors when implementing projects.[39] The Household Air Pollution Intervention Network (HAPIN) trial is one such project that is attempting to accomplish this.

HAPIN is a multi-center, randomized controlled trial in Guatemala, India, Peru, and Rwanda.[41] The intervention group consists of households receiving liquified petroleum gas (LPG) stoves with free fuel supply, while the control group consists of households using solid fuels. LPG is a widely available fuel in LMICs that is easy to use, reduces cook time, and can significantly reduce emissions.[38] But, LPG trials that have been conducted have yet to demonstrate significantly improved health outcomes.[41] The primary outcomes in the HAPIN trial are child birthweight, pneumonia incidence and linear growth, and blood pressure in OAW through the first 18 months.[41] However, the study received funding to extend follow-up of study

participants in Rwanda, India, and Guatemala through 24 months.

At this point, a cross-sectional analysis of the OAW in the trial found that there was a positive association between PM exposure and SBP and PP, which increased in women aged 65 years old.[42] This present study will further investigate this relationship by using data from the HAPIN trial to determine whether there is a difference in blood pressure of OAW in the intervention group versus the control group at 6 months after the intervention. Our hypothesis is that the intervention group will have lower SBP, DBP, PP, and MAP at 6 months after the intervention compared to the control group.

Reducing exposure to HAP is a critical opportunity to protect the health of women and their families, mitigate the effects of climate change, and improve social and economic experiences for many in LMICs.[30] With more data regarding the health impacts of an LPG intervention, our hope is to better inform policy makers on scalable and viable solutions to reduce HAP exposure.

Methods

Study Design and Location

This analysis is using baseline and 24-month follow up data from the HAPIN trial. The HAPIN trial is a multi-center, randomized controlled trial of a HAP intervention consisting of an LPG stove, continuous fuel distribution, and behavioral messaging among 3200 households in four resource-limited LMIC settings: Tamil Nadu, India; Jalapa, Guatemala; Puno, Peru; Kayonza Rwanda.[41] These settings were selected because of the widespread use of solid biomass as the primary fuel type. To encourage generalizability, the locations were intentionally selected to represent a diversity of factors that may influence the effect of the intervention, including altitude, population density, cooking practices, baseline pollution levels, and sources of pollution other than cooking.[41]

In each setting, pregnant women aged 18-35 years old were recruited from households that used solid biomass as the primary fuel, and then randomized in a 1:1 to ratio to the intervention and control groups. The intervention group received an LPG stove, a continuous supply of LPG fuel delivered to the homes for 18 months, and educational messages that promoted safe and exclusive use of the LPG stove for cooking. The stove and fuel were provided free of charge to all the intervention households. The control group did not receive an LPG stove or fuel during the study period and were anticipated to continue cooking with solid biomass fuel. Control households received compensation designed to achieve three aims: comply with applicable ethics requirements for treatment of controls, compensate control participants for the burden associated with this study, and offset the economic advantage to intervention households resulting from the provision of free stoves and fuels.[41]

Trial Population and Inclusion/Exclusion Criteria

Among the eligible households, OAW were actively recruited (one per household). OAW were included if they were aged 40 to <80 years old by self-report (confirmed with a government-issued ID whenever possible). Eligible participants were excluded if they were smoking cigarettes or using other tobacco products at the time of recruitment, were pregnant, were planning to move out of her household in the next 12 months, or were taking blood pressure medication at enrollment or at any point during follow-up. This group is the basis of this investigation.[41] All participants were initially followed longitudinally for ~18 months, however the study was extended to 24 months in Guatemala, India, and Rwanda. Since there is no 24-month data from Peru, all participants from Peru will be excluded from this analysis.

Outcomes and Measurements

Baseline Characteristics

Following the recruitment and informed consent of participants, a baseline visit was conducted by trained field workers to perform surveys and other assessments. Topics that participants were surveyed on included education, dietary diversity (via Minimum Dietary Diversity for Women), and household food insecurity (via The Food Insecurity Experience Scale).[41, 43, 44] Additionally, height and weight measurements of participants were collected and their body mass index (BMI) was calculated.

Blood Pressure

The primary outcomes for the HAPIN trial were child birthweight, pneumonia incidence and linear growth, and blood pressure in OAW. The focus of this analysis is blood pressure in OAW 6 months after the completion of the 18-month intervention (24-month follow-up).

A nurse or a trained field worker measured resting blood pressure in the right arm of the participants using an automatic blood pressure monitor (model HEM-907XL; Omron®). Prior to measurements, participants needed to confirm that they had not smoked, consumed alcohol or a caffeinated beverage (coffee, tea, or cola), or cooked using biomass in the 30 minutes prior to the measurement. If they had, they were asked not to do these activities for 30 minutes before proceeding. These precautions were taken to ensure accurate measurement of the participant's true blood pressure. The participants' blood pressures were measured after being seated in a back-supported chair in a quiet room for 5 minutes with their legs uncrossed and arms supported by a table. Three measurements were taken with at least 2 minutes between each measurement.

SBP and DBP were recorded. Per The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (2003 Guideline), an SBP <120 mmHg or a DBP <80 mmHg was 'Normal;' an SBP 120-139 mmHg or DBP 80-89 mmHg was 'At Risk;' and an SBP $\geq$ 140 mmHg or a DBP $\geq$ 90 mmHg was considered

'Hypertension.'[45] If a participant had an SBP ≥ 140 mmHg and/or a DBP ≥ 90 mmHg then their blood pressure was checked again during the same encounter. If the same result was seen in two subsequent measurements, then the participant was referred to the nearest health center or hospital to receive age-appropriate treatment. If a participant had an SBP <80 mmHg or a DBP <40 mmHg, they were also referred to the nearest health center or hospital for treatment.

In the analyses, the average of the three SBP and DBP measurements were used. SBP values that were <70 mmHg and DBP values that were <35 mmHg were excluded from analyses as implausible measurements. Finally, mean arterial pressure (MAP) was calculated as *DBP + (SBP – DBP)/3*, and pulse pressure (PP) was calculated as *SBP – DBP*. SBP, DBP, MAP and PP are the primary endpoints of interest.

Statistical Analysis

Intention-to-treat Analysis

The primary blood pressure outcomes were analyzed using an intention-to-treat analysis (ITT). The blood pressure measurements of OAW were analyzed using a regression model to examine the differences in mean blood pressure at 6 months after the intervention, while adjusting for study site. In this analysis, OAW from IRC sites Kayonza, Rwanda; Jalapa, Guatemala; Villupuram, India; and Nagapattinam, India were investigated.

The regression model was given by:

$$y_i = \beta_0 + \beta_1 X_{1i} + \beta_2 X_{2i} + \cdots \beta_5 X_{5i} + \varepsilon_i$$

where for individual i, y_i was the blood pressure measurement at 24-month follow-up, X_{1i} was an indicator variable (0 for control and 1 for intervention), X_{2i-5i} was the indicator variable for study

site (Kayonza, Jalapa, Villupuram, Nagapattinam), and $\varepsilon_i \sim N(0, \tau_i^2)$ represented independent Normal error. β_1 was the parameter of interest capturing the differences in blood pressure (i.e. SBP, DBP, MAP, and PP) due to the intervention. This analytical approach was the same pre-specified for the primary blood pressure outcome in the trial and published with the trial registration.

Subgroup Analysis

Age, BMI, baseline SBP and baseline DBP were treated as categorical variables and served as subgroups for the ITT analysis. Age was categorized as greater than or equal to the median age or less than the median age; BMI was categorized as a BMI greater than or equal to 25 was 'Overweight' and a BMI less than 25 was normal; baseline SBP was categorized as less than or equal to 120 was normal and greater than 120 was elevated; and baseline DBP was categorized as less than or equal to 80 was normal and greater than 80 was elevated. Based on these subgroups, effect modification analyses were performed with interaction terms between the indicator variable for the intervention and the effect modifiers. ITT effect was calculated for each subgroup, where an estimate capturing the difference between the intervention and control for the SBP, DBP, PP, and MAP at 24 months was reported.

All statistical analysis was completed using SAS statistical software.

The study protocol was reviewed and approved by institutional review boards at Emory University (00089,799), Johns Hopkins University (00007403), Sri Ramachandra Institute of Higher Education and Research (IEC-N1/16/JUL/54/49) and the Indian Council of Medical Research-Health Ministry Screening Committee (5/8/4–30/(Env)/Indo-US/2016-NCD-I), Universidad del Valle de Guatemala (146-08-2016) and Guatemalan Ministry of Health National Ethics Committee (11–2016), Asociación Benefica PRISMA (CE2981.17), the London School of

Hygiene and Tropical Medicine (11,664–5) and the Rwandan National Ethics Committee (No.357/RNEC/2018), and Washington University in St. Louis (201611159). The parent trial is registered with ClinicalTrials.gov (Identifier NCT02944682).

Results

Baseline Characteristics

In total, 285 OAW from India, Guatemala, and Rwanda (participants from Peru were excluded from this analysis) were randomized into either the LPG group or the control group (Figure 1). Among them 239 received baseline blood pressure measurements, and 158 OAW had 24-month follow up (Table 1 and 2). These 158 OAW are the basis of our analysis. Among the 158 participants, 82 were assigned to the control group, while 76 were randomized into the intervention group (Table 1). In the control group, most participants were from Guatemala (50%), followed by India (40.2%), then Rwanda (9.8%); in the intervention group the order was India (47.4%), Guatemala (40.8%), and Rwanda (11.8%) (Table 1). Overall, Guatemala (48.7%) had the most participants followed by India (40.5%) and Rwanda (10.8%) (Table 1).

The average age of all participants was 51.4 (SD: 6.4) while the median was 50.1 (Min: 40.2, Max: 73.8). This was balanced between the control and intervention groups (Table 1). Most participants did not have any formal education or incomplete primary school (92.2%), which was reflected evenly between the control and intervention groups (Table 1). The average body mass index (BMI) for all participants was 23.4 (SD: 4.5), with a median of 22.3 (Min: 15.4, 37.4); this was balanced between the control and intervention groups (Table 1). The minimum diet diversity was ‘Low’ for most participants (84.5%), but the intervention group had a higher proportion (85.5%) compared to the control group (80.5%) (Table 1). Only 1 participant overall had a minimum diet diversity of ‘High.’ Regarding household food insecurity, most participants from

both groups reported none (65%), but the intervention group had a higher proportion (71.1%) compared to the control group (59.3%). Additionally, more participants in the control group reported having 'Moderate/Severe' household food insecurity (17.3%) compared to the intervention group (5.2%).

Compared to the participants that were randomized but did not have 24-month follow up (Table 2), there was a smaller proportion of participants in both the control and intervention group who reported 'None' (Control: 47.5%, Intervention: 60.5%) regarding household insecurity (Table 2). Otherwise, this group was balanced with the 24-month follow up group.

Baseline and 24-month Blood Pressure Measurements

The baseline average SBP, DBP, and MAP for the intervention group (SBP = 119.8 mmHg, SD: 16.9; DBP mmHg = 74.6, SD: 10.3; MAP mmHg 89.7, SD: 11.9) was higher compared to that of the control group (SBP = 117.2 mmHg, SD: 14; DBP = 71.8 mmHg, SD: 10.7; MAP 86.9 mmHg, SD: 11.3) (Table 3). Additionally, the maximum SBP observed for the intervention group was higher (187.3 mmHg) compared to that of the control group (160.3 mmHg) (Table 3). The PP mean and median were balanced between the two groups (Table 3).

After 24 months, the mean SBP for the intervention group (115.6 mmHg, SD: 17.3) was higher compared to the control group (112.2 mmHg, SD: 11.1) (Table 5). The maximum SBP for the intervention group (197.3 mmHg) was also higher compared to the control group (151.3 mmHg). The mean DBP of the intervention group (72.3 mmHg, SD 11.1) was also higher compared to the control group (70.4 mmHg, SD: 7.7) (Table 5). The mean PP of the intervention group (43.3 mmHg, SD: 12) was also higher than that of the control group (41.8 mmHg, SD: 8.5). Finally, the mean MAP was higher in the intervention group (86.7 mmHg, SD: 12.2) compared to the control group (84.3 mmHg, SD: 8.0).

Intention-to-treat Analysis

For the ITT analysis, a linear regression model was fitted to investigate the effect of the intervention group on the SBP, DBP, PP, and MAP at 24-month follow up, while adjusting for study site. Starting with the SBP, the estimated coefficient for the study arm was 3.35 (CI: -1.207, 7.92), indicating that on average the intervention group had a 3.35 mmHg higher SBP compared to the control group at 24-month follow up, controlling for site (Table 6). This difference was not statistically significant.

For DBP, the estimated coefficient for the study arm was 1.935 (CI: -1.039, 4.944), indicating on average the intervention group had a 1.935 mmHg higher DBP compared to the control group, controlling for site (Table 6). This was not statistically significant.

For PP, the estimated coefficient for the study arm was 1.404 (CI: -1.807, 4.615), indicating that on average the intervention group had a 1.404 mmHg higher PP compared to the control group at 24-month follow up, controlling for study site (Table 6). This was not statistically significant.

For MAP, the estimated coefficient was 2.42 (CI: -0.838, 5.679), indicating on average the intervention group had a 2.42 mmHg higher MAP compared to the control group at 24-month follow up, controlling for study site (Table 6). This was not statistically significant.

Subgroup Analysis

For the subgroup analysis, we calculated the ITT effect for the outcomes of interest (SBP, DBP, PP, and MAP) for each subgroup (Table 7). The estimate of interest was the difference in the outcome between the intervention and control.

For participants greater than or equal to 50 years old, the SBP for the intervention group was on average 6.988 (CI: 0.34, 13.63) mmHg higher compared to the control group, which was statistically significant at the 0.05 level. The PP for the intervention group was on average 5.793

mmHg (CI: 1.23, 10.36) higher compared to the control group, which was statistically significant at the 0.05 level.

For participants with a baseline SBP less than or equal 120, the SBP for the intervention group was on average 8.76 mmHg (CI: -14.81, -2.71) lower compared to the control group, which was statistically significant at the 0.05 level. The DBP for the intervention group was on average 4.432 mmHg lower compared to the control group, which was statistically significant at the 0.05 level. The MAP for the intervention group was on average 5.87 mmHg (CI: -10.25, -1.49) lower compared to the control group, which was statistically significant at the 0.05 level.

For participants with a baseline SBP greater than 120, the SBP for the intervention group was on average 15.407 mmHg (CI: 9.62, 21.19) higher compared to the control group, which was statistically significant at the 0.05 level. The DBP for the intervention group was on average 7.88 mmHg (CI: 3.93, 11.83) higher compared to the control group, which was statistically significant at the 0.05 level. The PP for the intervention group was 7.526 mmHg (CI: 3.26, 11.79) higher compared to the control group, which was statistically significant at the 0.05 level. The MAP for the intervention group was on average 10.39 mmHg (CI: 6.2, 14.58) higher compared to the control group, which was statistically significant at the 0.05 level (Table 7).

For participants with a baseline DBP greater than 80, the SBP for the intervention group was on average 15.537 mmHg (CI: 8.25, 22.82) higher compared to the control group, which was statistically significant at the 0.05 level. The DBP for the intervention group was on average 8.196 mmHg (CI: 3.36, 13.03) higher compared to the control group, which was statistically significant at the 0.05 level. The PP for the intervention group was on average 7.34 mmHg (CI: 2.08, 12.6) higher compared to the control group, which was statistically significant at the 0.05 level. The MAP for the intervention group was on average 10.643 mmHg (CI: 5.43, 15.85) higher compared

to the control group, which was statistically significant at the 0.05 level (Table 7).

For participants with a baseline BMI greater than or equal to 25, the SBP for the intervention group was on average 10.01 mmHg (CI: 3.22, 16.79) higher compared to the control group, which was statistically significant at the 0.05 level. The DBP for the intervention group was on average 7.671 mmHg (CI: 3.32, 12.02) higher compared to the control group, which was statistically significant at the 0.05 level. The MAP for the intervention group was on average 8.45 mmHg (CI: 3.68, 13.22) higher compared to the control group, which was statistically significant at the 0.05 level (Table 7).

Discussion

In this RCT involving OAWs from India, Guatemala, and Rwanda, we hypothesized that an 18-month LPG stove intervention would result in lower SBP, DBP, PP, and MAP in participants compared to those using solid fuel cookstoves, at 6 months after intervention completion. Through an ITT analysis, we found that there was no statistically significant difference in the blood pressure outcomes between the control and intervention groups. Our subgroup analysis revealed that the intervention might reduce blood pressure for participants with baseline SBP less than or equal to 120. However, the intervention was associated with higher blood pressure among participants that had an SBP greater than 120, a DBP greater than 80, an age greater than or equal to 50, or a BMI greater than or equal to 25.

This study was part of the HAPIN trial, which has previously shown that a longitudinal LPG intervention is able to significantly reduce $PM_{2.5}$, black carbon and carbon monoxide exposure.[46, 47] Given the physiologic connection between HAP and blood pressure, as well as evidence from previous observational studies, we anticipated that a reduction in chronic HAP exposure would subsequently lead to a reduction in blood pressure in OAW. However, this was

not the case and there are several contributing factors.

A potential concern could be the sample size (82 control participants and 76 intervention participants). Participants from Peru discontinued the trial after 12 months, and they represented a significant proportion of the original study population (Figure 1). A larger sample may have more adequately captured the relationship between the intervention and blood pressure.

Another concern may be the characteristics of our study population. All participants were non-smokers, and most had baseline SBPs and DBPs within the "Normal" range and a BMI below 25 (Table 3). Previous studies investigating non-pharmacologic blood pressure reduction interventions (e.g. diet, weight loss, exercise) looked predominantly at participants with elevated blood pressure at baseline.[48-50] Our subgroup analysis suggests that there may be effect modification on the intervention based on baseline SBP, BMI, and age. But given the small sample for each of these subgroups, further investigation with participants at higher baseline cardiovascular risk may prove to be informative.

There is also the consideration that HAP's impact on blood pressure may be too small to measure or masked by other factors. Cardiopulmonary outcomes and Household Air Pollution (CHAP) trial in Peru was another LPG stove intervention that measured blood pressure as a health outcome of interest with women aged 25-64 years old.[51] Similar to our study, they also found no difference in blood pressure between the control and intervention groups after 12 months.[51] They hypothesized that HAP exposure may only have a small impact on blood pressure.[51] This is important to note, because the effect of HAP could be disguised by other lifestyle modifications occurring simultaneously, which may improve or worsen blood pressure. Different non-pharmacologic interventions for high blood pressure management have been shown to have different degrees of impact. For example, a weight loss of 10 kg can decrease SBP by 5-20 mmHg,

while the “Dietary Approaches to Stop Hypertension” (DASH) diet has been shown to reduce SBP by 5.5 mmHg.[50] In this investigation, we unfortunately did not have 24-month data on BMI or diet changes for the participants. Considering that blood pressure is a very dynamic measurement that is easily influenced by numerous factors, the impact of reducing HAP exposure must be evaluated in the context of other influential factors.

Strengths and Limitations

Much of the current literature describing the association between HAP exposure and high blood pressure in non-pregnant adult women has been observational studies or short-term exposure studies, with very few long-term controlled and uncontrolled studies.[20-24, 28, 52-55] The HAPIN trial is a longitudinal RCT investigating an LPG stove intervention, its impact on personal exposure to HAP, and its potential health implications (i.e. stunting in infants, child birthweight, pneumonia incidence and linear growth, and blood pressure in older adult women). Our study, as part of the HAPIN trial, contributes to our understanding of the effects of an LPG intervention on the blood pressure of non-pregnant adult women. Specifically, it provides insight on the latent effects of an LPG intervention 6 months after completion of the intervention. This is a strength because previous trials only had up to one year of follow up with little follow up after intervention completion.[28, 55] In addition, the ITT analysis ensured that the intervention reflected the best archivable real-world experiences and provided informative estimates on the impact of the intervention.

Although our investigation had several strengths, there were also limitations that are important to address. As noted above, the sample size of OAW with 24-month follow up was much smaller compared to the baseline sample size, which may have affected the power and generalizability of this study. In addition, previous studies within the HAPIN trial had conducted

exposure-response analysis. This study did not have personal exposure data for the participants at the time of analysis. This study also did not have stove utilization data at the time of analysis, which would have been valuable in understanding whether households continued to use LPG stoves or returned to solid fuel stoves after the intervention. Finally, this study would benefit from additional data collection to understand how the LPG stove affected the participant's life experiences. For example, understanding how the intervention changed the participants' diet or how it affected physical activity (collecting less solid fuel) could have provided important information on factors that can affect blood pressure.

Table 1. Baseline characteristics for older adult women with 24-month follow-up, by study arm

	Control (n=82)	Intervention (n=76)	Total (n=158)
Age			
Mean (SD)	51.7 (6.4)	51 (8.5)	51.4 (7.5)
Median [Min, Max]	50.3 [41,73.8]	49.3 [40.2, 71.7]	50.1 [40.2, 73.8]
< 50 y/o	37 (45.12%)	40 (52.63%)	77 (48.73%)
≥ 50 y/o	45 (54.88%)	36 (47.37%)	81 (51.27)
IRC			
India	33 (40.2%)	36 (47.4%)	64 (40.5%)
Guatemala	41 (50%)	31 (40.8%)	77 (48.7%)
Rwanda	8 (9.8%)	9 (11.8%)	17 (10.8%)
Highest education completed			
No Formal education or Primary school incomplete	74 (93.7%)	68 (90.7%)	142 (92.2%)
Primary school complete or Secondary school incomplete	5 (6.3%)	6 (8%)	11 (7.1%)
Secondary school complete or Vocational or Some college or university	0	1 (1.3%)	1 (0.7%)
Missing	3	1	4
Body Mass Index (BMI)			
Mean (SD)	23.4 (4.5)	23.5 (4.5)	23.4 (4.5)
Median [Min, Max]	22.8 [15.4, 37.4]	23 [15.7, 34.5]	22.8 [15.4, 37.4]
BMI < 25	51 (62.2%)	48 (63.16%)	99 (62.66%)
BMI ≥ 25	31 (37.8%)	28 (36.84%)	59 (37.34%)
Missing	1	0	1
Minimum Diet Diversity			
Low	66 (80.5%)	65 (85.5%)	131 (82.9%)
Medium	15 (18.3%)	11 (14.5%)	26 (16.4%)
High	1 (1.2%)	0	1 (0.7%)
Household food insecurity			
None	48 (59.3%)	54 (71.1%)	102 (65%)
Mild	19 (23.4%)	18 (23.7%)	37 (23.6%)
Moderate/Severe	14 (17.3%)	4 (5.2%)	18 (11.4%)
Missing	1	0	1

Table 2. Baseline characteristics for older adult women without 24-month follow-up, by study arm

	Control (n=41)	Intervention (n=43)	Total (n=84)
Age			
Mean (SD)	49.2 (7.1)	52.3 (8.1)	50.8 (7.8)
Median [Min, Max]	47.2 [40.5, 68]	50.8 [40.4, 71.6]	49.1 [40.4, 71.6]
IRC			
India	15 (36.6%)	12 (27.9%)	27 (32.1%)
Guatemala	13 (31.7%)	24 (55.8%)	37 (44.1%)
Rwanda	13 (31.7%)	7 (16.3%)	20 (23.81%)
Highest education completed			
No Formal education or Primary school incomplete	34 (85%)	40 (95.2%)	74 (90.2%)
Primary school complete or Secondary school incomplete	2 (5%)	2 (4.8%)	4 (4.9%)
Secondary school complete or Vocational or Some college or university	4 (10%)	0	4 (4.9%)
Missing	1	1	2
Body Mass Index			
Mean (SD)	23.6 (3.8)	23.9 (5.3)	23.8 (4.6)
Median [Min, Max]	23 [18.4, 33.3]	23 [16.1, 38.8]	23 [16.1, 38.8]
Missing	0	2	2
Minimum Diet Diversity			
Low	34 (82.9%)	37 (86.1%)	71 (84.5%)
Medium	7 (17.1%)	4 (9.3%)	11 (13.1%)
High	0	2 (4.7%)	2 (2.4%)
Household food insecurity			
None	19 (47.5%)	26 (60.5%)	45 (54.2%)
Mild	13 (32.5%)	11 (25.6%)	24 (28.9%)
Moderate/Severe	8 (20%)	6 (14%)	14 (16.9%)
Missing	1	0	1

Table 3. Baseline SBP, DBP, PP, & MAP for older adult women with 24-month follow-up, by study arm

	Control	Intervention	Total
N (missing)	82 (0)	76 (1)	158 (1)
SBP			
Mean (SD)	117.2 (14)	119.8 (16.9)	118.5 (15.5)
Median [Min, Max]	115 [92, 160.3]	117 [93.7, 187.3]	116.7 [92, 187.3]
SBP ≤ 120	48 (58.54%)	42 (55.26%)	90 (57%)
SBP >120	34 (41.46%)	34 (44.74%)	68 (43.04%)
DBP			
Mean (SD)	71.8 (10.7)	74.6 (10.3)	73.1 (10.6)
Median [Min, Max]	70.7 [49, 105]	74 [53.3, 106.3]	72.7 [49, 106.3]
DBP ≤ 80	63 (76.83%)	57 (75%)	120 (76%)
DBP >80	19 (23.17%)	19 (25%)	38 (24%)
PP			
Mean (SD)	45.4 (7.6)	45.2 (10.3)	45.4 (8.9)
Median [Min, Max]	44.5 [31, 71.7]	43.3 [26.7, 81]	44 [26.7, 81]
MAP			
Mean (SD)	86.9 (11.3)	89.7 (11.9)	88.2 (11.7)
Median [Min, Max]	85.4 [63.4, 133.3]	88,8 [68.2, 133.3]	87.3 [63.4, 133.3]

Table 4. Baseline SBP, DBP, PP, MAP for older adult women without 24-month follow-up, by study arm

	Control	Intervention	Total
N (Missing)	41 (2)	43 (0)	84 (2)
SBP			
Mean (SD)	121 (16.6)	115.5 (13.6)	118.1 (15.3)
Median [Min, Max]	116.7 [100.3, 169.3]	116.7 [86, 148.3]	116.7 [86, 169.3]
DBP			
Mean (SD)	72.8 (8.9)	70.2 (10.2)	71.4 (9.7)
Median [Min, Max]	72.3 [56.3, 94.3]	69 [46.3, 97]	70.5 [46.3,97]
PP			
Mean (SD)	48.2 (11.7)	45.3 (8)	46.7 (10)
Median [Min, Max]	45.3 [30.7, 83.3]	44.3 [33.7, 74]	45 [30.7, 83.3]
MAP			
Mean (SD)	88.9 (10.7)	85.3 (10.8)	87 (10.9)
Median [Min, Max]	88.7 [74.1, 113.8]	84.8 [59.6, 108.7]	86.7 [59,6, 113.8]

Table 5. SBP, DBP, PP, MAP for older adult women 24 months after intervention, by study arm

	Control	Intervention	Total
N (missing)	82 (1)	76 (1)	158 (2)
SBP			
Mean (SD)	112.2 (11.1)	115.6 (17.3)	113.8 (14.4)
Median [Min, Max]	111.3 [88.3, 151.3]	110.3 [92.7, 197.3]	111 [88.3, 197.3]
DBP			
Mean (SD)	70.4 (7.7)	72.3 (11.1)	71.3 (9.5)
Median [Min, Max]	70.3 [50.3, 88.3]	70.7 [53, 106.3]	70.7 [50.3, 106.3]
PP			
Mean (SD)	41.8 (8.5)	43.3 (12.0)	42.5 (10.3)
Median [Min, Max]	50 [26.3, 76.3]	42 [18.33, 100.3]	41.7 [18.3, 100.3]
MAP			
Mean (SD)	84.3 (8.0)	86.7 (12.2)	85.5 (10.3)
Median [Min, Max]	84.9 [63, 106.1]	84 [67.3, 130.4]	84.3 [63, 130.4]

Table 6. Results of ITT analysis for the difference between intervention and control arms for average SBP, DBP, PP, and MAP

	Estimate*	95% CI	p-value		Estimate	95% CI	p-value
SBP	3.35	-1.207, 7.92	0.148	**PP**	1.404	-1.807, 4.615	0.389
DBP	1.953	-1.039, 4.944	0.199	**MAP**	2.42	-0.838, 5.679	0.144

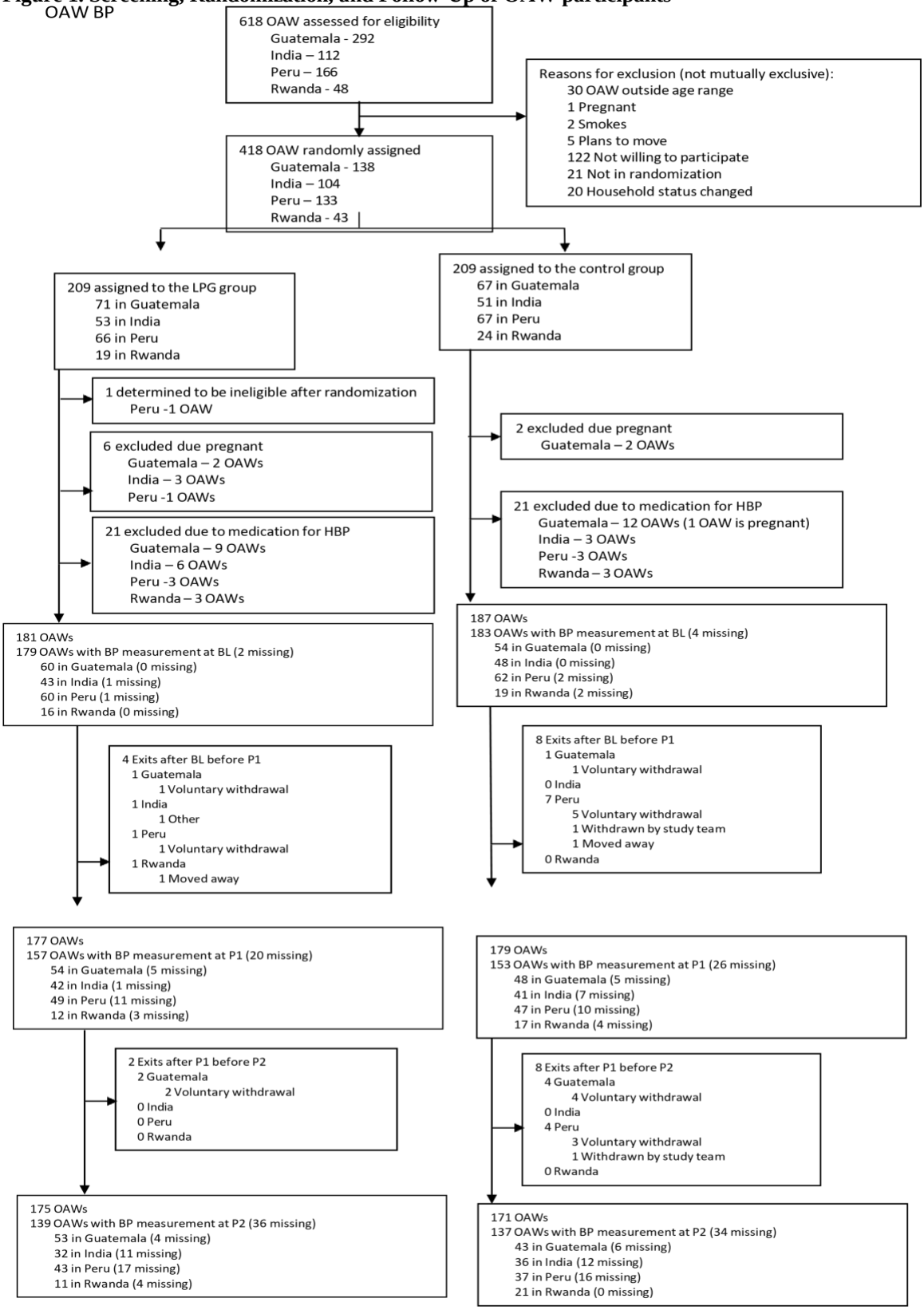
*Difference between the intervention and control's blood pressure outcomes

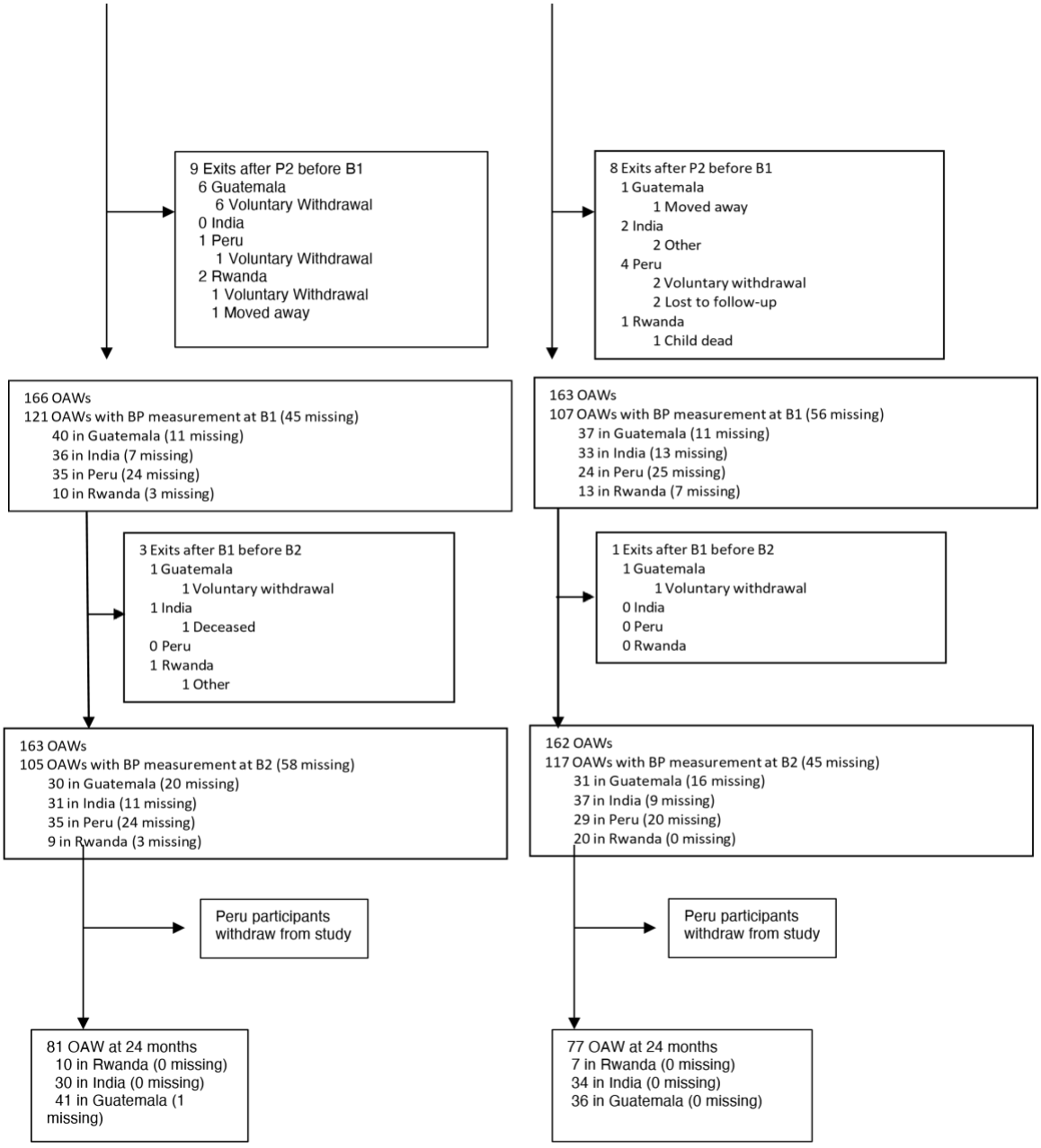
Table 7. ITT Effect by Age, Baseline BMI for ITT analyses of SBP, DBP, PP, and MAP

Outcome	Subgroup		Estimate*	Confidence Interval	P-value for interaction
SBP	**Age**	< 50 y/o	0.266	-5.89, 6.42	0.9322
		≥ 50 y/o	6.988	0.34, 13.63	<0.05
	Baseline SBP	SBP ≤ 120	-8.76	-14.81, -2.71	<0.05
		SBP >120	15.407	9.62, 21.19	<0.05
	Baseline DBP	DBP ≤ 80	-5.962	-13.33, 1.41	0.112
		DBP >80	15.537	8.25, 22.82	<0.05
	Baseline BMI	BMI < 25	1.95	-8.61, 4,69	0.5614
		BMI ≥ 25	10.01	3.22, 16.79	<0.05
DBP	**Age**	< 50 y/o	2.394	-1.65, 6.44	0.244
		≥ 50 y/o	1.196	-3.17, 5.57	0.59
	Baseline SBP	SBP ≤ 120	-4.432	-8.56, -0.30	<0.05
		SBP >120	7.88	3.93, 11.83	<0.05
	Baseline DBP	DBP ≤ 80	-3.759	-8.65, 1.13	0.1311
		DBP >80	8.196	3.36	<0.05
	Baseline BMI	BMI < 25	-3.211	-7.47, 1.05	0.1385
		BMI ≥ 25	7.671	3.32, 12.02	<0.05
PP	**Age**	< 50 y/o	-2.128	-6.36, 2.1	0.3216
		≥ 50 y/o	5.793	1.23, 10.36	<0.05
	Baseline SBP	SBP ≤ 120	-4.32	-8.79, 0.13	0.0572
		SBP >120	7.526	3.26, 11.79	<0.05
	Baseline DBP	DBP ≤ 80	-2.203	-7.52, 3.11	0.4143
		DBP >80	7.34	2.08, 12.6	<0.05
	Baseline BMI	BMI < 25	1.25	-3.5, 6.0	0.6032
		BMI ≥ 25	2.336	-2.51, 7.18	0.3427
MAP	**Age**	< 50 y/o	1.684	-2.74, 6.11	0.4531
		≥ 50 y/o	3.1267	1.65, 7.9	0.198
	Baseline SBP	SBP ≤ 120	-5.87	-10.25, -1.49	<0.05
		SBP >120	10.39	6.20, 14.58	<0.05
	Baseline DBP	DBP ≤ 80	-4.493	-9.76, 0.78	0.0941
		DBP >80	10.643	5.43, 15.85	<0.05
	Baseline BMI	BMI < 25	-2.7938	-7.47,1.88	0.2392
		BMI ≥ 25	8.45	3.68, 13.22	<0.05

*Difference between the intervention and control's blood pressure outcomes

Figure 1. Screening, Randomization, and Follow-Up of OAW participants




9 Exits after P2 before B1
6 Guatemala
6 Voluntary Withdrawal
0 India
1 Peru
1 Voluntary Withdrawal
2 Rwanda
1 Voluntary Withdrawal
1 Moved away
8 Exits after P2 before B1
1 Guatemala
1 Moved away
2 India
2 Other
4 Peru
2 Voluntary withdrawal
2 Lost to follow-up
1 Rwanda
1 Child dead
166 OAWs
121 OAWs with BP measurement at B1 (45 missing)
40 in Guatemala (11 missing)
36 in India (7 missing)
35 in Peru (24 missing)
10 in Rwanda (3 missing)
163 OAWs
107 OAWs with BP measurement at B1 (56 missing)
37 in Guatemala (11 missing)
33 in India (13 missing)
24 in Peru (25 missing)
13 in Rwanda (7 missing)
3 Exits after B1 before B2
1 Guatemala
1 Voluntary withdrawal
1 India
1 Deceased
0 Peru
1 Rwanda
1 Other
1 Exits after B1 before B2
1 Guatemala
1 Voluntary withdrawal
0 India
0 Peru
0 Rwanda
163 OAWs
105 OAWs with BP measurement at B2 (58 missing)
30 in Guatemala (20 missing)
31 in India (11 missing)
35 in Peru (24 missing)
9 in Rwanda (3 missing)
162 OAWs
117 OAWs with BP measurement at B2 (45 missing)
31 in Guatemala (16 missing)
37 in India (9 missing)
29 in Peru (20 missing)
20 in Rwanda (0 missing)
Peru participants withdraw from study
Peru participants withdraw from study
81 OAW at 24 months
10 in Rwanda (0 missing)
30 in India (0 missing)
41 in Guatemala (1 missing)
77 OAW at 24 months
7 in Rwanda (0 missing)
34 in India (0 missing)
36 in Guatemala (0 missing)

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