DISSERTATION

INDOOR AIR POLLUTION FROM COOKSTOVE SMOKE AND ADVERSE HEALTH EFFECTS AMONG HONDURAN WOMEN

Submitted by

Maggie L. Clark

Department of Environmental & Radiological Health Sciences

In partial fulfillment of the requirements

For the Degree of Doctor of Philosophy

Colorado State University

Fort Collins, Colorado

Fall 2007

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

UMI Number: 3299796

INFORMATION TO USERS

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleed-through, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.



UMI Microform 3299796 Copyright 2008 by ProQuest LLC. All rights reserved. This microform edition is protected against unauthorized copying under Title 17, United States Code.

> ProQuest LLC 789 E. Eisenhower Parkway PO Box 1346 Ann Arbor, MI 48106-1346

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

COLORADO STATE UNIVERSITY

November 1, 2007

WE HEREBY RECOMMEND THAT THE DISSERTATION PREPARED UNDER OUR SUPERVISION BY MAGGIE L. CLARK ENTITLED INDOOR AIR POLLUTION FROM COOKSTOVE SMOKE AND ADVERSE HEALTH EFFECTS AMONG HONDURAN WOMEN BE ACCEPTED AS FULFILLING IN PART REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY.

Committee on Graduate Work a w Advise **Co-Advise**

Department Head/Director

ABSTRACT OF DISSERTATION

INDOOR AIR POLLUTION FROM COOKSTOVE SMOKE AND ADVERSE HEALTH EFFECTS AMONG HONDURAN WOMEN

Elevated indoor air pollution exposures associated with the burning of biomass fuels in developing countries are well established. Improved cookstoves have the potential to substantially reduce these exposures. Adverse health endpoints, including acute respiratory infections, chronic obstructive pulmonary disease, asthma, and cataract have been associated with biomass-derived indoor air pollution in developing countries, although little research has been performed on cardiovascular health endpoints in these settings. Studies examining the relationship between stove use and adverse health effects have been inconsistent, relying mostly on proxies of exposure, such as type of stove or time spent cooking. Several studies have demonstrated the value of estimating indoor air pollution exposures by evaluating personal cooking practices and household parameters in addition to stove type. We conducted a cross-sectional survey among 79 non-smoking Honduran women in two communities, one semi-urban and one rural. Thirty-eight women cooked with traditional stoves and 41 with improved stoves with chimneys. For a subgroup of these women (N=54-58), carbon monoxide and particulate matter ($PM_{2.5}$) levels were assessed via eight-hour indoor monitoring, as well as eight-hour personal $PM_{2.5}$ monitoring. Kitchen parameters were determined to estimate ventilation rates that may affect carbon monoxide and $PM_{2.5}$. Stove quality was assessed using a four-level subjective scale representing the potential for indoor emissions, ranging from poorly functioning traditional stoves to well-functioning improved stoves. The utility of the four-level stove scale appears to be important as a clear trend of decreasing pollutants was demonstrated as the quality of the stove improved. In multivariate models, the stove scale and ventilation factors predicted more than 50% of the variation in personal and indoor $PM_{2.5}$ and 85% of the variation in indoor carbon monoxide. In addition to the stove scale, other factors predicting exposure measurements were the age of the stove (personal $PM_{2.5}$); the total area of the kitchen windows, the number of kitchen walls, and the primary material of the kitchen walls (indoor $PM_{2.5}$); and the volume of the kitchen and the number of walls with eave spaces (indoor carbon monoxide 1-hr max). Pulmonary function testing, including forced expiratory volume in one second and peak expiratory flow, was performed at the end of each sampling period and respiratory symptoms and demographic characteristics were assessed. Finger-stick blood samples were collected and dried on filter paper in order to assess a biomarker of inflammation, C-reactive protein (CRP). Women using traditional stoves reported symptoms of cough, phlegm, wheeze, headache, and shortness of breath more frequently than those using improved stoves; although associations may have been influenced by the presence of reporting bias. Associations remained, although attenuated, for the association of indoor PM_{2.5} and carbon monoxide with symptoms. Associations consistent with a null association were observed between cookstove exposures and lung function and CRP although some confidence intervals were wide. This study was one of the first to incorporate quantitative air quality measurements, stove quality, and housing characteristics to evaluate the relationship between indoor air pollution and health effects, such as reported symptoms, lung function, and C-reactive protein levels while adjusting

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

for potential confounders. Results of the exposure assessment could provide a costeffective alternative to air quality monitoring. The ease and convenience of collecting, storing, and transporting finger-stick blood samples, could prove to be a useful tool for larger community-based, epidemiologic investigations especially in developing countries.

> Maggie Lynn Clark Department of Environmental & Radiological Health Sciences Colorado State University Fort Collins, CO 80523 Fall 2007

> > v

ACKNOWLEDGEMENTS

This research represents a collaborative effort by many individuals and organizations. I thank Trees Water & People (Fort Collins, Colorado) and the Honduran Association for Development (Tegucigalpa, Honduras) for providing needed logistical assistance during the planning and data collection phases of this project. I gratefully acknowledge these organizations for being dedicated to solving the problem of extreme indoor pollutant exposures experienced by the world's poorest populations through the use of culturally appropriate technologies.

I am extremely thankful for the generous guidance of my committee members throughout my dissertation experience. I am especially grateful to my adviser, Jennifer Peel, for mentoring me through this process, for being an example of an outstanding and dedicated epidemiologist, and for being committed to my development. I greatly appreciate your encouragement and continuous support through our often daily (and sometimes morethan-daily) meetings. I also thank my co-adviser, Stephen Reynolds, for providing direction, leadership, and expertise in exposure monitoring techniques. This project would not have been possible without the monitoring equipment donations of Dr. Reynolds and SKC, Inc. I thank my committee members, James Burch, Tracy Nelson, and Annette Bachand for so willingly sharing with me their knowledge in a variety of specialties, from biostatistics to CRP physiology; and for always supporting me and this project with their time and advice.

vi

I also want to thank my fellow Environmental Health graduate students (both past and present) for sharing this experience with me through late-night study sessions, lunchroom chats, and unending epi-method debates and discussions.

I thank the Honduran women for being such gracious hosts. It is because of these 79 women that this research has the potential to influence and improve the lives of countless other women and children. On a personal note, these women were willing to overcome language and cultural barriers in order to share their lives with us. Because of this, getting to know the women and their families has been one of the greatest experiences I have known.

Finally, I am forever indebted to the Honduras field team, Mara Fernandez Schultz, Shannon Oliver, Augusto Ramirez, and Benjamin Osorto for their tireless efforts and devotion to this project, their camaraderie during the summer of 2005, and especially for their compassion for the people of Suyapa and Santa Lucia.

DEDICATION

For Mom and Dad (Pamela and William Clark):

Thank you for your unending love and support throughout this long, long process of an education. You've taught me how to maintain balance in my life and instilled in me the confidence necessary to pursue my goals. I am forever grateful for your continued belief in me not only as a developing scientist but also as a developing person.

TABLE OF CONTENTS

Chapter:

1. HYPOTHESIS / SPECIFIC AIMS

Introduction	1
Hypothesis	2
Specific Aims	3

2. BACKGROUND & SIGNIFICANCE

Health Effects	5
Respiratory disease & symptoms	7
Lung function	9
Acute lower respiratory infections	11
Lung cancer	12
Perinatal effects	13
Developed countries	14
Mechanisms of Action	15
Particulate matter	16
C-reactive protein	20
Carbon monoxide	21
Summary	23
Improved Stoves & Exposure Assessment	23
Summary	.27

3. RESEARCH DESIGN AND METHODS

Study population	29
Exposure assessment	30
Lung function	33
C-reactive protein	33
Questionnaire	36
Data analyses	37
Exposure assessment models	38

Health effects assessments	42
Lung function & CRP	42
Respiratory symptoms	47
Power.	49
Tables	51

4. **RESULTS**

Descriptive	53
Exposure assessment models	54
Health effect assessments	
Forced expiratory volume in one second	59
Peak expiratory flow	63
C-reactive protein	67
Symptoms	70
Tables	74

5. DISCUSSION

General	209
Limitations	225
Strengths & Summary	232

6.	REFERENCES	235
APPENI	DIX A: QUESTIONNAIRE	253

LIST OF TABLES

3.1	Power estimates to detect differences in FEV_1 among improved and traditional stove users with a sample size = 80 and alpha = 0.05	51
3.2	Power estimates to detect partial correlations between C-reactive protein and indoor pollutant levels for a multivariate model with 4 predictors, a sample size = 60, and alpha = 0.05	52
4.1	Personal characteristics and Spearman correlation coefficients of the study population; total population and stratified by stove type	74
4.2	Characteristics of the study population; total population and stratified by stove type	75
4.3	Mean kitchen and cooking characteristics for the study population; total population and stratified by stove type	.77
4.4	Stove, kitchen, and cooking characteristics of the study population; total population and stratified by stove type	.78
4.5	Air quality means, standard deviations, minimum measures, maximum measures, and geometric means for the total population and stratified by stove type	80
4.6	Air quality means, standard deviations, and geometric means for the study population across levels of the stove scale	81
4.7	Spearman correlation coefficients for air quality measurements	82
4.8	Univariate variation (R-square) in natural logarithm transformed personal PM _{2.5} explained by kitchen and cooking characteristics	83
4.9	Univariate variation (R-square) in natural logarithm transformed indoor $PM_{2.5}$ explained by kitchen and cooking characteristics	84
4.10	Univariate variation (R-square) in natural logarithm transformed indoor carbon monoxide 1-hr maximum explained by kitchen and cooking characteristics.	85

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

4.11	R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed personal PM _{2.5} levels utilizing the stove scale variable (n=58)
4.11.01	Variation in natural logarithm transformed personal $PM_{2.5}$ explained by the stove scale and the age of the stove and stratified by exposures to second-hand smoke and outdoor $PM_{2.5}$ levels
4.12	R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed personal $PM_{2.5}$ levels utilizing the stove type variable (n=58)88
4.13	R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed indoor PM _{2.5} levels utilizing the stove scale variable (n=57)89
4.13.01	Variation in natural logarithm transformed indoor $PM_{2.5}$ explained by the stove scale, the total area of the kitchen windows, the number of kitchen walls, and the primary material of the kitchen walls and stratified by exposures to second-hand smoke and outdoor $PM_{2.5}$ levels
4.14	R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed indoor $PM_{2.5}$ levels utilizing the stove type variable (n=57)
4.15	R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed indoor $PM_{2.5}$ levels utilizing the chimney condition variable (n=57)92
4.16	R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed indoor carbon monoxide 1-hr maximum levels utilizing the stove scale variable (n=54)
4.16.01	Variation in natural logarithm transformed indoor carbon monoxide 1-hr maximum explained by the stove scale, the natural log transformed volume of the kitchen, and the number of kitchen walls with eave spaces and stratified by exposure to second-hand smoke
4.17	R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed indoor carbon monoxide 1-hr maximum levels utilizing the stove type variable (n=54)
4.18	Means and standard deviations (SD) of health endpoints for the study population; total population and stratified by stove type

4.19	Lung function and CRP means and standard deviations for the studypopulation across levels of the stove scale
4.20	Spearman correlation coefficients for three health endpoints among thestudy population
4.21	Reported symptoms among the study population; total population and stratified by stove type
4.22	Reported symptoms among the study population stratified by stove scale 100
4.23	Univariate linear regression estimates and 95% confidence intervals (CI) for the association of air quality measures and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) and potential confounders101
4.24	Selected models of the association between personal $PM_{2.5}$ and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (n=44)102
4.24.01	Adjusted association between Personal PM _{2.5} assessed as a categorical variable and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) (adjusted for age and height) (N=44)103
4.24.02	Adjusted association between Personal PM _{2.5} and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) stratified by various factors
4.24.03	Adjusted association between Personal PM _{2.5} and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) among subgroups of participants105
4.25	Selected models of the association between indoor $PM_{2.5}$ and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=43) 106
4.25.01	Adjusted association between indoor PM _{2.5} assessed as a categorical variable and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) (adjusted for age and height) (N=43)107
4.25.02	Adjusted association between indoor PM _{2.5} and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) stratified by various factors108
4.25.03	Adjusted association between Indoor PM _{2.5} and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) among subgroups of participants109

4.26	Selected models of the association between indoor carbon monoxide 1-hr maximum and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=40)
4.26.01	Adjusted association between indoor carbon monoxide 1-hr maximum assessed as a categorical variable and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) (adjusted for age and height) (N=40)111
4.26.02	Adjusted association between indoor carbon monoxide 1-hr maximum and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) stratified by various factors
4.26.03	Adjusted association between Indoor carbon monoxide 1-hr maximum and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) among subgroups of participants
4.27	Selected models of the association between stove type (Traditional vs. Improved) and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=52)
4.27.01	Adjusted association between stove type (Traditional vs. Improved) and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) stratified by various factors
4.27.02	Adjusted association between stove type (Traditional vs. Improved) and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) among subgroups of participants
4.27.03	Adjusted association between stove type (Traditional vs. Improved) and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) among various ventilation subgroups
4.28	Selected models of the association between stove scale and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=52)118
4.28.01	Adjusted association between stove scale and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) stratified by various factors
4.28.02	Adjusted association between stove scale and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) among subgroups of participants 122

4.28.03	Adjusted association between stove scale and Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) among various ventilation subgroups	123
4.29	Univariate Odds Ratios (OR) and 95% confidence intervals (CI) for the association of air quality measures and dichotomized percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) (less than 80% versus 80% or more) and potential confounders	124
4.30	Selected models for the association between personal $PM_{2.5}$ and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more): crude association, full model, crude model with the addition of each variable individually to evaluate potential confounding (n=44).	125
4.30.01	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of Personal $PM_{2.5}$ assessed as a categorical variable and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) (N=44).	126
4.30.02	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of Personal $PM_{2.5}$ and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) stratified by various factors.	127
4.30.03	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of Personal $PM_{2.5}$ and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) among subgroups of participants.	128
4.31	Selected models for the association between indoor $PM_{2.5}$ and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more): crude association, full model, crude model with the addition of each variable individually to evaluate potential confounding (n=43).	129
4.31.01	Odds Ratio (OR) and 95% confidence intervals (CI) for the association of indoor $PM_{2.5}$ assessed as a categorical variable and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) (N=44).	130
4.31.02	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of indoor $PM_{2.5}$ and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) stratified by various factors.	131

4.31.03	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of indoor $PM_{2.5}$ and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) among subgroups of participants	132
4.32	Selected models for the association between indoor carbon monoxide 1-hr maximum and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more): crude association, full model, crude model with the addition of each variable individually to evaluate potential confounding (n=40)	133
4.32.01	Odds Ratio (OR) and 95% confidence intervals (CI) for the association of indoor carbon monoxide 1-hr maximum assessed as a categorical variable and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) (N=40)	134
4.32.02	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of indoor carbon monoxide 1-hr maximum and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) stratified by various factors	135
4.32.03	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of indoor carbon monoxide 1-hr maximum and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) among subgroups of participants	136
4.33	Selected models for the association between stove type (Traditional vs. Improved) and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more): crude association, full model, crude model with the addition of each variable individually to evaluate potential confounding (n=52).	.137
4.33.01	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove type (Traditional vs. Improved) and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) stratified by various factors.	.138
4.33.02	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove type (Traditional vs. Improved) and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) among subgroups of participants.	139
4.33.03	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove type (Traditional vs. Improved) and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) among various ventilation subgroups	.140

4.34	Selected models for the association between stove scale and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more): crude association, full model, crude model with the addition of each variable individually to evaluate potential confounding (n=52).	141
4.34.01	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove scale and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) stratified by various factors	.142
4.34.02	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove scale and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) among subgroups of participants.	. 145
4.34.03	Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove scale and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less than 80% versus 80% or more) among various ventilation subgroups	. 146
4.35	Univariate linear regression estimates and 95% confidence intervals (CI) for the association of air quality measures and Peak Expiratory Flow (PEF; L/minute) and potential confounders	. 147
4.36	Selected models for the association between personal PM _{2.5} and Peak Expiratory Flow (PEF; L/minute): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (n=44).	. 148
4.36.01	Adjusted association between Personal PM _{2.5} assessed as a categorical variable and Peak Expiratory Flow (PEF; L/minute) (adjusted for age and height) (N=44).	. 149
4.36.02	Adjusted association between Personal PM _{2.5} and Peak Expiratory Flow (PEF; L/minute) stratified by various factors	.150
4.36.03	Adjusted association between Personal PM _{2.5} and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants	151
4.37	Selected models of the association between indoor $PM_{2.5}$ and Peak Expiratory Flow (PEF; L/minute): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=43)	152

4.37.01	Adjusted association between indoor PM _{2.5} assessed as a categorical variable and Peak Expiratory Flow (PEF; L/minute) (adjusted for age and height) (N=43).	153
4.37.02	Adjusted association between indoor PM _{2.5} and Peak Expiratory Flow (PEF; L/minute) stratified by various factors	154
4.37.03	Adjusted association between Indoor PM _{2.5} and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants	155
4.38	Selected models of the association between indoor carbon monoxide 1-hr maximum and Peak Expiratory Flow (PEF; L/minute): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=40)	156
4.38.01	Adjusted association between indoor carbon monoxide 1-hr maximum assessed as a categorical variable and Peak Expiratory Flow (PEF; L/minute) (adjusted for age and height) (N=40)	157
4.38.02	Adjusted association between indoor carbon monoxide 1-hr maximum and Peak Expiratory Flow (PEF; L/minute) stratified by various factors	158
4.38.03	Adjusted association between Indoor carbon monoxide 1-hr maximum and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants	159
4.39	Selected models of the association between stove type (Traditional vs. Improved) and Peak Expiratory Flow (PEF; L/minute): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=52)	.160
4.39.01	Adjusted association between stove type (Traditional vs. Improved) and Peak Expiratory Flow (PEF; L/minute) stratified by various factors	161
4.39.02	Adjusted association between stove type (Traditional vs. Improved) and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants	162
4.39.03	Adjusted association between stove type (Traditional vs. Improved) and Peak Expiratory Flow (PEF; L/minute) among various ventilation subgroups	163
4.40	Selected models of the association between stove scale and Peak Expiratory Flow (PEF; L/minute): crude association, full model, reduced model, and reduced model with the addition of each variable individually to test for potential confounding (N=52)	164

xviii

4.40.01	Adjusted association between stove scale and Peak Expiratory Flow(PEF; L/minute) stratified by various factors
4.40.02	Adjusted association between stove scale and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants
4.40.03	Adjusted association between stove scale and Peak Expiratory Flow (PEF; L/minute) among various ventilation subgroups
4.41	Univariate linear regression estimates and 95% confidence intervals (CI) for the association of air quality measures and natural logarithm transformed C-reactive protein (CRP; mg/L on the dried blood scale) and potential confounders
4.42	Selected models of the association between personal $PM_{2.5}$ and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=50)171
4.42.01	Adjusted association between Personal PM _{2.5} assessed as a categorical variable and natural logarithm transformed C-reactive protein (adjusted for age, height, and waist circumference) (N=50)
4.42.02	Adjusted association between Personal PM _{2.5} and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors 173
4.42.03	Adjusted association between Personal PM _{2.5} and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants
4.43	Selected models of the association between indoor PM _{2.5} and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=49) 175
4.43.01	Adjusted association between indoor $PM_{2.5}$ assessed as a categorical variable and natural logarithm transformed C-reactive protein (mg/L) (adjusted for age, height, and waist circumference) (N=49)176
4.43.02	Adjusted association between indoor PM _{2.5} and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors 177
4.43.03	Adjusted association between Indoor PM _{2.5} and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants

4.44	Selected models of the association between indoor carbon monoxide 1-hr maximum and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=47)
4.44.01	Adjusted association between indoor carbon monoxide 1-hr maximum assessed as a categorical variable and natural logarithm transformed C-reactive protein (CRP; mg/L) (adjusted for age, height, and waist circumference) (N=47)
4.44.02	Adjusted association between indoor carbon monoxide 1-hr maximum and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors
4.44.03	Adjusted association between Indoor carbon monoxide 1-hr maximum and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants
4.45	Selected models of the association between stove type (Traditional vs. Improved) and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=71)
4.45.01	Adjusted association between stove type (Traditional vs. Improved) and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors
4.45.02	Adjusted association between stove type (Traditional vs. Improved) and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants
4.45.03	Adjusted association between stove type (Traditional vs. Improved) and natural logarithm transformed C-reactive protein (CRP; mg/L) among various ventilation subgroups
4.46	Selected models of the association between stove scale and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=71)189
4.46.01	Adjusted association between stove scale and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors 190

4.46.02	Adjusted association between stove scale and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants
4.46.03	Adjusted association between stove scale and natural logarithm transformed C-reactive protein (CRP; mg/L) among various ventilation subgroups
4.47	Odds Ratios (OR) and 95% confidence intervals (CI) for the association between cookstove exposures and the usual presence of a cough as assessed by the question, "Do you usually have a cough?"
4.47.01	Odds Ratios (OR) and 95% confidence intervals (CI) for the association between cookstove exposures (including co-pollutant models) and the usual presence of a cough as assessed by the question, "Do you usually have a cough?"
4.48	Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the usual presence of phlegm as assessed by the question, "Do you usually bring up phlegm from your chest?"197
4.48.01	Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures (including co-pollutant models) and the usual presence of phlegm as assessed by the question, "Do you usually bring up phlegm from your chest?"
4.49	Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the usual presence of wheeze as assessed by the question, "Does your chest usually sound wheezy or whistling?" 199
4.49.01	Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures (including co-pollutant models) and the usual presence of wheeze as assessed by the question, "Does your chest usually sound wheezy or whistling?"
4.50	Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the presence of nasal irritation as assessed by the question, "Do you have current nasal stuffiness, runny nose, sneezing, and/or nasal itch?"
4.51	Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the usual presence of a headache as assessed by the question, "Do you usually develop a headache during cooking?"

4.51.01	Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures (including co-pollutant models) and the usual presence of a headache as assessed by the question, "Do you usually develop a headache during cooking?"	203
4.52	Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the usual presence of shortness of breath as assessed by the question, "Are you troubled by shortness of breath?"	204
4.52.01	Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures (including co-pollutant models) and the usual presence of shortness of breath as assessed by the question, "Are you troubled by shortness of breath?"	205
4.53	Differences in mean Forced Expiratory Volume in 1 second (FEV ₁ ; Liters) for participants with and without current respiratory symptoms (N = 52)	206
4.54	Differences in mean Peak Expiratory Flow (PEF; L/minute) for participants with and without current respiratory symptoms (N = 52)	207
4.55	Differences in geometric mean C-reactive protein (CRP; mg/L on dried blood scale) for participants with and without current respiratory symptoms (N = 71)	208

CHAPTER 1

HYPOTHESIS/SPECIFIC AIMS

Introduction

More than half of the world's population still relies on biomass combustion to meet basic domestic energy needs (Rehfuess et al. 2006). Cooking in many developing countries usually consists of burning solid fuels over an open fire or in a poorly functioning traditional stove (Rehfuess et al. 2006). Improved stoves have been designed to burn fuel more efficiently and have usually incorporated a chimney or flue. These new designs have the potential to significantly reduce pollutant emissions and indoor air pollution exposures (Albalak et al. 2001; Bruce et al. 2004; Ezzati and Kammen 2002; Khushk et al. 2005; Naeher et al. 2000a; Naeher et al. 2000b; Smith 2002); however, evaluations of improved stoves are limited (Saksena and Smith 2003; Smith 2002). Biomass-derived indoor air pollution in developing countries has often been associated with increased risks of respiratory diseases (Bruce et al. 2000; Bruce et al. 2002; Budds et al. 2001; Ezzati and Kammen 2002; Naeher et al. 2005; Schei et al. 2004; Smith 2002). Although consistent associations between acute lower respiratory infections in children and chronic obstructive pulmonary disease in women have been demonstrated (Smith et al. 2004), limited research has been performed on the association between indoor air pollution and cardiovascular disease in developing countries. Studies examining the relationship between stove use and adverse health effects have been inconsistent, relying mostly on proxies of exposure, such as type of stove or fuel (Bruce et al. 1998; Bruce et al. 2002; Chauhan and Johnston 2003; Smith 2002). The use of these proxies overlooks the large variability within each of these groups (Naeher et al. 2000a) and can result in broad exposure categories that may not correspond with indoor air pollution concentrations. A multilevel stove scale, incorporating quality of stove in addition to type of stove, may provide a more accurate exposure assessment compared to a dichotomous stove type. The utility of a stove scale is that it can be performed at a fraction of the cost and time when compared to quantitative indoor air pollution assessments. Studies are needed to determine how well a stove scale predicts indoor pollutant concentrations. Studies quantitatively assessing both indoor air pollution levels and health effects while adjusting for potential confounders are necessary.

Hypothesis

We conducted a cross-sectional investigation to assess whether or not more easily collected stove quality and kitchen parameters could explain the variation in measured levels of pollutants and to evaluate the association between indoor air pollution exposures and adverse health among women in Honduras. We hypothesized that homes with traditional stoves would have higher concentrations of carbon monoxide and particulate matter (PM), resulting in decreased pulmonary function, increased adverse respiratory symptoms, and increased levels of a biomarker of inflammation (C-reactive protein, CRP) among women in these homes compared with women from homes with improved stoves.

<u>Specific Aims</u>

- 1. Quantify indoor air pollution exposures in homes with traditional and improved stoves and identify factors contributing to elevated indoor air pollution levels.
 - a. Characterize indoor air pollution in homes with and without improved stoves by measuring continuous carbon monoxide and time-weighted average fine particulate matter ($PM_{2.5}$) concentrations over one 8-hr time period.
 - b. Evaluate the contribution of domestic factors (fuel type, cooking practices, and housing conditions) to measured indoor air pollution concentrations.
 - c. Develop a four-level subjective stove scale representing potential for indoor emissions, ranging from poorly functioning traditional stoves to well-functioning improved stoves and assess how well the scale predicts quantitative air quality measurements.
- 2. Characterize respiratory health among women in homes with and without improved stoves.
 - a. Perform lung function measurements (forced expiratory volume in one second, FEV₁ and peak expiratory flow, PEF) and ascertain respiratory symptom prevalence among participating women.
 - b. Validate CRP concentrations in dried blood spots by assessing the correlation with CRP concentrations in whole blood samples.

3

- 3. Evaluate the association between indoor air pollution exposures and respiratory health effects.
 - a. Determine the association of respiratory symptoms with exposure to elevated carbon monoxide and $PM_{2.5}$ concentrations, with the use of traditional versus improved stoves, as well as with the four-level stove scale.
 - b. Determine the association of impaired lung function with exposure to elevated carbon monoxide and $PM_{2.5}$ concentrations, with the use of traditional versus improved stoves, as well as with the four-level stove scale.
 - c. Determine the association of a biomarker of inflammation, CRP measured in dried blood spots, with elevated carbon monoxide and $PM_{2.5}$ concentrations, with the use of traditional versus improved stoves, as well as with the four-level stove scale.
 - d. Assess the effect of outdoor air pollution on the relationship between indoor carbon monoxide and $PM_{2.5}$ concentrations and respiratory health.

CHAPTER 2

BACKGROUND AND SIGNIFICANCE

Health Effects

Many studies have been and are currently being performed on the health effects of ambient air pollution in urban areas of developed countries (e.g., Brunekreef and Forsberg 2005; Brunekreef and Holgate 2002; Glinianaia et al. 2004; Nyberg and Pershagen 2000; Routledge and Ayres 2005; Schwela 2000; Wilson and Spengler 1996). However, these results are applicable to a relatively small range of particulate matter exposures; mainly those less than 200 μ g/m³ (Ezzati and Kammen 2001a; WHO 1998). The relationships between exposures and health effects at levels more typically found in developing countries, concentrations up to thousands of μ g/m³, are relatively unknown (Ezzati and Kammen 2001a). Around 80% of the total global exposure to particulate matter occurs indoors in developing countries (Smith 1988; Smith 1993); therefore, the exposure-response relationship needs to be studied at levels typical of these locations (Ezzati and Kammen 2001a).

Elevated indoor air pollution exposures associated with the burning of biomass fuels are well established. The burden of this pollution is felt most by the world's poorest and most vulnerable people, primarily women and children (Boy et al. 2000; Smith 2002). The burning of biomass accounts for over 50% of domestic energy in many developing countries and up to 95% in rural areas (Ezzati and Kammen 2002; Rehfuess et al. 2006).

Cooking usually consists of burning solid fuels over an open fire or in a poorly functioning stove (a stove that burns fuel inefficiently). Consequently, this leads to high levels of indoor air pollution, a complex mix of pollutants including carbon monoxide, PM, sulfur oxides (particularly with coal), nitrous oxides, formaldehyde, and various carcinogenic combustion by-products (Bruce et al. 2002; Zhang and Smith 1999). These pollutants are released directly into the living area where women and children spend the majority of time. Studies in developing countries consistently reveal high indoor air pollution concentrations, and exposures to these extreme concentrations typically last between 3 and 7 hours each day over many years (Budds et al. 2001; Engel et al. 1997). Current World Health Organization (WHO) guidelines, as defined in the Global Update for 2005, are 25 μ g/m³ (24-hr mean) for PM_{2.5} (particles with an aerodynamic diameter of less than or equal to 2.5 µm) and 50 µg/m³ (24-hr mean) for PM₁₀ (particles with an aerodynamic diameter of less than or equal to 10 µm) (WHO 2005). Typical PM₁₀ concentrations in homes using biofuels may range from 200 to 5,000 µg/m³ (Ezzati and Kammen 2002).

Indoor air pollution from biomass and coal smoke is responsible for approximately 1.6 million premature deaths per year worldwide, representing about 3% of the global disease burden (Smith et al. 2004). Acute respiratory infections, chronic obstructive pulmonary disease (COPD), chronic bronchitis, cancer of the lung, mouth, nasopharynx, or larynx, asthma, tuberculosis, cataract, low birth weight, and infant mortality have been associated with biomass-derived indoor air pollution in developing countries; however, evidence is extremely limited or inconsistent for most of these health endpoints (Bruce et al. 2000;

Bruce et al. 2002; Budds et al. 2001; Ezzati and Kammen 2002; Smith 2002). While cardiovascular disease has been linked to tobacco smoke and outdoor air pollution, there have been only two studies, to our knowledge, on the effects of biomass smoke exposures in developing countries on the risk of heart disease (McCracken et al. 2007; Ray et al. 2006; Smith et al. 2004).

Respiratory disease & symptoms: Several studies have examined symptoms of chronic adverse respiratory health among women cooking with biomass fuel. Eleven such studies which incorporated specific outcome definitions and controlled for smoking and age have been included in a meta-analysis (Smith et al. 2004). Outcome assessments included chronic bronchitis, defined as cough and sputum on most days for at least three consecutive months of two successive years, and COPD, defined as FEV₁/FVC (forced vital capacity) less than 70% or FEV₁ less than 70% of predicted value (Smith et al. 2004). Of the studies included in the analysis, one was a cohort study (Dutt et al. 1996), three were case-control studies (Dennis et al. 1996; Dossing et al. 1994; Perez-Padilla et al. 1996), and seven were cross-sectional studies (Albalak et al. 1999; Behera et al. 1991; Gupta and Mathur 1997; Malik 1985; Menezes et al. 1994; Pandey 1984; Qureshi 1994). The three case-control studies were hospital-based with control groups consisting of visitors to patients other than the study subject (Dossing et al. 1994), patients with illnesses not of the respiratory tract (Dennis et al. 1996), and a mix of visitors and tuberculosis, interstitial lung disease, and otolaryngological patients (Perez-Padilla et al. 1996). The use of visitors to the hospital as controls could have led to a bias if the likelihood for visiting the hospital was related to exposure to indoor smoke (Smith et al. 2004). The use of hospital patients as controls also could have biased the results if exposure to indoor smoke made the patients more or less likely to come to the hospital (Smith et al. 2004). The meta-analysis included only studies that had some control measure for smoking and age and, although some of the studies included men, the meta-analysis used only results applicable to women (Smith et al. 2004). Women exposed to indoor air pollution from the burning of biomass fuel were 3.2 (95% confidence interval (CI): 2.3, 4.8) times as likely to develop COPD or chronic bronchitis as compared to women not exposed to indoor air pollution (Smith et al. 2004). A limitation of these studies is that all used proxies, such as the use of biofuel for cooking, cooking inside versus outside, and time spent near the fire, to evaluate exposure to indoor air pollution. This exposure misclassification is likely non-differential which may lead to a bias toward the null association.

In one of the few studies to directly assess indoor air pollution levels, investigators assessed exposure to biomass smoke by measuring kitchen PM_{10} concentrations and ascertained symptoms via questionnaire among indigenous women and children living in two rural communities in Chiapas, Mexico (Riojas-Rodriguez et al. 2001). An improved stove model, the Ceta, had been introduced to some members of the community five years prior to the study as an alternative to the traditional open or three-stone fire. Among women, factors such as sleeping where they cook, kilograms of wood used per day, age, and somebody smoking in the house were associated with symptoms (common cold, difficulty in breathing, and headache) in multivariate models adjusted for age (Riojas-Rodriguez et al. 2001). The use of the Ceta stove and the PM_{10} concentrations in

the kitchens were not associated with these outcomes, although the use of the Ceta stove was suggestive of an increase in difficulty in breathing (RR = 1.63, 95% CI: 0.70, 3.70) (Riojas-Rodriguez et al. 2001). The authors noted that the deterioration of the Ceta stoves over the five year period, as well as the small sample size (42 women), may have limited the ability to detect differences among these groups (Riojas-Rodriguez et al. 2001).

Lung function: Spirometry has often been utilized to evaluate airflow obstruction, chronic obstructive pulmonary disease, and asthma in adults and children (Bruce et al. 2000). FEV₁ and PEF are both obtained from the forced expiratory maneuver. PEF is the maximum flow achieved during an expiration performed with maximal force starting from the level of maximum lung inflation (Quanjer et al. 1997) and FEV₁ is the volume of air exhaled during the first second of the maneuver. Reductions in both measures have been associated with lung abnormalities. FEV₁ reflects the caliber of both large and small airways, while PEF is more a reflection of the caliber of the large airways (Leroyer et al. 1998). FEV₁ is often considered the index of choice (Bellia et al. 2003) because it is reproducible; has a strong prognostic value in important diseases, especially moderate to severe COPD (Siafakas et al. 1995; Thomason and Strachan 2000); and is recognized as a measure of global health, predicting all-cause mortality (Hole et al. 1996), even over long periods of time (Schunemann et al. 2000)

Epidemiologic evidence exists for the relationship between lung function and ambient particulate air pollution. Cross-sectional studies of community air pollution levels and individual lung function data from national surveys or collected cohorts have indicated small but often significant associations between particulate matter and declines in various measures of lung function (Ackermann-Liebrich et al. 1997; Chestnut et al. 1991; Gauderman et al. 2004; Naeher et al. 1999; Raizenne et al. 1996; Schwartz 1989; Tashkin et al. 1994; Wang et al. 1999). In developing countries, associations between indoor air pollution and decreased FEV₁, FVC (forced vital capacity), and PEF have been observed (Behera et al. 1994; Dutt et al. 1996; Ekici et al. 2005; Gharaibeh 1996; Guneser et al. 1994; Kiraz et al. 2003; Malik 1985; Pandey et al. 1985; Regalado et al. 1996; Saha et al. 2005), although inconsistently (Reddy et al. 2004). However, the evidence may be unreliable since these studies used proxies of exposure and often did not consider additional factors possibly related to the outcome (Bruce et al. 2000).

Norboo and colleagues conducted a study in a Himalayan village where they quantitatively measured carbon monoxide in the kitchen, exhaled carbon monoxide in the breath of participants, and lung function (FEV₁, FVC, and the ratio of the two) during the winter and summer seasons (Norboo et al. 1991). Indoor fires burning wood and dried yak dung were used to cook and heat the homes. Exposures to pollutants were expected to be higher during the winter months when families spend long periods of time in poorly ventilated homes (Norboo et al. 1991). The authors used linear regression to test the associations between carbon monoxide and lung function while adjusting for age and height and stratifying by gender. Among non-smoking women, FEV₁ was inversely associated with exhaled carbon monoxide, although non-significantly (Norboo et al. 1991). The authors also examined individual changes in lung function and pollution

levels from summer to winter. Among non-smoking men and women, the change in the level of exhaled carbon monoxide from summer to winter was positively associated (p < 0.01) with the individual decrease in FEV₁ (a 0.23 liter decrease among women with a greater than 10 ppm increase in exhaled carbon monoxide), but not with forced vital capacity (Norboo et al. 1991). This may suggest that the pollution has a relatively acute affect on the lungs (Norboo et al. 1991). However, the authors also reported that the decreases in FEV₁ were greater among those with chronic symptoms as assessed via questionnaire (Norboo et al. 1991). The relationship between increases in carbon monoxide and decreases in FEV₁ is suggestive of an association between indoor pollution from domestic fires and adverse respiratory effects. However, the decrease in FEV₁ over a six month period is not necessarily indicative of chronic obstructive disease. Rather, those who already have disease may be more susceptible to the irritant effects of the pollution (Norboo et al. 1991).

Acute lower respiratory infections: Smith and colleagues. (Smith et al. 2004) performed a meta-analysis of 8 studies examining the risk of acute lower respiratory infections (ALRI) associated with the use of solid fuels in children aged less than 5 years (Campbell et al. 1989; Collings et al. 1990; de Francisco et al. 1993; Johnson and Aderele 1992; Morris et al. 1990; O'Dempsey et al. 1996; Pandey et al. 1989; Robin et al. 1996). The majority of these studies occurred in developing countries; however, two were carried out in populations of Navajo and Hopi Indians in the United States (Morris et al. 1990; Robin et al. 1996). These studies among Native American populations show a significant association between ALRI and use of woodstoves with indoor pollutant levels much lower than those typically found in developing countries (Smith et al. 2004). Although the US populations are likely to differ with respect to socio-economic status when compared to the developing country populations, the results of the meta-analysis did not change with the inclusion or exclusion of these studies (Smith et al. 2004). The overall risk of ALRI due to indoor air pollution caused by the use of solid fuels was 2.3 (95% CI: 1.9, 2.7) (Smith et al. 2004). None of these studies used direct measures of indoor air pollutants to assess exposure. Rather, proxies such as use of solid fuel, duration of time child spent near the cooking fire, and whether or not the child was carried on the mother's back were used (Smith et al. 2004). Quantitative exposure measurements were performed in a study of Navajo children living in Arizona (Robin et al. 1996). Risk of ALRI was increased among children living in households that cooked with any wood, as well as households with indoor air PM_{10} concentrations greater than or equal to 65 μ g/m³ (Robin et al. 1996). More recently, Ezzati and Kammen performed a study in Kenya of acute respiratory infections (ARI) in adults and children in which they assessed exposure in households using a variety of fuel types by taking real-time measurements of PM_{10} (Ezzati and Kammen 2001a). Personal exposure levels were estimated by utilizing timeactivity patterns as reported by the participants. Elevated PM₁₀ levels were associated with ARI and ALRI in both children and adults (Ezzati and Kammen 2001a).

Lung cancer: The majority of studies examining the association between lung cancer and indoor air pollution have been among women cooking with open coal stoves in China (Smith et al. 2004). A meta-analysis among studies adjusting for smoking and chronic respiratory disease indicated a 1.94-fold increased lung cancer risk among women cooking with coal (95%CI: 1.09, 3.47) (Smith et al. 2004). Limited evidence exists on the association between lung cancer in women and use of biomass fuels (Gao et al. 1987; Liu et al. 1993; Sobue 1990).

Perinatal effects: Few studies have examined the risk of adverse pregnancy outcomes due to cooking with biomass fuels. Although the consistency and strength of association differs between outcomes, outdoor air pollution (mainly in developed countries), at concentrations much lower than those typically found in homes using biomass fuels for cooking, has been associated with intrauterine mortality, low birth weight, prematurity, and early infant death (Bobak 2000; Glinianaia et al. 2004; Loomis et al. 1999; Maisonet et al. 2004; Pereira et al. 1998; Perera et al. 1999; Ritz and Yu 1999; Sram et al. 2005; Woodruff et al. 1997). In India, exposure to cooking smoke during pregnancy was associated with an increased risk of stillbirth (adjusted odds ratio (OR) = 1.5; 95% CI: 1.0, 2.1) (Mavalankar et al. 1991). More recently, Mishra and colleagues reported that women cooking with biomass fuels were more likely to experience a stillbirth than those cooking with electricity, liquid petroleum gas, biogas, or kerosene (adjusted OR = 1.44; 95% CI: 1.04, 1.97) (Mishra et al. 2005). In Guatemala, children born to mothers cooking primarily with wood fuel had mean birth weights of 63 g less than children born to mothers cooking primarily with gas or electricity (adjusting for parity, mother's age, floor material, area of residence, third trimester vitamins, and maternal calf circumference) (p = 0.05) (Boy et al. 2002). Similarly, among 1734 births in Zimbabwe, children born to mothers cooking with wood, dung, or straw were 175 g lighter than children born to mothers cooking with liquid petroleum gas, natural gas, or electricity (adjusted for sex of child, birth order of child, mother's age, mother's body mass index,
iron supplement during pregnancy, malaria drug during pregnancy, mother's education, mother's religion, standard of living index, and geographic region) (p< 0.01) (Mishra et al. 2004). No studies of perinatal effects have examined the potential benefits of improved wood-burning stoves as compared to traditional wood-burning stoves. The relationship between exposures to biomass cooking smoke and adverse pregnancy needs further attention using more direct measures of indoor air pollution (Mishra et al. 2005).

Developed countries: Adverse effects of wood smoke are also found in developed countries (Naeher et al. 2005). In the U.S., the use of woodstoves was associated with a 30% increase in respiratory symptoms and children in those homes were more likely to suffer physician confirmed acute respiratory infections (Dockery et al. 1989; Morris et al. 1990). Woodstove use has also been associated with adverse health effects in Native American children (Morris et al. 1990; Robin et al. 1996). Firefighters also experience acute health effects, including decreased lung function, from high exposures to air pollutants; often during periods when respiratory protection is not worn (Brandt-Rauf et al. 1989; Burgess et al. 2001; Large et al. 1990; Musk et al. 1979; Sherman et al. 1989). Chronic health effects may also occur; however, the increased use of protective equipment appears to have had a beneficial effect (Burgess et al. 2001). Health effects results from smoke exposures occurring in developing countries can apply to certain developed-country populations with similarly high levels of exposures and also may be important in identifying associations for further study in populations experiencing exposures on smaller scales.

<u>Mechanisms of Action</u>

Smoke from the burning of biomass fuels contains a complex mix of pollutants (Naeher et al. 2007; Smith et al. 2000). The magnitude and type of emissions from these mixtures can vary greatly based on factors such as materials burned, stove type, combustion conditions, and time since generation (Budds et al. 2001; Smith 1987; Smith et al. 2000). Because of this variation, specific statements or conclusions concerning the potentially health-damaging properties of smoke are difficult. The limited work to identify the physical and chemical characteristics of smoke has been performed on wood burning metal stoves in developed countries (Larson and Koenig 1994). Therefore, arguments linking cookstove smoke and health effects in the developing countries are mostly based on generalizations and are not mixture specific (Budds et al. 2001). The main healthrelated components of wood smoke are carbon monoxide, particulate matter, nitrogen oxides, and hydrocarbons (Budds et al. 2001; Smith et al. 2000). In addition, several organic compounds considered to be toxic or carcinogenic, such as formaldehyde, benzene, and polyaromatic hydrocarbons, have been identified in the aerosol portion of the mixture (Smith et al. 2000). Unlike coal, biomass fuels contain relatively small amounts of sulfur, trace metals, and ash (Smith et al. 2000). While correlations between carbon monoxide and $PM_{2.5}$ have been demonstrated in homes where open fires and some improved stoves were used, the correlations decreased substantially in homes where concentrations were low as compared to open fire conditions (Naeher et al. 2001). Although biomass can burn cleanly under ideal conditions, complete combustion is rarely achieved with the inexpensive stoves commonly found in developing countries (Naeher et al. 2007; Smith et al. 2000). In general terms, there are two ways that indoor air pollution can affect health. The pollutants in the smoke can be directly responsible for the adverse health endpoint, e.g. carcinogens, or the pollutants can cause damage to the respiratory system's mechanical and immune defenses leaving the individual more vulnerable to infection by bacteria or virus (Budds et al. 2001).

Particulate matter: Although recent emphasis has been placed on the health effects of coarse particulate matter (Brunekreef and Forsberg 2005), health effects due to fine particulates are well established in the air pollution literature. Evidence suggests that particles, generally of the smaller sizes, may be the most damaging to health (Donaldson 2003; Donaldson and MacNee 2001; Smith 1987). Particles in smoke are a combination of solids and liquids of varying size and composition (Budds et al. 2001). The size of the particle is important in determining deposition within the lungs and smaller particles are able to penetrate more deeply into the respiratory system (Budds et al. 2001). Generally, respirable particulate matter is divided into two distinct size fractions (Suh et al. 2000). Coarse particles (PM_{2.5-10}, particles having an aerodynamic diameter between 2.5 and 10 μ m) can reach the upper airways and larger lower airways while fine particles (PM_{2.5}) are able to penetrate the small airways of the bronchioles and alveoli (Budds et al. 2001). The two size fractions have different origins and compositions. $PM_{2.5}$ is generated primarily from combustion sources, such as automobiles, power plants, and wood stoves, either through the volatilization of primary particulate matter or from precursor gases reacting in the atmosphere to form secondary particles (Suh et al. 2000). Coarse particles are formed primarily by mechanical crushing, grinding, or abrasion of surfaces and are suspended and dispersed by wind and human activity, such as traffic and agriculture (Suh et al. 2000). Due to primary combustion and no evident indoor source of coarse particulate matter, $PM_{2.5}$ may be a more relevant exposure to assess when examining the health effects of wood-burning stoves (Naeher et al. 2007).

The biologic mechanisms linking inhaled particulate matter to cardiovascular adverse health conditions are unclear but could involve either the direct effect of the pollutants on the tissues or the indirect effect of the pollutants which are mediated through pulmonary oxidative stress and inflammatory responses (Bai et al. 2007; Brook et al. 2004). The direct mechanism may occur when ultrafine particulate matter crosses from the pulmonary epithelium into the circulation. This may lead to changes in autonomic tone which could contribute to the instability of vascular plaque or initiate cardiac arrhythmias. Direct effects may explain the occurrence of acute (within a few hours) cardiovascular responses to elevated pollution exposures (Brook et al. 2004). The indirect mechanism may be responsible for the less acute (several hours to days) and chronic health effects of exposures to elevated pollutants. This pathway contributes to systemic inflammation which may activate hemostatic pathways, impair vascular function, and accelerate atherosclerosis (Brook et al. 2004). The direct and indirect mechanisms are presumable not independent and may interact to result in adverse health conditions (Bai et al. 2007). General mechanisms are discussed below.

The mucociliary escalator is an important defense mechanism against inhaled particles. In the large airways, goblet cells secrete mucus which traps deposited particles. Ciliated cells then propel the mucus upward to be expectorated or swallowed (MacNee and Donaldson 1999). Increased exposures to particles may result in increased mucus secretion (MacNee and Donaldson 1999). Mucus hypersecretion for most days over three consecutive months, for two years or more is the defining feature of chronic bronchitis (American Thoracic Society 2005; Medical Research Council 1965). Chronic bronchitis is a part of the spectrum of chronic airways disease termed chronic obstructive pulmonary disease, which also includes emphysema, and some cases of chronic asthma (Celli et al. 1995). In COPD patients, goblet cells extend into the bronchioles (Jeffery 1998). Thus, even though mucus hypersecretion generally has a protective role, excessive mucus hypersecretion, especially in the smaller airways, results in mucus plugging, a feature commonly present in patients dying of COPD and asthma (Lamb 1995). Excessive mucus hypersecretion, in addition to damage to the cilia, overwhelms the mucociliary escalator and reduces the ability of the lungs to deal adequately with inhaled particles (MacNee and Donaldson 1999). Another defense mechanism in the airways is the release of inflammatory mediators upon exposure of epithelial cells to particles. In addition, macrophages are present which can phagocytose particles also resulting in the release of inflammatory mediators. In airway disease patients, increased levels of macrophages and inflammatory cells can be measured and it is thought that the additional insult of inhaled air pollutants could exacerbate this background inflammation (MacNee and Donaldson 1999).

Inhaled particles also deposit beyond the ciliated airways, into the terminal airways and proximal alveoli (Brody et al. 1984). Here, macrophages play the most important role in removing particles by phagocytosis and migration to the mucociliary escalator. Although

some adverse health effects of particulates are focused on the larger airways, inflammatory events in the alveolar region are important in the potential systemic effects of inhaled particulates (MacNee and Donaldson 1999). It is in this region that the mediators released as a result of the local inflammatory effects of particles in the airspace are more readily transmitted to the blood (Donaldson and Tran 2002; MacNee and Donaldson 1999). The resulting sequestration and mobilization of leukocytes into the region, which is associated with evidence of oxidative stress (Rahman et al. 1996), may potentially lead to lung injury and systemic cardiovascular effects (Donaldson 2003; MacNee and Donaldson 1999). A potential mechanism is as follows: airway injury or activation of blood cells caused by particles in the alveoli leads to a release of proinflammatory cytokines, interleukin (IL)-6 and IL-8; increases in IL-6 and IL-8 activate mononuclear and endothelial cells, which initiate the hepatic synthesis of acutephase proteins, such as CRP (Peters et al. 2001b), and an upregulation of the expression of adhesion molecules as markers of endothelial dysfunction; the enhanced acute-phase response as well as endothelial cell activation leads to increased procoagulation activities (Seaton et al. 1995); and these changes, along with plaque instability, may result in increased risks of coronary events (Ruckerl et al. 2006). In addition to the cardiovascular response, evidence from human and animal studies suggests that the altered alveolar macrophage response following exposures to nitrogen dioxide and possibly particulate matter may lead to a decreased oxidant response, increasing the risks of bacterial and viral infections (Budds et al. 2001; Chauhan and Johnston 2003).

19

C-reactive protein: As mentioned above, the inhalation of fine particles has been linked with acute pulmonary inflammation and oxidative stress (Brook et al. 2003). The resulting stimulation of a systemic inflammatory response could link air pollution exposures to the development of cardiovascular disease. CRP is a tightly regulated acute phase protein produced in the liver in response to injury, infection, or other inflammatory stimuli (Donaldson et al. 2001). During an acute response, CRP is rapidly upregulated, usually within hours. It has a half-life of approximately 13-19 hours and is considered a sensitive, robust, and uniquely quantitative marker of the acute phase response (Peters et al. 2001b). The recent development of high-sensitivity CRP assays (Rifai et al. 1999; Roberts et al. 2000; Wilkins et al. 1998) has led to the discovery that slight elevations in CRP, previously thought to be in the range of normal variation, may indicate low-grade inflammatory processes related to the development of cardiovascular disease (McDade et al. 2004). Whether or not CRP is a marker of vascular disease or involved directly in the pathogenesis leading to cardiovascular disease is unclear (Bai et al. 2007). The mechanism by which CRP increases cardiovascular disease risk is currently a topic of intense research (Brook et al. 2004). It is possible that CRP impairs endothelial vasoreactivity among diseased individuals. Additionally, CRP may directly influence the development and progression of atherosclerosis through enhanced foam cell formation, recruitment of monocytes into the arterial wall, stimulation of prothrombotic tissue factors, and expression of adhesion molecules (Brook et al. 2004). Because CRP plays a role in the development of atherosclerosis and in plaque instability, it is possible that the inflammatory response due to air pollution exposures could both promote atherosclerosis formation over the long term and also instigate acute plaque instability and sudden cardiovascular events in the short term (Brook et al. 2004). Investigators in the Women's Health Study reported that CRP was a strong predictor of future cardiovascular events among seemingly healthy postmenopausal women (Ridker et al. 1998; Ridker et al. 2000). Studies from developed countries have reported associations between air pollution exposures (PM_{10} , $PM_{2.5}$) and increased CRP levels among healthy men aged 45 to 64 years (Peters et al. 2001b), men and women 54 years and older (Pope et al. 2004; Seaton et al. 1999), and male patients with coronary heart disease (Peters et al. 2001b; Pope et al. 2004; Ruckerl et al. 2006; Seaton et al. 1999), although no clear association was reported among men and women aged 45 to 84 years in the Multi-ethnic Study of Atherosclerosis (Diez Roux et al. 2006).

Carbon monoxide: Carbon monoxide is a colorless, odorless, poisonous gas formed when carbon in fuels is not burned completely. The health effects of carbon monoxide exposures from the burning of biomass fuels seem to be due to forms of hypoxia which result from the reduction of oxygen availability to body tissues (McGrath 2000; Smith 1987). Carbon monoxide is inhaled into the respiratory system and absorbed into the blood where it binds to hemoglobin with about 200-250 times the affinity of oxygen (Maynard and Waller 1999; Rodkey et al. 1974). The amount of carboxyhemoglobin in the blood is a function of the carbon monoxide concentration in the air, the duration of exposure, and the physiologic status of the individual (Smith 1987). Relatively small concentrations of carbon monoxide can lead to significant reductions in the body's ability to transport oxygen to tissues, such as the heart and brain, as well as to the developing fetus, among pregnant women (Hass 1992; Mishra et al. 2005; Smith et al. 2000). Acute

poisoning is caused from high exposures to carbon monoxide; however, these exposures are not encountered in outdoor urban settings (Katsouyanni 2003). The effect of chronic exposures of relatively low levels of carbon monoxide is unclear (Maynard and Waller 1999). A chronic flu-like illness with symptoms such as headache, irritability, and malaise, has been described and attributed to prolonged and repeated exposures to low concentrations of carbon monoxide (Baker et al. 1988; Crispen 1989; Maynard and Waller 1999). Associations between carbon monoxide exposures and cardiac arrhythmia (Peters et al. 2000a), respiratory and cardiovascular emergency department visits and hospital admissions for heart disease (Metzger et al. 2004; Peel et al. 2005; Schwartz 1999), and mortality (Samet et al. 2000b; Touloumi et al. 1996) have been described in a limited number of outdoor air pollution epidemiologic studies (Katsouyanni 2003). In controlled studies of coronary artery disease patients, cardiovascular endpoints, such as shortening of time to onset of angina, were associated with carboxyhemoglobin levels between 2 and 6% (Katsouyanni 2003; Maynard and Waller 1999). In addition to cardiovascular effects, carboxyhemoglobin levels were correlated with decrements in lung function (FEV_1) among structural firefighters (Burgess et al. 2001). Levels of carboxyhemoglobin resulting from indoor air pollution exposures from cookstove smoke are thought to be high enough to result in adverse health effects (Bruce et al. 2002). Average daily levels of carbon monoxide in homes using biofuels have ranged from 5 to 10 ppm, while concentrations of 20 to 50 ppm or more have been reported during use of the fire (Dary et al. 1981; Norboo et al. 1991); and associated carboxyhemoglobin levels have reached 1.5-2.5% (Dary et al. 1981) to as high as 13% (Behera et al. 1988). No threshold of effects for percent carboxyhemoglobin has been defined; however, effects

have been reported at 2%, the lowest concentration of carboxyhemoglobin studied (Maynard and Waller 1999).

Summary: Several mechanisms by which air pollutants may increase risk of respiratory and cardiovascular health problems have been identified. Biological mechanisms include bronchial irritation, inflammation and increased reactivity (acute exposures); reduced mucociliary clearance; reduced macrophage response; and autonomic imbalance, procoagulant activity, and oxidative stress (Bruce et al. 2002). The potential health effects associated with these processes include wheezing and exacerbation of asthma; respiratory infections; chronic bronchitis and COPD; exacerbation of COPD; and excess mortality (which includes cardiovascular disease) (Bruce et al. 2002). Knowledge of these underlying biological mechanisms of the association between air pollution and human health is somewhat limited (Pope 2000). However, biologic plausibility is enhanced by the repeated observations of cardiopulmonary health effects and the fact that associations with non-cardiopulmonary health end points and air pollution are not generally identified in the literature (Pope 2000). In addition, more recent studies have examined specific endpoints, such as lung function and biomarkers of inflammation, which may be a part of the mechanistic pathway between exposure and cardiopulmonary mortality (Pope 2000).

Improved Stoves & Exposure Assessment

Although the use of cleaner fuels tends to occur naturally with development, the economic state of the world implies that a significant proportion of the world's population will continue to use biomass fuels for many decades (Albalak et al. 2001;

Smith 1993). Therefore, it is critical to find economically feasible and cultural acceptable alternatives to the traditional stove. Many programs have attempted to do this by introducing improved stoves with reduced emissions based on the addition of a flue or chimney or the design of a more efficient combustion chamber (Albalak et al. 2001). However, only a few studies in developing countries have objectively shown that improved stoves can significantly reduce pollutant emissions. A study of 30 households in rural Guatemala found that using improved cookstoves resulted in an 85% reduction in PM_{3.5} concentrations compared with levels obtained when cooking over open fires (Albalak et al. 2001). Another found that well-operated improved stoves resulted in 15-30% reductions of PM₁₀ and 10-20% reductions of PM_{2.5} compared with open fires (Naeher et al. 2000a). A study in Kenya estimated that interventions could reduce PM_{10} exposures by 35-95%, leading to a 24-64% reduction in acute respiratory infections in children (Ezzati and Kammen 2002). However, the difference in the mean PM_{10} concentrations in the kitchens of households with and without improved stoves was not significant ($80 \mu g/m^3$) in a study of Ceta stoves in Mexico (Riojas-Rodriguez et al. 2001). While most of these studies have examined air quality differences soon after the introduction of improved stoves, the study in Mexico was performed five years after introduction. Differences in the above-mentioned studies underline the need to examine all types of improved stoves as designs vary across geographic and cultural regions. It is also important to evaluate improved stoves at different times during the life of the stove as factors such as environmental conditions, building materials, and maintenance may influence emissions.

Research on the health effects of indoor air pollution from the burning of biomass is limited by a lack of quantitative exposure assessments (Ezzati and Kammen 2001a). Many investigators rely on indirect measures of personal exposure, such as stove type or fuel. Because the use of biomass for cooking and heating is so common in rural areas of developing countries, many people are grouped into a single exposure category (Ezzati and Kammen 2001a). In reality, there are large variations in emissions from specific stove types (Ballard-Tremeer and Jawurek 1996; Naeher et al. 2000a), as well as large variations in exposure profiles of individuals residing in the same households (Ezzati et al. 2000; Saksena et al. 1992). These variations are due to different types and qualities of stoves and fuels, housing characteristics (i.e. ventilation), cooking and heating methods, differences in time-activity patterns, and season (Balakrishnan et al. 2002; Smith et al. 2004). Indirect exposure assessments and groupings of people most likely lead to risk estimates that are poorly quantified and subject to bias (Bruce et al. 2002; Ezzati and Kammen 2001a). Therefore, further study utilizing quantitative indoor and personal exposure assessments are needed if accurate dose-response relationships are to be identified.

Detailed quantitative assessments of indoor pollutants may also provide useful information for larger-scale epidemiologic studies of indoor air pollution and adverse health. Housing and stove conditions may greatly influence concentrations of pollutants (Bruce et al. 2004; Bruce et al. 2002; Dasgupta et al. 2004; Desai et al. 2004; Riojas-Rodriguez et al. 2001; Smith 2002). Comprehensive exposure assessments and the collection of detailed housing conditions can be used to build statistical models that can

25

identify important factors that influence or predict exposures. Investigators of one study in Mexico reported that the use or non-use of the improved stove, the number of windows in the kitchen, and the amount of firewood used best explained the variation in PM_{10} concentrations measured at an average distance of 3.25 meters from the stove; however, these variables accounted for only a quarter of the variation in PM_{10} (adjusted $R^2 = 0.26$) (Riojas-Rodriguez et al. 2001). The investigators were not able to identify variables that explained the particulate concentrations at an average distance of 1.12 meters from the stove. This study was limited by the use of a relatively poorly functioning improved stove that did not significantly differ from the traditional stove in particulate emissions and also by a small sample size (N = 33) (Riojas-Rodriguez et al. 2001). A larger sample size and the use of stoves emitting a larger range of pollutants may result in models accounting for a higher percentage of the variation (Riojas-Rodriguez et al. 2001). Fuel type, kitchen type, and proximity to the stove during cooking were associated with concentrations of respirable particulate matter in India (Balakrishnan et al. 2002). The investigators stressed the need for further assessment of factors such as room/window dimensions, fuel quantity, and ventilation levels to allow for a better evaluation of the most important determinants of indoor air pollution exposures in households (Balakrishnan et al. 2002). Although quantitative exposure assessments are ideal, larger studies will be more feasible if investigators can confidently utilize the more easily collected housing and stove condition variables in order to estimate exposure levels.

<u>Summary</u>

Despite past achievements, more research is needed to identify methods for reducing indoor air pollution exposures and the associated disease burden. One need is to directly quantify exposures, incorporating housing conditions such as ventilation, rather than solely relying on proxies such as fuel type, stove type, or time spent cooking (Bruce et al. 2004; Bruce et al. 2002; Dasgupta et al. 2004; Desai et al. 2004; Smith 2002). Past investigations have also failed to appropriately account for additional factors related to exposures and health outcomes (Bruce et al. 2004; Bruce et al. 1998; Bruce et al. 2002). These methodological shortcomings limit interpretations of the existing evidence (Bruce et al. 2002; Smith 2002). Practical, vigorous, and valid methods for measuring exposures and related outcomes are needed not only for health risk investigations but also for evaluating interventions (Bruce et al. 2002; Smith-Sivertsen et al. 2004). This could lead to a better understanding of disease mechanisms, better control measures, and improved disease prevention strategies (Chauhan and Johnston 2003).

In summary, extraordinarily high indoor air pollution exposures associated with biomass combustion have been documented in developing countries. Intervention studies among households with and without improved stoves are greatly needed (Saksena and Smith 2003). We incorporated quantitative indoor air pollution exposure assessment, biomonitoring, and pulmonary function testing to evaluate exposures and health effects associated with improved stoves in Honduras. We evaluated whether indoor air pollution reductions due to wood-burning stove use and function was associated with the occurrence of adverse health effects. In addition, we evaluated a field method for collecting dried blood spot samples via finger-stick and a laboratory method for analyzing CRP in dried blood spots; and examined this novel marker of inflammation in relation to indoor air pollution levels. The ease and convenience of this field method, utilizing finger-stick blood samples, could prove to be a useful tool for community-based, epidemiologic investigations (McDade et al. 2004; Mei et al. 2001; Parker and Cubitt 1999). Final results of this study may provide further insight regarding the relationship between indoor air pollution and health effects, as well as provide a foundation for the development of longitudinal community intervention trials that would more rigorously evaluate this issue in a larger study population.

CHAPTER 3

RESEARCH DESIGN AND METHODS

Study Population

A local not-for-profit organization, Trees Water & People (TWP, Fort Collins, Colorado) is spearheading efforts in Honduras to disseminate improved stoves that reduce biofuel usage and unhealthy smoke exposures. TWP is working in collaboration with the Honduran Association for Development (AHDESA, Tegucigalpa, Honduras) to achieve this goal. The improved Justa stoves were designed with a chimney and a combustion chamber that increases fuel efficiency while maintaining a culturally acceptable design. We have partnered with TWP and AHDESA to evaluate health conditions and determine the effectiveness of their stove distribution program. The field stage of this project began in June 2005 and was completed in August 2005. Fifty-nine nonsmoking participants were selected from two communities, 30 from Santa Lucia and 29 from Suyapa, by AHDESA staff. Recruitment was planned so that approximately half of the women used improved stoves and half used traditional stoves. Suyapa is a suburb of the capital city and is considered semi-urban. Santa Lucia is located in the mountains approximately 15 miles from the capital and is considered semi-rural. These two communities were chosen in order to examine the effect, if any, that outdoor air pollution has on indoor air pollution and the health of the women living in these locations. Women were non-randomly selected by AHDESA from those receiving improved stoves in the past, those on a list to receive stoves in the future, and by recommendations from women already participating. To increase the power of the study to detect differences in health outcomes related to the use of different stoves, an additional 20 women from Santa Lucia (10 with traditional stoves and 10 with improved stoves) were recruited to participate in an abbreviated study protocol that did not include the air quality measurements.

Data collection for the original 59 participants occurred over one eight-hour period. We began exposure monitoring in the morning by setting up indoor and outdoor air quality meters. A survey was completed by field investigators to obtain information on housing and stove conditions. Health endpoint assessments began immediately following the eight-hour exposure monitoring period, and included lung function testing and the collection of a finger-stick blood sample in order to assess CRP levels. A Spanish-language questionnaire was also administered at this time to assess information such as the presence of respiratory symptoms and cooking practices. The additional 20 women participated in all aspects of the study with the exception of the eight-hour air quality monitoring.

In order to validate the CRP laboratory method in the dried blood spots, a validation substudy was performed on 40 volunteers. Volunteers were recruited from the Department of Health & Exercise Science at Colorado State University (CSU).

Exposure assessment

 $PM_{2.5}$ was assessed via eight-hour indoor and outdoor monitoring, as well as eight-hour personal monitoring. Carbon monoxide was assessed via eight-hour indoor monitoring

and 20-minute outdoor monitoring. Personal $PM_{2.5}$ was assessed by attaching the sampler to the participant's clothing nearest her breathing zone and placing the pump in a pack to be worn around her waist. $PM_{2.5}$ and carbon monoxide indoor sampling devices were collocated inside the kitchen at a height representative of women's breathing zones. $PM_{2.5}$ outdoor sampling devices were placed in locations estimated to be far enough away from the kitchen so as not to be in any direct plume of smoke and at a height representative of women's breathing zones. The carbon monoxide monitor was placed in the outdoor location for the first and last 10 minutes of the data sampling period. These quantitative exposure assessments were performed on the original 60 participants but not the additional 20 participants.

 $PM_{2.5}$ was assessed using PEM samplers (SKC Inc., PA). Sampling was performed using laboratory pre-weighed 37 mm Teflon polytetraflouroethylene filters with 2 µm pore size (SKC Inc, PA). Sampling pump (Universal, SKC Inc., PA) flow rates were set to 2 liters/minute and calibrated in the laboratory with the SKC DryCal DC-Lite (SKC Inc., PA) and in the field before and after sampling events using a rotameter. The rotameter was calibrated in the laboratory with the SKC DryCal DC-Lite. Sampling filters were pre-weighed and post-weighed in the laboratory at CSU using the Mettler MT5 balance (Mettler-Toledo, Inc.). Filters were dessicated for at least 24 hours in a dessicant chamber before weighing. Filter weights were adjusted with blank filters collected daily in the field. These adjusted weights for the personal, indoor, and outdoor filters were then divided by the air volume (the number of minutes used multiplied by the flow rate of 2 liters/minute). Approximate eight-hour time-weighted average $PM_{2.5}$ values were assessed in statistical analyses.

Carbon monoxide was assessed using a Q-TRAK Plus IAQ Monitor (TSI Inc., MN). The Q-TRAK is a direct reading instrument that records and stores carbon monoxide, carbon dioxide, temperature, and relative humidity. Carbon monoxide is measured using a chemical sensor and carbon dioxide is measured using a non-dispersive infra-red detector. Data were logged at one minute intervals and downloaded to a computer upon completion of sampling. The Q-TRAK was pre- and post-calibrated using zero air, 35 ppm carbon monoxide, and 1000 ppm carbon dioxide calibrated gas cylinders (TSI, Inc, MN) and a sling psychrometer (for temperature and relative humidity). Average, maximum, and 1-hr maximum carbon monoxide values were assessed in statistical analyses.

Kitchen volume, building materials, size of eave spaces and windows, and temperature were determined via an investigator housing survey (modified from ITDG – Smoke, Health and Household Energy project; Practical Action, Warwickshire, UK) and air quality sampling to estimate ventilation rates that may affect carbon monoxide and $PM_{2.5}$. Personal cooking practices were assessed via questionnaire. Variables such as time spent cooking were used in conjunction with kitchen air quality measurements to estimate personal exposures. Recall accuracy was high among Guatemalan women asked to recall durations of activities occurring during the previous 24 hours (Engel et al. 1997). Field investigators assessed stove quality using a four-level subjective scale representing

potential for indoor emissions, based on factors such as chimney and plancha (griddle) condition and maintenance. The scale ranged from poorly functioning traditional stoves to well-functioning improved stoves. We assessed how well the stove scale predicted quantitative air quality measurements, as well as examined the association between the stove scale and health endpoints.

Lung function

Lung function was examined by measuring PEF and FEV_1 using the portable PiKo-1 peak flow meter (Pulmonary Data Services, Inc., Colorado). Lung function measurements were performed in the afternoon, following air quality assessment. The participants were asked to sit in an upright position with both feet flat on the ground. They were instructed to inhale completely, place the meter in their mouth, and to exhale with maximal force as soon as their lips were sealed around the mouthpiece, while maintaining an upright position. An investigator demonstrated the maneuver. The meter has an indicator that notified the investigator if a cough was detected, the blow effort was too short, or the blow effort had a slow start. Each subject attempted three acceptable maneuvers and the maximum and average were recorded. The use of these objective endpoints, in addition to CRP, should minimize potential recall or reporting bias that may be more evident when assessing outcomes such as symptoms.

<u>C-reactive protein</u>

CRP was measured in dried blood spot samples. Assays using whole blood collected via a finger stick and dried on filter paper provide a viable alternative to obtaining venous blood and should be less complicated for the participant than obtaining a complete overnight urine void (McDade et al. 2004; Mei et al. 2001; Parker and Cubitt 1999). Several community-based applications have shown this to be a convenient and reliable means to facilitate sample collection, storage, and transportation (Mei et al. 2001; Parker and Cubitt 1999). Dried blood spots are a highly valuable resource for epidemiologic investigations and the utility for assessing environmental exposures particularly among dried blood spots routinely collected among newborns in the United States has been examined (Olshan 2007).

A finger-stick blood sample was collected from each participating woman after personal exposure monitoring. The puncture site was selected and cleansed with 70% isopropanol. Participant's fingers were pricked with a sterile, disposable Tenderlett (ITC, NJ) lancet with a 1.75 mm point. Up to five drops of blood were spotted onto standardized filter paper (903 Protein Saver Card, Schleicher & Schuell, NH). Blood samples were dried overnight at room temperature in a horizontal position and then stored in low gaspermeable zip-closure bags with desiccant packets and humidity indicator cards (Hannon et al. 1997). Samples were frozen in Honduras and transported to CSU. McDade and colleagues (McDade et al. 2004) had validated the assay to measure CRP in dried blood spots. However, several of the assay reagents were not longer available for purchase upon completion of the data collection; therefore, slight changes had to be made to Dr. McDade's original protocol. In order to validate this revised laboratory methodology, we examined how well the dried blood spot CRP levels correlated with venous-drawn plasma levels. To perform the validation, both venous and finger-stick blood samples

were collected from 40 volunteers. Validation blood draws were performed by trained staff in the Human Performance/Clinical Research Laboratory (HPCRL) at CSU. CRP assays for the original and validation studies were performed in the HPCRL.

The high-sensitivity enzyme immunoassay procedure for CRP in dried blood spots has been described elsewhere (McDade et al. 2004). The modified version of this laboratory enzyme linked immunosorbent assay is described elsewhere (Robinson et al. 2007). Both dried blood and plasma CRP were assayed using the VIRGO CRP 150 Kit (Catalog # 66203; Hemagen Diagnostics, Inc, Columbia MD). Briefly, standards for the enzyme linked immunosorbent assay on dried blood spots were created using isolated red blood cells from whole blood samples. Whole blood was collected as previously described and the plasma and buffy coat were removed following centrifugation. The red blood cells were washed by adding EDTA (2% K3-EDTA in saline) to a 50% hematocrit and mixing by inversion. This mixture was centrifuged (2800 RPM at 0°C for 10 minutes) and the supernatent was discarded along with any additional buffy coat. This process was repeated for a total of three washes. The final isolated red blood cell layer was suspended with equal part K3-EDTA. CRP standards were created by serial dilution of the 50 mg/L standard using dilution buffer (0.01 M phosphate buffer, 0.5 m NaCl, pH 7.2+0.3, adjusted to 1 L with ultra-pure H₂O) to produce concentrations of 25, 12.5, 6.75, 3.125, 1.56, 0.39, 0.195, and 0.09 mg/L. Blanks were created using the 0 mg/L standard provided in the kit. The standards were diluted 1:2 with washed erythrocytes and K3-EDTA mixture, then 50 μ L drops were pipetted to filter cards which were allowed to dry overnight and then sealed with desiccant for storage at -80°C. CRP control samples were prepared in a similar manner using the provided 5, 10, and 15 mg/L CRP standards. Dried blood spots were reconstituted by obtaining 3.2 mm discs using a standard holepunch and eluting them overnight at 4°C in 250 μ L of wash buffer (dilution buffer with 1 ml Tween 20). The following morning, samples were rotated (300 rpm for 1 hour at room temperature). The eluate was then analyzed following manufacturer specifications for the enzyme linked immunosorbent assay protocol. Breifly, 100 μ L of reconstituted standard, sample, and control was added to each well and incubated for 30 minutes at room temperature. Wells were washed using the previously described wash buffer, then 100 μ L of anti-human horseradish peroxidase conjugate was added and allowed to incubate for 30 minutes at room temperature. Wells were added. Fifty microliters of sulfuric acid was added to stop the enzymatic reaction and absorbance was read at 450 nm. A linear fit was applied to the standard curve and correlations were determined (Robinson et al. 2007).

<u>Questionnaire</u>

A standardized respiratory symptoms and disease questionnaire developed and validated by the American Thoracic Society was translated into Spanish and administered to all participating women at the end of the personal monitoring period. Prior to initiating the survey, a study investigator measured the participant's height, weight, and waist circumference. The survey collected information on respiratory symptoms/illnesses and related variables (e.g., asthma, smoking history, family history) (Ferris 1978). The usual presence of symptoms was assessed by asking questions about the usual frequency and duration of symptom occurrence. For example, in addition to "Do you usually have a cough?," questions also asked "Do you usually cough as much as four to six times a day, four or more days out of the week?" and "Do you usually cough like this on most days for three consecutive months or more during the year?" This information was collected for cough, phlegm, wheezing, shortness of breath, nasal irritation, and headache. We modified the survey to collect demographic information, occupation, and information related to exposure (e.g., fuel type, time spent cooking, housing materials) (Albalak et al. 2001) and outcome (e.g. medication and supplement use, recent illnesses, menopausal status, and fish consumption).

A brief questionnaire was also developed for the CRP validation study. This survey collected information on age, gender, height, weight, ethnicity, race, smoking status, amount of exercise, recent and chronic illnesses, any medication and vitamin use, and occupation.

Data analyses

Data was analyzed using the SAS computer program (SAS 9.1, SAS Institute, Cary, NC). Study population characteristics; including air quality, health endpoints, personal characteristics, and kitchen and cooking characteristics, were assessed descriptively by calculating the mean and standard deviation (SD) for continuous variables and the frequency and percent for categorical variables for the total population as well as stratified by stove type. Air quality means and standard deviations were also calculated across levels of the stove scale. Geometric means were also calculated for air quality parameters. Spearman correlations were calculated for air quality measures and also for FEV_1 , PEF and CRP. For categorical variables, frequency tables were created and, in the case of sparse cells, categories were combined. Only variables with responses from at least 75% of the population were used in multivariate methods. Variables with missing data were the result of adding questions to the survey after the study began and not due to participants refusing to answer questions.

Exposure assessment models: The goal of this analysis was to determine a set of variables that best explained the variation in the air quality measurements using linear regression. Univariate associations were calculated to determine the individual contributions of potential predictors. Then a best subsets selection method was performed to determine the final multivariate model. Initially, adjusted mean pollutant levels were calculated for traditional and improved stoves, as well as for the four levels of the stove scale. Stove type (Justa versus traditional), stove scale, chimney condition scale, total area of windows (no windows, window area < 700 square inches, and window area > 700 square inches), the number of walls in the kitchen (less than four walls versus four walls), the number of doors in the kitchen (no doors, one door, and more than one door), the volume of the kitchen (cubic feet), the number of walls with eave spaces (no walls, 1-2 walls, and 3-4 walls), the primary material of the walls (blocks/bricks, wood, and iron sheets), the age of the stove (years), the hours the fire burns on a typical day (hours), and the time spent in the room with the fire burning (hours; for personal $PM_{2.5}$ models only), were evaluated as predictors in univariate and potentially, multivariate models predicting air quality measurements. Square and cube polynomial terms of the continuous variables were also assessed.

Correlations (for continuous variables) and chi-square tests (for categorical variables) were used to assess the potential for collinearity between variables. Stove type, stove scale, and chimney condition were considered to be collinear with each other and therefore were not allowed in the same model.

Next, univariate R-square calculations were performed separately for each of the three exposure measurements (personal $PM_{2.5}$, indoor $PM_{2.5}$, and indoor carbon monoxide 1-hr maximum) as the dependent variable. For continuous independent variables, studentized residual plots and Quantile-Quantile plots were created to evaluate the assumptions of linear regression (linearity, homoscedasticity, and the errors are normally distributed). The natural logarithms of the exposure parameters were created for use as the dependent variables in order to satisfy assumptions.

In multivariate assessments, separate models for predicting the particulate matter and carbon monoxide concentrations were performed for stove type and stove scale in order to assess the importance of the manner in which the stove was described. Chimney condition was assessed in a separate model predicting indoor $PM_{2.5}$ because it univariately explained more of the variation as compared to stove scale. All independent variables were considered as potential predictors in each multivariate regression. Additionally, all first-order interaction terms between the predictor variables were

assessed in multivariate models. Analyses were limited to first-order interaction terms due to the small sample size.

The selection criterion (an index computed for each candidate model and used to compare models (Kleinbaum et al. 1998) consisted of a combination of R-square and Mallow's Cp that was used to determine the best multivariate prediction model. The selection criteria compare the maximum model with all potential predictors to a reduced model with fewer predictors. Considering more than one criterion is often recommended because no single criterion is best (Kleinbaum et al. 1998). Due to the small sample size, if criteria for model selection are similar for multiple models, the most parsimonious model was chosen. In general, the smaller the sample, the smaller the maximum model should be with the weakest requirement being to have a minimum of 10 error degrees of freedom ($n \ge 10 + k + 1$; where k = predictors); another rule of thumb is to have at least 5 (or 10) observations per predictor ($n \ge 5k$ or $n \ge 10k$) (Kleinbaum et al. 1998).

Although the use of R-square is a common selection criterion, a limitation is that the R-square will always increase with the addition of variables to the model. One method frequently used is to choose the model in which the addition of variables has resulted in only a minor increase in the R-square. The plot of R-square verses the number of model predictors yields a number of maximum R-square values which remain quite flat as the number of model predictors decreases and then turns sharply downward. The number of model predictors at which this "knee" occurs is frequently used to indicate the number of terms in the model (Hocking 1976). Because of the subjective nature of defining this

"knee," another limitation of the R-square is that important predictors are often deleted during this step. It is, therefore, useful to combine this criterion with others, such as Mallow's Cp (an estimate of total prediction error). When comparing sets of models, choosing the model with the lowest Cp will result in the model with the smallest mean squared prediction error.

A variable selection technique was used based on the addition of variables that would most increase the adjusted R-square as well as a technique that would result in the lowest Mallow's Cp. Several models with increased R-square and decreased Cp were identified as candidates for the best prediction model. If these criteria were similar, then the model with the fewest predictors was chosen. In order to evaluate the assumptions of linear regression, partial plots were used to test for linearity; a plot of studentized residuals versus predicted values was used to check for homoscedasticity; and a stem-and-leaf plot, boxplot, normal probability plot, and the Shapiro-Wilk statistic were performed on the residuals in order to assess normality. The natural logarithm transformation of the dependent exposure variables was used to satisfy the assumptions of linear regression.

In order to determine whether or not exposure to second-hand smoke influenced the predictive abilities of the stove and kitchen parameters, analyses were stratified by any exposure to second-hand smoke versus no exposure to second-hand smoke. In addition, the particulate matter models were stratified by high and low outdoor $PM_{2.5}$ to determine the influence of ambient particulate matter levels on the relationship between stove/kitchen parameters and personal/indoor $PM_{2.5}$.

41

ī

The goal of this analysis was to determine whether or not the more easily obtained stove and housing variables could be as informative in assessing exposure as the more time and cost intensive exposure measurements.

Health effects assessments: The goal of the health effects analyses was to obtain a valid estimate of the association between cookstove exposures and lung function, CRP, and symptoms. Linear regression coefficients and logistic regression odds ratios were evaluated to determine the relationship between each of the health endpoints and each of the cookstove exposures, separately.

Lung function & CRP: Means and standard deviations for CRP (geometric means and standard deviations were calculated for CRP), FEV_1 , and PEF and symptom frequencies were calculated for women using improved versus traditional stoves, as well as for women in each of the 4 levels of stove scale.

Linear regression was used to assess the relationship between lung function and CRP and cookstove exposures. Cookstove exposures were assessed separately as personal $PM_{2.5}$, indoor $PM_{2.5}$, indoor carbon monoxide 1-hr maximum, stove type, and stove scale. Initially, the univariate associations between lung function and the exposures of interest and potential confounders were assessed. The $PM_{2.5}$ and carbon monoxide exposures were assessed continuously and divided by the interquartile range (IQR) for each exposure in order to create a more meaningful regression estimate. Therefore, coefficients were expressed per increase in IQR. Multivariate associations were assessed

by first entering the exposure of interest in a model with age and height. These potential confounders were chosen based on previous literature and the potential for the variable to influence the association of interest. Due to the small sample size, a low number of covariates was preferable. Waist circumference, second-hand smoke exposure, education level (as a potential measure of socio-economic status), and outdoor afternoon average temperature were also considered potential confounders, based on previous literature and the potential for the variable to influence the association of interest, and were entered into the model one at a time to determine the influence of each potential confounder on the estimate of interest (cookstove exposure and lung function). The influence of each potential confounder on the estimate of interest was based on clinically meaningful changes in the estimate. The final model was chosen based on the validity of the estimate of interest (the estimate did not meaningfully change when variables were entered into or removed from the model) and the efficiency of the model (if model estimates were similar, the model with the fewest parameters was chosen). Assumptions of linear regression were assessed. Partial plots were used to test linearity; plots of studentized residuals versus predicted values were used to check homoscedasticity; and stem-and-leaf plots, boxplots, normal probability plots, and Shapiro-Wilk statistics were performed on the residuals in order to assess normality. The fit of the final model was assessed by evaluating the R-square and collinearity was assessed using variance inflation factors. A variance inflation factor greater than ten was used to indicate collinearity. These analyses were also checked for subjects that may have had too much influence on the coefficient of interest. DFBETAS were calculated for the exposure variable within each model. The DFBETA is an indicator of how many standard deviations the coefficient of interest changes when the particular data point (or subject) is removed. If the DFBETA was greater than 2 divided by the square-root of the sample size $(2/\sqrt{n})$ then a sensitivity analysis without the subject(s) was performed to determine the effect, if any, the potential outliers had on the coefficient of interest.

After completion of the main analyses, models were stratified by the following variables in order to evaluate the heterogeneity of the estimates: age (less than 40 yrs versus 40 yrs and older; approximate median split), second-hand cigarette smoke exposure (any in the kitchen or home and none in the kitchen or home), outside levels of PM_{2.5} (less than 167 $\mu g/m^3$ and greater than 167 $\mu g/m^3$; approximate median split), village of residence (Santa Lucia and Suyapa), the amount of time the woman spent in the room with the fire burning (less than three hours and three or more hours) (used in all models except personal $PM_{2.5}$ models), any medication intake versus no medication intake, outside average afternoon temperature (less than 22.5 degrees Celsius and greater than 22.5 degrees Celsius; approximate median split), reported concern that stove smoke causes health problems (yes or no), the stove type (traditional versus improved; for analyses where stove type was not the exposure of interest), any current respiratory symptoms (yes or no), and the length of time with the current stove (less than three years and three or more years). The goal of the stratification by outdoor levels of PM_{2.5} was to determine if the reductions in indoor concentrations could lower the risks of adverse health effects despite elevated outdoor concentrations (Naeher et al. 2000a). Due to the small sample size, sensitivity analyses were also performed among the following subgroups of participants: those performing at least two successful lung function maneuvers (lung function models only),

women having their current stove for at least six months (Ramirez-Venegas et al. 2006), those without a history of smoking, women indicating their day would not have been different if monitoring had not taken place, and among women not taking bronchodilator medications (lung function models only). Sensitivity analyses were also performed by creating tertiles of particulate matter and carbon monoxide exposures in order to assess the potential for a dose-response relationship on a non-linear scale. In the models using the stove type or the stove scale as the exposure of interest, several factors thought to influence the ventilation inside the kitchen, and therefore, the exposure were assessed as potential effect modifiers. These included the presence or absence of kitchen windows, the volume of the kitchen (greater than 700 cubic feet versus less than 700 cubic feet; approximate median split), kitchens with walls with eave spaces, the number of kitchen walls, and kitchens attached to or part of the main living area.

Stratified coefficients of interest and 95% confidence intervals were reported to assess effect modification. Due to our sample size, performing stratifications were preferable to interaction terms because this method allowed for the visual inspection of coefficients among the two groups while interaction term p-values would likely not have reached statistical significance. When the small sample size did not allow for stratification, sensitivity analyses were performed by excluding the women in the category with the small sample size. The coefficient of interest for the remaining population was then determined in order to evaluate the influence of that particular factor.

45

Methods for assessing the relationship between the exposure of interest and CRP levels were performed in a similar manner as compared to the lung function analyses except waist circumference was included, along with age and height, in the multivariate models and fish consumption, menopausal status, and the presence of a cold or sinus problem in the previous week were considered as potential confounders in addition to those previously listed. In addition, a more specific stratification by medication intake was performed by including only ant-inflammatory and heart medications. Stratification based on whether or not the woman indicated a cold or sinus problem during the previous week was also performed because recent illness is known to affect CRP levels.

A sensitivity analysis was performed in order to assess the relationship between air quality measures and percent predicted FEV_1 (less than 80% versus 80% or more) using logistic regression. Because standardized reference equations for Honduran women do not exist, predicted FEV_1 for each participant was based on age and height-adjusted reference equations for Mexican-American women (Hankinson et al. 1999). Initially, the univariate associations (odds ratios) between exposures of interest and dichotomized percent predicted FEV_1 were calculated. The particulate matter and carbon monoxide exposures were divided by the IQR for each exposure. Therefore, the odds ratios and 95% confidence intervals presented are per increase in IQR. Multivariate associations were assessed by adding age, height, waist circumference, second-hand smoke exposure, education level, and outdoor average temperature one at a time to a model including the exposure of interest. The influence of each potential confounder on the odds ratio of interest was based on clinically meaningful changes in the estimate. Models were chosen

based on the validity of the odds ratio of interest (the odds ratio did not meaningfully change when variables were entered into or removed from the model) and the efficiency of the model (if odds ratios were similar, the model with the fewest parameters was chosen). In logistic regression, the variability and hence the width of the confidence intervals of the estimated coefficients increases as variables are added to the model. Therefore, it is important to find a parsimonious model especially when the sample size is small (Robinson and Jewell 1991). Goodness of fit was not assessed due to the small sample size (Hosmer & Lemeshow 2000). Stratified and sensitivity analyses were performed using the same variables as in the FEV₁ linear regression models.

Respiratory symptoms: Logistic regression was used to assess the relationship between respiratory symptoms and cookstove exposures. Initially, the univariate odds ratios between the exposures of interest and respiratory symptoms were calculated. The particulate matter and carbon monoxide exposures were divided by the IQR for each exposure in order to create a more meaningful regression estimate. Therefore, the odds ratios and confidence intervals presented are per increase in IQR. Multivariate associations were assessed by first entering the exposure of interest in a model with age. Due to the small sample size, a low number of covariates was preferable. Height, waist circumference, second-hand smoke exposure, education level, and average outdoor temperature were also considered potential confounders and were entered into the model one at a time to determine the influence of each potential confounder on the odds ratio of interest was based on clinically meaningful changes in the estimate. Models were chosen based on

achieving a more valid odds ratio of interest (the odds ratio did not meaningfully change when variables were entered into or removed from the model) and the efficiency of the model (if odds ratios were similar, the model with the fewest parameters was chosen). Again, in logistic regression, the variability and hence the width of the confidence intervals of the estimated coefficients increases as variables are added to the model. Therefore, it is important to find a parsimonious model especially when the sample size is small (Robinson and Jewell 1991). Crude estimates, age or age and second-hand smoke adjusted estimates, adjusted estimates including outdoor average temperature, and adjusted estimates eliminating those on bronchodilator medications were presented. Goodness of fit was not assessed due to the small sample size (Hosmer & Lemeshow 2000). Due to the small number of subjects with the respiratory symptom outcomes, effect modification was not assessed. However, when $PM_{2.5}$ and carbon monoxide were associated with symptoms, co-pollutant models were performed with both $PM_{2.5}$ and carbon monoxide in the same model to assess whether or not one of the pollutants was acting as a confounder of the other.

The relationships between current symptoms and FEV_1 , PEF, and CRP were assessed by calculating the means and 95% confidence intervals for each of the health endpoints across categories of current symptoms. In addition, the p-value comparing the health endpoint means (mild, moderate, or severe symptoms versus no symptoms) was calculated.

48

<u>Power</u>

Power calculations for several health endpoints were performed during the study design phase and prior to data analysis. Presence of cough was used to represent respiratory symptoms. Among improved stoves users in Guatemala, 27% reported morning cough during the cool wet season (Bruce et al. 1998). This percentage was used to estimate the prevalence of disease among the unexposed. With a sample size of 80 participants, 80% power, and an alpha = 0.05, the minimum detectable odds ratio for cough comparing traditional and improved stove users is 4.1 (Epi Info, version 3.3.2). Although this is relatively high, risk estimates for respiratory symptoms have been reported from 2.0 to 4.8 in developing countries (Bruce et al. 1998; Khushk et al. 2005; Shrestha and Shrestha 2005). The proposal had only 23% power to detect an OR of 2.0. A limitation of this method was that it assumed a univariate assessment. Power will most likely decrease upon adjustment.

A two-group independent sample t-test was used to estimate the power to detect differences in FEV₁ among improved and traditional stove users with a sample size of 80 women and alpha = 0.05 (Table 3.1) (SAS, version 9.1). According to Table 3.1, this study had more than 99% power to detect a difference of 0.34 liters and 73% power to detect a difference of 0.29 liters. Saha and colleagues reported a 0.29 liter decrease in FEV₁ for biomass users as compared to non-users (Saha et al. 2005). Gharaibeh reported a 0.34 liter decrease in FEV₁ among children exposed to wood and kerosene stoves as compared to those exposed to electric or modified kerosene stoves (Gharaibeh 1996);
however, children may be more susceptible to biomass smoke exposures as compared to adult women. Again, a limitation is that analysis was based on univariate associations.

No studies to date have examined CRP levels among traditional and improved stove users in developing countries. Power was calculated using a multiple regression type III F test to determine the minimum detectable partial correlation between CRP and an indoor pollutant while adjusting for 3 potential confounders (Table 3.2) (SAS, version 9.1). With approximately 60 participants with both indoor pollutant and CRP measurements, this study could detect a minimum partial correlation between 0.35 and 0.40 with 80% power. An advantage of this method was that it incorporated multivariate regression. However, its utility was somewhat limited as expected partial correlations between CRP and PM_{2.5} or carbon monoxide are generally not presented in the literature.

Reference	Difference in mean FEV1 (liters)	Pooled standard deviation	Power
Gharaibeh NS, 1996	0.34	0.14	>99%
Saha A, 2005	0.29	0.50	73%
Behera D, 1997	0.07	0.14	60%

Table 3.1. Power estimates to detect differences in FEV_1 among improved and traditional stove users with a sample size = 80 and alpha = 0.05.

Partial correlation	Power
0.25	47%
0.30	63%
0.35	77%
0.40	88%
0.45	95%

Table 3.2. Power estimates to detect partial correlations between C-reactive protein and indoor pollutant levels for a multivariate model with 4 predictors, a sample size = 60, and alpha = 0.05.

CHAPTER 4

RESULTS

Descriptive

Seventy-nine women participated in the study (41 with improved stoves and 38 with traditional stoves). Personal characteristics are presented for the total study population as well as stratified by stove type (Improved or *Justa* stoves versus traditional stoves) in Tables 4.1 and 4.2. Women with improved stoves (mean age: 45.0 years) were older than those with traditional stoves (mean age: 37.8 years) (Table 4.1). Weight, height, waist circumference, and BMI were similar among women in the two stove groups (Table 4.1). Thirty-eight percent of women reported taking any medications on a regular basis or as prescribed by a doctor while 13.9% reported, specifically, the intake of anti-inflammatory or heart medications (Table 4.2). A higher proportion of women with traditional stoves reported having had a cold or the flu during the week prior to data collection (32.4% as compared to 14.6%; Table 4.2). Approximately 30% of women were unable to provide a successful lung function maneuver and these women were evenly distributed across stove type (Table 4.2). Only four women (5.1%) reported being previous cigarette smokers; however, 31.7% reported the presence of smokers in the house or kitchen (Table 4.2).

Stove, kitchen, and cooking characteristics are presented in Tables 4.3 and 4.4. Women with traditional stoves had their current stove for a longer amount of time (9.0 years) as compared to women with improved stoves (2.4 years) (Table 4.3). Reported hours

typically spent cooking each day, the hours the fire burns on a typical day, and the hours spent in the room with the fire burning were similar for women with traditional and improved stoves (Table 4.3). Forty-one (51.9%) women cooked using an Improved *Justa* stove and 38 (48.1%) cooked using a traditional stove (Table 4.4). The distribution of women within categories of the stove scale was 23 for high quality stoves, 17 for high-mid quality stoves, 15 for low-mid quality stoves, and 24 for low quality stoves (Table 4.4). Fifty-five percent of women with traditional stoves cooked in kitchens with no windows as compared to 37% of women with improved stoves (Table 4.4). Forty-seven percent of the traditional stoves had chimneys; however, zero were rated as "very good," five (13%) were rated as "fairly good," and 13 (34%) were rated as "poor" (Table 4.4).

Exposure assessment models

The indoor carbon monoxide average and the indoor carbon monoxide 1-hr maximum were highly correlated (spearman correlation coefficient = 0.98); therefore, the 1-hr maximum was used in analyses. Means, standard deviations, minimum measures, maximum measures, and geometric means for personal $PM_{2.5}$ (µg/m³), indoor $PM_{2.5}$ (µg/m³), and indoor carbon monoxide 1-hr maximum (ppm), as well as outdoor $PM_{2.5}$ (µg/m³), indoor carbon dioxide (ppm), indoor temperature (degrees Celsius), indoor percent relative humidity, and afternoon outdoor average temperature (degrees Celsius) are presented in Table 4.5. Elevated mean outdoor $PM_{2.5}$ levels among homes with traditional stoves as compared to improved stoves indicate that outdoor monitors may not have been placed far enough away from the stoves (or out of the direct plume of smoke). Mean personal $PM_{2.5}$ levels for traditional stoves users as compared to improved stoves

users were 197.71 μ g/m³ and 73.56 μ g/m³, respectively; mean indoor PM_{2.5} levels were 1002.29 μ g/m³ and 266.24 μ g/m³, respectively; and indoor carbon monoxide 1-hr maximum levels were 14.34 ppm and 1.84 ppm, respectively (Table 4.5). Air quality means and standard deviations are also presented across the 4-level stove scale with a clear pattern of increasing pollutant levels as the stove quality deteriorates (Table 4.6). Personal PM_{2.5} (μ g/m³), indoor PM_{2.5} (μ g/m3), and indoor carbon monoxide 1-hr maximum (ppm) were moderately correlated (spearman correlation coefficients ranged from 0.61 to 0.69), while correlations with outdoor PM_{2.5} were reduced (Table 4.7).

Stove, housing, and cooking parameters were initially assessed univariately in linear regression models with measured pollutants as the dependent variables (separate models for each pollutant) to determine how much of the variation in the air pollutants was explained by each factor (Tables 4.8, 4.9, and 4.10). From the univariate results, the stove scale best predicted personal $PM_{2.5}$ levels (R-square = 0.51) and the most important kitchen parameter was the total area of the kitchen windows (R-square = 0.14) (Table 4.8). Univariate variation in indoor $PM_{2.5}$ was explained best by the four-level chimney condition (R-square = 0.44) and the stove scale (R-square = 0.40); the total area of the kitchen windows explained more variation than other kitchen parameters (R-square = 0.25) (Table 4.9). The stove scale univariately explained 79% of the variation in indoor carbon monoxide 1-hr maximum levels while the chimney condition explained 61% and the stove type explained 54% (Table 4.10). Important kitchen parameters were the volume of the kitchen (natural logarithm transformation) (R-square = 0.24), the number of kitchen doors (R-square = 0.21), and the total area of the kitchen windows (R-square = 0.24), the number

0.19) (Table 4.10). For all three exposure levels, polynomial terms did not meaningfully increase the R-square as compared to the respective linear term in univariate models and therefore, were not considered in the multivariate assessments.

Multivariate assessments utilizing the four-level stove scale and including a term for the age of the stove (years) explained 54% of the variation in natural logarithm transformed personal $PM_{2.5}$ (Table 4.11). Stove scale and the age of the stove resulted in the lowest Mallow's Cp statistic, and the addition of how long the fire burns on a typical day and the time typically spent in the kitchen with the fire burning only increased the R-square by 0.03 (Table 4.11). The addition of a first-order interaction term between age of the stove and the hours the fire burns on a typical day to the main effects model only resulted in an overall R-square of 0.60 (Table 4.11). Therefore, the final predictors of personal $PM_{2.5}$, resulting in the most parsimonious model, were the stove scale and the age of the stove (Table 4.11). Stratifying the model with stove scale and age of the stove by second-hand smoke exposure resulted in a 14% increase in the R-square among women exposed to second-hand smoke although this was among only 17 women which limits the interpretation (Table 4.11.01). Stratifying the model by outdoor $PM_{2.5}$ resulted in a 10% increase in R-square among those with elevated outdoor concentrations (Table 4.11.01). However, interpretations of outdoor PM_{2.5} stratifications are limited because of the apparent influence of the stove on outdoor concentrations. It is possible that the outdoor monitors were not placed far enough away from the kitchens. The use of stove type (Justa versus traditional) rather than the stove scale in multivariate assessments including the age of the stove predicted 45% of the variation in personal $PM_{2.5}$ (Table 4.12). Age

of the stove was important and the addition of other variables only increased the R-square by 0.03 (Table 4.12).

The multivariate model using the four-level stove scale with the lowest Mallow's Cp explained 57% of the variation in natural logarithm transformed indoor PM_{2.5} (Table 4.13). In addition to the four-level stove scale, important predictors were the total area of the kitchen windows, the number of kitchen walls, and the primary material of the kitchen walls. The use of these three kitchen parameters increased the R-square 0.17 above the R-square of the stove scale alone (Table 4.13). The addition of the volume of the kitchen only increased the R-square by 0.01 (Table 4.13). Stratification of the model explaining 57% of the variation in indoor $PM_{2.5}$ by second-hand smoke resulted in similar R-square estimates (Table 4.13.01). Among women exposed to decreased outdoor $PM_{2.5}$ concentration, 68% of the variation in indoor PM_{2.5} was explained by stove scale, area of the kitchen windows, number of kitchen walls, and the primary material of the kitchen walls (Table 4.13.01). Again, however, the interpretation of this analysis is limited by the apparent influence of the stove smoke on outdoor concentrations. The model utilizing stove type did not explain the variation in indoor $PM_{2.5}$ as well as the use of the stove scale. The most parsimonious model using stove type included the total area of the kitchen windows and the number of kitchen walls and explained 45% of the variation in indoor PM_{2.5} (Table 4.14). Because the univariate R-square for chimney condition was higher than that of stove scale, a multivariate model using chimney condition was also evaluated (Table 4.15). The most parsimonious model included chimney condition, the total area of the kitchen windows, the number of kitchen walls, the primary material of the kitchen walls, and the volume of the kitchen and explained 60% of the variation in indoor $PM_{2.5}$ which was slightly higher than the model using the stove scale (Tables 4.13 and 4.15). First-order interaction terms did not further explain the variation in indoor $PM_{2.5}$ in the stove scale, stove type, or chimney condition models.

Multivariate assessments using the stove scale and including the volume of the kitchen and the number of walls with eave spaces explained 85% of the variation in natural logarithm transformed indoor carbon monoxide 1-hr maximum levels (Table 4.16). The addition of the volume of the kitchen and the number of walls with eave spaces increased the R-square only 0.06 above that of the stove scale alone (R-square = 0.79); however, this three variable model resulted in the lowest Mallow's Cp (Table 4.16). The addition of how many hours the fire typically burns increased the R-square by 0.01 (Table 4.16). Stratification of the model including stove scale, volume of the kitchen, and the number of walls with eave spaces by second-hand smoke exposure did not influence the variation explained (Table 4.16.01). The multivariate model using stove type explained 63% of the variation in indoor carbon monoxide 1-hr maximum when the volume of the kitchen was added (Table 4.17). The utility of the stove scale as compared to stove type was most pronounced when assessing indoor carbon monoxide 1-hr maximum levels. The multivariate model using the stove scale explained 22% more of the variation as compared to the multivariate model using stove type (Tables 4.16 and 4.17). As in the indoor PM_{2.5} models, the use of first-order interaction terms did not improve the prediction capabilities of the stove and housing characteristics.

<u>Health effects assessments</u>

Means, standard deviations, minimum measures, and maximum measures of lung function and CRP among all women and stratified by stove type are presented in Table 4.18 (geometric means and standard deviations were presented for CRP). The highest eligible FEV₁ was highly correlated with the mean of up to 3 eligible maneuvers of FEV₁ (Spearman correlation coefficient = 0.99). Similarly, the highest eligible PEF was highly correlated with the mean of up to 3 eligible maneuvers of FEV₁ and PEF were used in further analyses. Lung function and CRP means and standard deviations are presented across levels of the stove scale in Table 4.19. Correlation coefficients for the three continuous health endpoints (PEF, FEV₁, and CRP) are presented in Table 4.20. Symptom frequencies are presented for the total population and stratified by stove type in Table 4.21 and stratified by stove scale in Table 4.22. A higher percentage of women with traditional stoves reported symptoms of cough, phlegm, wheeze, shortness of breath, and headache (Table 4.21). Similar patterns, although not as distinct, are demonstrated for symptom frequencies across the stove scale (Table 4.22).

Forced Expiratory Volume in 1 second: Univariate linear regression coefficients for the relationship between FEV₁ and cookstove exposures and potential confounders are presented in Table 4.23. Age, height, waist circumference, second-hand smoke exposure, and education level were at least moderately associated with changes in FEV₁ (Table 4.23). A 10 year increase in age was associated with a 0.21 liter decrease in FEV₁; a 3 inch (IQR) increase in height was associated with a 0.25 liter increase in FEV₁; a 5.5 inch

(IQR) increase in waist circumference was associated with a 0.16 liter decrease in FEV₁; no education as compared to more than 5 years of education was associated with a 0.53 liter decrease in FEV₁; and, although not statically significant, exposure to second-hand smoke was associated with a 0.21 liter decrease in FEV_1 (Table 4.23). Various multivariate models for the association between personal $PM_{2.5}$ and FEV_1 are presented in Table 4.24. Waist circumference, second-hand smoke exposure, education level, and outdoor temperature did not meaningfully alter the estimate of interest (Table 4.24). The final multivariate model included adjustment for age and height (Table 4.24). This reduced model was chosen because of the small sample size and because the model had similar estimates to those of the full model. Although likely not clinically meaningful, an increase in the IQR (106.1 μ g/m³) of personal PM_{2.5} was associated with a 0.07 liter (95% CI: 0.01 to 0.13) increase in FEV₁ (Table 4.24). The use of tertiles to assess personal PM_{2.5} indicates that the effect was driven mainly by the high tertile exposure group because there was no difference between the low and medium exposure tertiles (Table 4.24.01). The effect modification and sensitivity analyses are presented in Table 4.24.02 and Table 4.24.03; most stratifications did not meaningfully change the association between personal $PM_{2.5}$ and FEV_1 . However, among women exposed to second-hand smoke and among women with any medication intake, an increase in the IQR of personal PM_{2.5} was associated with a 0.15 liter or 0.18 liter increase, respectively, in FEV_1 (Table 4.24.02). Although these associations approached significance, the coefficients are in the direction opposite to that hypothesized and the magnitude of the coefficients are likely not clinically meaningful.

Various multivariate models for the association between indoor $PM_{2.5}$ and FEV_1 are presented in Table 4.25. The estimate of interest was consistent with a null association regardless of adjustment. The final multivariate model to be used in stratified analyses included adjustment for age and height (Table 4.25). An increase in the IQR (572.3 μ g/m³) of indoor PM_{2.5} was not associated with a change in FEV₁ (Table 4.25). The use of tertiles to assess indoor PM_{2.5} had similar results (Table 4.25.01). The effect modification and sensitivity analyses are presented in Table 4.25.02 and Table 4.25.03; most stratifications did not meaningfully change the association between indoor PM_{2.5} and FEV₁. However, the association among women less than 40 years of age was consistent with the null while a 0.27 liter increase in FEV₁ was associated with an increase in IQR indoor PM_{2.5} among women 40 years and older (Table 4.25.02). This may indicate that the unexpected positive associations between exposures and lung function are driven mainly by women in the 40 years and older age category.

Various multivariate models for the association between indoor carbon monoxide 1-hr maximum and FEV_1 are presented in Table 4.26. The estimate of interest was consistent with a null association regardless of adjustment. The final multivariate model to be used in stratified analyses included adjustment for age and height (Table 4.26). The coefficient for this model was slightly smaller than that of the full model including indoor carbon monoxide 1-hr maximum, age, height, waist circumference, second-hand smoke, and education level; however, the difference is likely not clinically meaningful. Therefore, the more parsimonious model adjusted for only age and height was used in further analyses. An increase in the IQR (4.62 ppm) of indoor carbon monoxide 1-hr

maximum was not associated with a change in FEV_1 (linear regression beta coefficient = 0.02; 95% CI: -0.05 to 0.10; Table 4.26). The use of tertiles to assess indoor carbon monoxide 1-hr maximum had similar results (Table 4.26.01). The effect modification and sensitivity analyses are presented in Table 4.26.02 and Table 4.26.03; most stratifications did not meaningfully change the association between indoor carbon monoxide 1-hr maximum and FEV₁. However, the association among women indicating that they were concerned that stove smoke causes health problems was consistent with the null while a 0.33 liter increase in FEV₁ was associated with an increase in IQR indoor carbon monoxide 1-hr maximum among women indicating no concern (Table 4.26.02). Although statistically significant, this result should be interpreted with caution as only nine women indicated no concern.

Multivariate models for the association between stove type and FEV_1 are presented in Table 4.27. Waist circumference, second-hand smoke exposure, and education level did not meaningfully alter the estimate of interest (Table 4.27). The use of a traditional stove as compared to an improved stove was suggestive of a small increase in FEV_1 (crude coefficient = 0.21; 95% CI: -0.03 to 0.45); however, this increase was attenuated upon adjustment for age and height (coefficient = 0.05; 95% CI: -0.11 to 0.20; Table 4.27). The effect modification and sensitivity analyses are presented in Table 4.27.01 through Table 4.27.03; stratifications, including stratifications by variables potentially affecting the ventilation in the kitchen, did not meaningfully change the association between stove type and FEV_1 . However, similar to the association between indoor carbon monoxide 1-hr maximum and FEV_1 , a positive association between exposure and FEV_1 was observed

among women indicating concern that the stove smoke caused health problems (Table 4.27.01).

Various multivariate models for the association between stove scale and FEV_1 are presented in Table 4.28. No association, crude or adjusted, was observed between stove scale and FEV_1 and no clear pattern was demonstrated across the four levels of the stove scale (Table 4.28). The effect modification and sensitivity analyses are presented in Table 4.28.01 through Table 4.28.03; stratifications, including stratifications by variables potentially affecting the ventilation in the kitchen, did not meaningfully change the association between stove scale and FEV_1 .

Results of sensitivity analyses assessing the relationship between air quality measures and dichotomized percent predicted FEV_1 are presented in Tables 4.30 through 4.34.03. Conclusions of the sensitivity analyses are similar to those of the FEV_1 linear regression models. The odds ratios vary among stratified groups of women, although confidence intervals are wide. Interpretation is limited due to the instability of the logistic regression models.

Peak Expiratory Flow: Similar results were observed between cookstove exposures and PEF. Univariate linear regression coefficients for the relationship between PEF and cookstove exposures and potential confounders are presented in Table 4.35. Age, height, and second-hand smoke exposure were associated with changes in PEF (Table 4.35). A 10 year increase in age was associated with 13.82 liter/minute (L/min) decrease in PEF; a

3 inch increase in height was associated with a 17.19 L/min increase in PEF; and exposure to second-hand smoke was associated with a 29.31 L/min decrease in PEF (Table 4.35). Various multivariate models for the association between personal $PM_{2.5}$ and PEF are presented in Table 4.36. Waist circumference, second-hand smoke exposure, education level, and outdoor temperature did not meaningfully alter the estimate of interest (Table 4.36). The final multivariate model included adjustment for age and height (Table 4.36). This reduced model was chosen because of the small sample size and because the model had similar estimates to those of the full model. Although likely not clinically meaningful, an increase in the IQR (106.1 μ g/m³) of personal PM_{2.5} was associated with a 16.0 L/min (95% CI: 2.71 to 29.30) increase in PEF (Table 4.36). The use of tertiles to assess personal $PM_{2.5}$ indicated that the positive association between personal PM_{2.5} and PEF is driven primarily by women in the highest tertile of exposure (Table 4.36.01). The effect modification and sensitivity analyses are presented in Table 4.36.02 and Table 4.36.03. Most stratification did not meaningfully change the association between personal PM_{2.5} and PEF. However, among 15 women with outdoor $PM_{2.5}$ levels less than 167 μ g/m³, an increase in the IQR of personal $PM_{2.5}$ was associated with a 69.9 L/min (95% CI: 1.14 to 138.72) increase in PEF; and among women living in Suyapa (n=20), among women exposed to an elevated outdoor temperature (n=20), and among women indicating no concern about the health effects of stove smoke (n=10), no association between personal PM_{2.5} and PEF was observed (Table 4.36.02).

64

Multivariate models for the association between indoor PM_{2.5} and PEF are presented in Table 4.37. The estimate of interest was consistent with a null association regardless of adjustment for waist circumference, second-hand smoke exposure, education level, or outdoor average temperature. The final multivariate model to be used in stratified analyses included adjustment for age and height (Table 4.37). An increase in the IQR $(572.3 \ \mu g/m^3)$ of indoor PM_{2.5} was not associated with a change in PEF (coefficient = -4.79; 95% CI: -20.10 to 10.53; Table 4.37). The use of tertiles to assess indoor PM_{2.5} indicated that the positive association between indoor $PM_{2.5}$ and PEF is driven primarily by women in the highest tertile of exposure (Table 4.37.01). The effect modification and sensitivity analyses are presented in Table 4.37.02 and Table 4.37.03. Most stratification did not meaningfully change the association between indoor PM_{2.5} and PEF. However, stratification by age (among those 40 years and older, there was a positive association between increasing exposure and increasing PEF), stratification by outdoor temperature (among those exposed to elevated outdoor average temperature, there was a positive association between increasing exposure and increasing PEF), and stratification by stove type did change the estimates; although the changes in PEF were small and may not be clinically meaningful (Table 4.37.02).

Various multivariate models for the association between indoor carbon monoxide 1-hr maximum and PEF are presented in Table 4.38. The estimate of interest was consistent with a null association regardless of adjustment for waist circumference, second-hand smoke exposure, education level, or outdoor average temperature. The final multivariate model to be used in stratified analyses included adjustment for age and height (Table

4.38). An increase in the IQR (4.62 ppm) of indoor carbon monoxide 1-hr maximum was not associated with a change in PEF (linear regression beta coefficient = -4.36; 95% CI: - 23.49 to 14.48; Table 4.38). The use of tertiles to assess indoor carbon monoxide 1-hr maximum had similar results (Table 4.38.01). The effect modification and sensitivity analyses are presented in Table 4.38.02 and Table 4.38.03; stratifications did not meaningfully change the association between indoor carbon monoxide 1-hr maximum and PEF. An increased positive association between indoor carbon monoxide and PEF was observed among women with improved stoves; however, the confidence interval is wide (Table 4.38.02).

Various multivariate models for the association between stove type and PEF are presented in Table 4.39. Waist circumference, second-hand smoke exposure, and education level did not meaningfully alter the estimate of interest (Table 4.39). The use of a traditional stove as compared to an improved stove was suggestive of a small increase in PEF (crude coefficient = 22.91; 95% CI: -9.96 to 55.78); however, this increase was attenuated upon adjustment for age and height (coefficient = 12.72; 95% CI: -19.93 to 45.36; Table 4.39). The effect modification and sensitivity analyses are presented in Table 4.39.01 through Table 4.39.03; stratifications, including stratifications by variables potentially affecting the ventilation in the kitchen, did not meaningful change the association between stove type and PEF.

Various multivariate models for the association between stove scale and PEF are presented in Table 4.40. No association, crude or adjusted, was observed between stove

scale and PEF and no clear pattern was demonstrated across the 4 levels of the stove scale (Table 4.40). The effect modification and sensitivity analyses are presented in Table 4.40.01 through Table 4.40.03; stratifications, including stratifications by variables potentially affecting the ventilation in the kitchen, did not meaningfully change the association between stove scale and PEF.

C-reactive Protein: The laboratory validation between plasma CRP and dried blood CRP resulted in an R-square of 0.75 and dried blood CRP was consistently 4-5 times higher than plasma CRP (Robinson et al. 2007). Although the dried blood CRP assay did not perform as well as other laboratory validations (McDade et al. 2004), results were analyzed for potential associations with cookstove exposures and interpreted with caution. Specifically, the scale, although reported in mg/L, is not consistent with previous and existing CRP literature. One value resulted in a negative concentration which was converted to one half of the smallest positive concentration (0.02 mg/L). This value may change slightly once an assay limit of detection is established (Robinson et al. 2007).

The natural logarithm transformation of CRP was used in analyses. Univariate linear regression coefficients for the relationship between CRP and cookstove exposures and potential confounders are presented in Table 4.41. Age, height, and waist circumference were univariately associated with the natural logarithm transformation of CRP (Table 4.41). Although the changes in CRP may not be clinically meaningful, increasing age was associated with increasing CRP, increasing height was associated with decreasing

CRP, and increasing waist circumference was associated with increasing CRP (Table 4.41). The use of vitamins or supplements was not associated with CRP levels (coefficient = -0.02; 95% CI: -1.00 to 0.96). Various multivariate models for the association between personal PM_{2.5} and the natural logarithm transformation of CRP are presented in Table 4.42. Second-hand smoke exposure, education level, fish consumption, menopausal status, and outdoor temperature did not meaningfully alter the estimate of interest (Table 4.42). The final multivariate model included adjustment for age, height, and waist circumference (Table 4.42). This reduced model was chosen because of the small sample size and because the model had similar estimates to those of the full model. An increase in the IQR (106.1 μ g/m³) of personal PM_{2.5} was not associated with a change in the natural logarithm transformation of CRP (coefficient = -0.22; 95% CI: -0.53 to 0.10; Table 4.42). The use of tertiles to assess personal $PM_{2.5}$ had similar results (Table 4.42.01). The effect modification and sensitivity analyses are presented in Table 4.42.02 and Table 4.42.03; most stratifications did not meaningfully change the association between personal $PM_{2.5}$ and the natural logarithm transformation of CRP. The difference in estimates among women owning their current stove less than three years as compared to three or more years suggests that the associations (although non-significant) observed between personal PM_{2.5} and CRP in the direction opposite to that hypothesized may be driven by women owning their current stove for a shorter amount of time (Table 4.42.02).

Various multivariate models for the association between indoor $PM_{2.5}$ and the natural logarithm transformation of CRP are presented in Table 4.43. The final multivariate

model to be used in stratified analyses included adjustment for age, height, and waist circumference (Table 4.43). An increase in the IQR (572.3 μ g/m³) of indoor PM_{2.5} was not associated with a change in the natural logarithm transformation of CRP (coefficient = 0.0003; 95% CI: -0.23 to 0.23; Table 4.43). The use of tertiles to assess indoor PM_{2.5} had similar results (Table 4.43.01). The effect modification and sensitivity analyses are presented in Table 4.43.02 and Table 4.43.03; stratification did not meaningfully change the association between indoor PM_{2.5} and the natural logarithm transformation of CRP.

Various multivariate models for the association between indoor carbon monoxide 1-hr maximum and the natural logarithm transformation of CRP are presented in Table 4.44. The final multivariate model to be used in stratified analyses included adjustment for age, height, and waist circumference (Table 4.44). An increase in the IQR (4.62 ppm) of indoor carbon monoxide 1-hr maximum was not associated with a change in the natural logarithm transformation of CRP (coefficient = 0.06; 95% CI: -0.22 to 0.34; Table 4.44). The use of tertiles to assess indoor carbon monoxide 1-hr maximum had similar results (Table 4.44.01). The effect modification and sensitivity analyses are presented in Table 4.44.02 and Table 4.44.03; stratifications did not meaningfully change the association between indoor carbon monoxide 1-hr maximum and the natural logarithm transformation of CRP.

Multivariate models for the association between stove type and the natural logarithm transformation of CRP are presented in Table 4.45. Second-hand smoke exposure, education level, fish consumption, and menopausal status did not meaningfully alter the estimate of interest (Table 4.45). When adjusted for age, height, and waist

69

circumference, the use of a traditional stove as compared to an improved stove did not influence the natural logarithm transformation of CRP (coefficient = -0.24; 95% CI: -0.90 to 0.42; Table 4.45). The effect modification and sensitivity analyses are presented in Table 4.45.01 through Table 4.45.03; most stratifications, including stratifications by variables potentially affecting the ventilation in the kitchen, did not meaningfully change the association between stove type and the natural logarithm transformation of CRP. However, although in the direction opposite to that hypothesized, the association between stove type and natural logarithm transformed CRP among women cooking in kitchen without windows was statistically significant (Table 4.45.03).

Various multivariate models for the association between stove scale and the natural logarithm transformation of CRP are presented in Table 4.46. No association, crude or adjusted, was observed between stove scale and the natural logarithm transformation of CRP and no clear pattern was demonstrated across the 4 levels of the stove scale (Table 4.46). The effect modification and sensitivity analyses are presented in Table 4.46.01 through Table 4.46.03; stratifications, including stratifications by variables potentially affecting the ventilation in the kitchen, did not meaningfully change the association between stove scale and the natural logarithm transformation of CRP.

Symptoms: Crude and adjusted odds ratios for the association between cookstove exposures and symptoms are presented in Tables 4.47 through 4.52. In addition, because of the reduction in sample size, adjusted odds ratios including outdoor temperature as a potential confounder and the adjusted odds ratios excluding those women taking

bronchodilators are also presented (Tables 4.47-4.52). Although not statistically significant, there was an increase in the odds of exposure to indoor PM_{2.5} among those women reporting the usual presence of a cough as compared to those not reporting cough (Table 4.47). A similar relationship was demonstrated for indoor carbon monoxide 1-hr max (Table 4.47). In the cough co-pollutant model, odds ratios for indoor $PM_{2.5}$ remained elevated (Table 4.47.01). The odds of cooking with a traditional stove was about 8-9 times as high among those reporting cough as compared to those not reporting cough and odds ratios increased as stove scale quality decreased (Table 4.47). The odds ratios for the association between cookstove exposures and the usual presence of phlegm were similar to those of cough; however, the odds ratios were attenuated and not as consistent (Table 4.48). As in the cough models, only odds ratios for indoor $PM_{2.5}$ remained elevated in the co-pollutant model for phlegm (Table 4.48.01). The percentages of women reporting phlegm were 0%, 17.65%, 13.33%, and 20.83% across the four-level stove scale ranging from high quality to low quality stoves. Similarly, the presence of chest wheeze or whistle was associated with increased indoor PM2.5 as well as indoor carbon monoxide 1-hr maximum (Table 4.49). Again, only indoor $PM_{2.5}$ remained elevated in the co-pollutant model for wheeze (Table 4.49.01). Nearly 24% of the women using traditional stoves reported wheeze while 0% of the women cooking with improved stoves reported the symptom. There did not appear to be a clear association between cookstove exposures and nasal stuffiness, runny nose, sneezing, and/or nasal itch although non-significant elevated odds ratios were reported when comparing the high quality to the high-mid quality stoves in the stove scale analyses (Table 4.50). The usual presence of a headache during cooking was associated with cookstove exposures (Table 4.51). Although not statistically significant, increased personal PM_{2.5}, indoor PM_{2.5}, and indoor carbon monoxide 1-hr maximum were associated with the presence of headache (Table 4.51). In the co-pollutant model for headache, only odds ratios for indoor carbon monoxide 1-hour maximum remained elevated (Table 4.51.01). The odds of cooking with a traditional stove was about five times as high among those reporting a headache during cooking as compared to those not reporting a headache (Table 4.51). The percentages of women reporting headache were 21.74%, 0%, 20.00%, and 54.17% across the four-level stove scale ranging from high quality to low quality stoves. Finally, although there was no association between personal PM_{2.5} and shortness of breath, there was a non-significant association between indoor PM_{2.5}, indoor carbon monoxide 1-hr maximum, and stove type and shortness of breath (Table 4.52). In the co-pollutant model for shortness of breath, only the odds ratios for indoor carbon monoxide 1-hour maximum remained elevated (Table 4.52.01). In addition, for the stove scale and shortness of breath analysis, increased odds ratios for the 3 lower quality categories of stoves were reported as compared to the high quality reference category; although no clear trend was observed (Table 4.52).

Mean measures of FEV₁, PEF, and CRP were examined among women reporting and not reporting current respiratory symptom severities (Tables 4.53-4.55). No clear pattern was observed for FEV₁ and respiratory symptoms (Table 4.53). Mean PEF appeared to be slightly elevated among women reporting increased respiratory symptom severity (Table 4.54). Differences were statistically significant for current throat irritation and cough (Table 4.54). Similarly, mean CRP levels were elevated among women reporting

increased respiratory symptom severity (with the exception of current shortness of breath) although statistically significant differences were observed only for current wheeze, throat irritation, and cough (Table 4.55).

	TOTAL			h	mproved St	toves	<u> </u>	uditional S	toves_	Spec	Spearman Correlation CoefficientsAgeWeightHeightWaistBAcirc.00101.00100.421.00380.790.171.00			ents
Personal characteristics	N	Mean	SD	N	Mean	SD	N	Mean	SD	Age	Weight	Height	Waist circ.	BMI
Age (years)	79	41.5	14.7	41	45.0	13.0	38	37.8	15.7	1.00				
Weight (lbs)	78	132.7	25.1	41	133.5	28.3	37	131.8	21.3	0.10	1.00			
Height (in)	79	59.5	2.4	41	59.0	2.1	38	60.0	2.7	-0.10	0.42	1.00		
Waist Circumference (in)	79	36.1	4.6	41	36.3	5.3	38	35.9	3.6	0.38	0.79	0.17	1.00	
BMI (kg/m^2)	78	26.3	4.5	41	26.9	5.2	37	25.6	3.5	0.15	0.89	0.02	0.79	1.00

Table 4.1. Personal characteristics and Spearman correlation coefficients of the study population; total population and stratified by stove type.

BMI, body mass index; SD, standard deviation

	TO	TAL	Improv	ed Stoves	Traditio	nal Stoves
Characteristics	N=79	Percent	N=41	Percent	N=38	Percent
Do you take any medications on a regu	lar basis	or as				
prescribed by a doctor?						
Yes	30	37.97	17	41.46	13	34.21
No	49	62.03	24	58.54	25	65.79
Do you take any anti-inflammatory or basis or as prescribed by a doctor?	heart med	lications on a	regular			
Yes	11	13.92	6	14.63	5	13.16
No	68	86.08	35	85.37	33	86.84
Have you had a cold or the flu during t	he past w	eek?				
Yes	18	23.08	6	14.63	12	32.43
No	60	76.92	35	85.37	25	67.57
Have you had sinus problems during the	ne past we	ek?				
Yes	6	7.69	3	7.32	3	8.11
No	72	92.31	38	92.68	34	91.89
Do you take vitamins or supplements of	on a regula	ar basis?	• •			
Yes	11	13.92	3	7.32	8	21.05
No	68	86.08	38	92.68	30	78.95
No. of eligible lung function maneuver	rs	00.00	00	2.00		10000
0	24	31.58	12	30.00	12	33 33
1	9	11.84	7	17.50	2	5.56
2	10	13.16	5	12.50	5	13.89
>2	33	43.42	16	40.00	17	47.22
Fish consumption						
Low	61	77.22	30	73.17	31	81 58
High	18	22.78	11	26.83	7	18.42
Have you had your menstrual period d	uring the	last 6		-0.00		101.12
months?						
Yes	51	64.56	24	58.54	27	71.05
No	28	35.44	17	41.46	11	28.95
Are you currently experiencing more t amount of stress?	han the us	sual				
Yes	32	40.51	19	46.34	13	34.21
No	47	59.49	22	53.66	25	65.79
Are you concerned that breathing smol	ke from th	ne fire in you	r home			
Yes	60	75.95	28	68.29	32	84.21
No	19	24.05	13	31.71	6	15 79
Village	17	21.05	15	51.71	Ŭ	15.17
Suvapa	29	36 71	16	60.98	13	65 79
Santa Lucia	50	63 29	25	39.02	25	34.21
Education	20	00.67	tur I	59.04	20	57.21
0 vrs	13	1646	7	17 07	6	15 79
0.5-5 vrs	35	44 30	16	39.02	19	50.00
>5 vrs	31	39.24	18	43.90	13	34 21
<i>>y</i> 18		39.24	18	43.90	13	34.21

Table 4.2. Characteristics of the study population; total population and stratified by stove type.

Table 4.2. (continued)

	ΤC	DTAL	Improv	ed Stoves	Traditio	nal Stoves
	N=79	Percent	N=41	Percent	N=38	Percent
Years in current home						
0-6 yrs	25	32.05	11	27.50	14	36.84
7-20 yrs	24	30.77	12	30.00	12	31.58
>20 yrs	29	37.18	17	42.50	12	31.58
Occupation						
Homemaker	52	65.82	27	65.85	25	65.79
Owns store/Vendor/Job outside of	27	34.18	14	34.15	13	34.21
home						
Prepares food or drink to sell						
Yes	17	21.52	11	26.83	6	15.79
No	62	78.48	30	73.17	32	84.21
Previous smoker						
No	75	94.94	38	92.68	37	97.37
Yes	4	5.06	3	7.32	1	2.63
Do others smoke in the house or						
Ves	25	31.65	15	36 50	10	26.32
No	23 54	68 35	26	63 41	28	73.68
Average meals cooked per week	54	08.55	20	05.41	20	75.00
-21	14	17 70	6	14 62	0	21.05
21	56	70.80	20	14.05	0 20	72.69
21	50	11 20	20	17.07	20	/ 5.00
>21	9	11.39	/	17.07	2	5.20
burning						
Less than 3 hours	11	13.92	4	9.76	7	18.42
3 hours or more	68	86.08	37	90.24	31	81.58
Concern that stove smoke causes		00100	• • •	20121		01.00
health problems						
No	19	24.05	13	31.71	6	15.79
Yes	60	75.95	28	68.29	32	84.21
No of people she cooks for						
<=6	38	54.29	16	43.24	22	66.67
>6	32	45.71	21	56.76	11	33.33
Would your day have been different						
if monitoring had not taken place?						
Yes	9	15.52	5	16.13	4	14.81
No	49	84.48	26	83.87	23	85.19

76

		TOTAI		In	nproved Si	oves	Traditional Stoves				
Kitchen & Cooking characteristics	N	Mean	SD	N	Mean	SD	N	Mean	SD		
Kitchen volume (cubic feet)	79	889.7	559.5	41	1049.9	542.4	38	717.0	531.7		
Number of years with current stove	79	5.6	7.2	41	2.4	1.4	38	9.0	9.2		
Hours typically spent cooking each day	78	5.8	3.0	41	6.1	2.7	37	5.6	3.3		
Hours fire burns on typical day	79	12.7	5.0	41	12.8	5.9	38	12.5	4.0		
Hours spent in room with fire burning on typical day	79	6.1	4.0	41	6.5	4.1	38	5.7	3.9		

Table 4.3. Mean kitchen and cooking characteristics for the study population; total population and stratified by stove type.

SD, standard deviation

	ТО	TAL	Impi Sta	roved oves	Tradit Sto	tional ves
Characteristic	N=79	Percent	N=41	Percent	N=38	Percent
Stove						
Improved stove	41	51.90				
Traditional stove	38	48.10				
Stove scale						
High quality	23	29.11	23	56.10	0	0.00
High-mid quality	17	21.52	17	41.46	0	0.00
Low-mid quality	15	18.99	1	2.44	14	36.84
Low quality	24	30.38	0	0.00	24	63.16
Type of kitchen						
Enclosed	72	91.14	40	97.56	32	84.21
Semi-open	7	8 86	1	2.44	6	15 79
Source Source	,	0.00	•	2.11	0	15.17
Separate building	12	15.19	8	19.51	4	10.53
Attached to main living area	64	81.01	31	75.61	33	86.84
Part of main living area	3	3.80	2	4.88	1	2.63
Pets in or around the house						
Yes	59	86.76	30	85.71	29	87.88
No	9	13.24	5	14.29	4	12.12
Material of kitchen roof						
Iron sheets	59	74.68	28	68.29	31	81.58
Tiles	13	16.46	8	19.51	5	13.16
Asbestos	2	2.53	0	0.00	2	5.26
Wood	5	6.33	5	12.20	0	0.00
No. of walls with eaves						
0 walls	16	20.25	11	26.83	5	13.16
1-2 walls	37	46.84	14	34.15	23	60.53
3-4 walls	26	32.91	16	39.02	10	26.32
No of windows in kitchen						
0	36	45.57	15	36.59	21	55.26
1	35	44 30	19	46 34	16	42.11
>1	8	10.13	7	17.07	1	2.63
Area of kitchen windows	0			11101	-	2.00
0	36	45.57	15	36.59	21	55.26
<700 sq. in	23	29.11	11	26.83	12	31.58
>700 sq. in	20	25.32	15	36 59	5	13.16
No of doors in kitchen	20	20.02	10	00.07	5	10.10
	5	6 33	1	2 44	4	10.53
1	36	45 57	12	29.27	24	63.16
	38	48 10	28	68 29	10	26.32
Chimney condition	50	40.10	20	00.27	10	20.52
No chimney	20	25 32	0	0.00	20	52 63
Poor	1/	17 72	1	2 11	13	34 21
Fairly good	8	10.13	2	7 37	5	13 16
Very good	37	46.84	3	90.24	0	0.00
10112000	51	TU.07	51	JU.2T		0.00

Table 4.4. Stove, kitchen, and cooking characteristics of the study population; total population and stratified by stove type.

Table 4.4. (continued)

	TOTAL Improved Stoves				Tradi Sta	tional ves
	N=79	Percent	N=41	Percent	N=38	Percent
Had stove longer than 6 months:						
Yes	70	88.61	37	90.24	33	13.16
No	9	11.39	4	9.76	5	86.84
Number of walls						
Less than 4 walls	8	10.13	1	2.44	7	18.42
4 walls	71	89.87	40	97.56	31	81.58
Primary material of walls						
Blocks/bricks	53	67.09	30	73.17	23	60.53
Wood	19	24.05	8	19.51	11	28.95
Iron sheets	7	8.86	3	7.32	4	10.53
Plastic used to start the fire						
Yes	17	24.64	8	22.22	9	27.27
No	52	75.36	28	77.78	24	72.73
Pitch pine used to start the fire						
Yes	64	92.75	33	91.67	31	93.94
No	5	7.25	3	8.33	2	6.06
Quality of fuel wood						
Dry	35	64.81	18	64.29	17	65.38
Damp	19	35.19	10	35.71	9	34.62
Owns second gas/electric stove						
Yes	16	20.25	11	26.83	5	13.16
No	63	79.75	30	73.17	33	86.84

Table 4.5. Air quality means, standard deviations, minimum measures, maximum measures, and geometric means for the total population and stratified by stove type.

			TO	TAL					mprov	ed Sto	ves				Tradition	nal Sto	ves	
Air Quality	N	Mean	<u>SD</u>	Min	Max	GM	N	Mean	SD	Min	Max	GM	N	Mean	SD	Min	Max	GM
$PM_{2.5}$, personal 8-hr average ($\mu g/m^3$)	58	133.5	114.9	20.7	679.8	101.9	30	73.6	34.0	20.7	138.5	65.3	28	197.7	135.5	38.4	679.8	164.2
$PM_{2.5}$, indoor 8-hr average ($\mu g/m^3$)	57	614.9	847.5	42.7	4835.4	329.5	30	266.3	240.2	42.7	902.1	178.7	27	1002.3	1089.4	59.8	4835.4	650.2
$PM_{2.5}$, outdoor 8-hr average (μ g/m ³)	49	282.3	313.3	14.0	1782.0	176.8	26	215.4	168,3	37.4	549.4	152.0	23	357.9	413.1	14.0	1782.0	209.7
Carbon monoxide, indoor 1-hr maximum (ppm)	54 n	7.9	11.2	0.1	54.0	2.0	28	1.8	3.2	0.1	11.9	0.5	26	14.3	13.1	1.5	54.0	9.5
Carbon dioxide, indoor average (ppm)	58)	329.8	62.5	245.0	618.0	324.9	31	297.0	29.9	245.0	370.0	295.5	27	367.4	69.1	295.0	618.0	362.2
Temperature, indoor average (°C)	58	26.0	3.4	19.6	33.9	25.7	31	25.9	3.7	19.6	33.3	25.6	27	26.0	3.1	19.7	33.9	25.9
Percent relative humidity, indoor average	58	61.8	12.4	35.4	90.0	60.6	31	61.9	12.7	44.1	85.2	60.7	27	61.6	12.2	35.4	90.0	60.5
Temperature, afternoon outdoor average (°C)	55	23.0	3.5	15.6	32.2	22.8	31	23.1	3.8	15.6	29.9	22.8	24	22.9	3.2	18.4	32.2	22.7

PM, particulate matter; SD, standard deviation; GM, geometric mean

08

Stove scale:		<u>High</u>	qualit	Y		High-m	iid qual	i <u>ty</u>		Low-m	id qua	lity		Low o	quality	
Air Quality	N	Mean	SD	GM	N	Mean	SD	GM	N	Mean	SD	GM	N	Mean	SD	GM
$PM_{2.5}$, personal 8-hr average (µg/m ³)	19	66.1	29.5	60.0	10	81.7	37.2	71.3	10	118.5	45.4	108.2	19	236.1	147.3	202.4
$PM_{2.5}$, indoor 8-hr average ($\mu g/m^3$)	19	218.2	211.4	149.1	10	294.0	214.1	214.3	10	531.7	232.5	484.9	18	1258.2	1259.9	779.3
Carbon monoxide, indoor 1-hr maximum (ppm)	18	0.7	2.3	0.2	9	3.4	3.5	2.4	10	6.5	5.2	5.0	17	18.6	14.1	13.7

Table 4.6. Air quality means, standard deviations, and geometric means for the study population across levels of the stove scale.

SD, standard deviation; GM, geometric mean

	Personal PM _{2.5}	Indoor PM _{2.5}	Indoor carbon monoxide 1-hr max
$PM_{2.5}$, personal 8-hr average ($\mu g/m^3$)	1.00		
$PM_{2.5}$, indoor 8-hr average ($\mu g/m^3$)	0.61	1.00	
Carbon monoxide, indoor 1-hr maximum (ppm)	0.67	0.69	1.00
$PM_{2.5}$, outdoor 8-hr average ($\mu g/m^3$)	0.38	0.28	0.004

Table 4.7. Spearman correlation coefficients for air quality measurements.

PM, particulate matter

Kitchen & Cooking characteristics	N	R^2
Stove type (Traditional vs. Improved)	58	0.40
Stove scale	58	0.51
Chimney condition	58	0.49
Total area of the kitchen windows	58	0.14
Number of kitchen walls	58	0.01
Number of kitchen doors	58	0.07
Volume of the kitchen (cubic feet)*	58	0.07
Number of walls with eave spaces	58	0.06
Primary material of the walls	58	0.04
Age of the stove	58	0.01
Hours the fire burns on a typical day	58	0.00
Time spent in the kitchen with the fire burning	58	0.00
Polynomial terms:		
Volume of the kitchen (squared)	58	0.08
Volume of the kitchen (cubed)	58	0.06
Age of the stove (squared)	58	0.00
Age of the stove (cubed)	58	0.00
Hours the fire burns on a typical day (squared)	58	0.00
Hours the fire burns on a typical day (cubed)	58	0.00
Time spent in the kitchen with the fire burning (squared)	58	0.00
Time spent in the kitchen with the fire burning (cubed)	58	0.01

Table 4.8. Univariate variation (R-square) in natural logarithm transformed personal $PM_{2.5}$ explained by kitchen and cooking characteristics.

*Natural logarithm transformed PM, particulate matter

Kitchen & Cooking characteristics	N	R^2
Stove type (Traditional vs. Improved)	57	0.33
Stove scale	57	0.40
Chimney condition	57	0.44
Total area of the kitchen windows	57	0.25
Number of kitchen walls	57	0.12
Number of kitchen doors	57	0.13
Volume of the kitchen (cubic feet)*	57	0.15
Number of walls with eave spaces	57	0.01
Primary material of the walls	57	0.01
Age of the stove	57	0.02
Hours the fire burns on a typical day	57	0.02
Polynomial terms:		
Volume of the kitchen (squared)	57	0.02
Volume of the kitchen (cubed)	57	0.00
Age of the stove (squared)	57	0.00
Age of the stove (cubed)	57	0.00
Hours the fire burns on a typical day (squared)	57	0.02
Hours the fire burns on a typical day (cubed)	57	0.02

Table 4.9. Univariate variation (R-square) in natural logarithm transformed indoor $PM_{2.5}$ explained by kitchen and cooking characteristics.

*Natural logarithm transformed PM, particulate matter

Table 4.10. Univariate variation (R-square) in natural logarithm transformed indoor carbon monoxide 1-hr maximum explained by kitchen and cooking characteristics.

	37	
Kitchen & Cooking characteristics	N	<u></u>
Stove type (Traditional vs. Improved)	54	0.54
Stove scale	54	0.79
Chimney condition	54	0.61
Total area of the kitchen windows	54	0.19
Number of kitchen walls	54	0.05
Number of kitchen doors	54	0.21
Volume of the kitchen (cubic feet)*	54	0.24
Number of walls with eave spaces	54	0.01
Primary material of the walls	54	0.03
Age of the stove	54	0.10
Hours the fire burns on a typical day	54	0.03
Polynomial terms:		
Volume of the kitchen (squared)	54	0.12
Volume of the kitchen (cubed)	54	0.06
Age of the stove (squared)	54	0.06
Age of the stove (cubed)	54	0.04
Hours the fire burns on a typical day (squared)	54	0.04
Hours the fire burns on a typical day (cubed)	54	0.04

*Natural logarithm transformed PM, particulate matter
Model No.	No. of Effects in Model	R-square	Ср	Variables in Model
1	2	0.51	1.4	Stove scale (4 categories)
2	3	0.54	-0.2	Stove scale (4 categories) Age of the stove (years)
3	5	0.57	0.9	Stove scale (4 categories) Age of the stove (years) Hours the fire burns on a typical day (hours) Time spent in the kitchen with the fire burning (hours)
4	5	0.60	*	Stove scale (4 categories) Age of the stove (years) Hours the fire burns on a typical day (hours) Interaction: Age of stove and Hours the fire burns

Table 4.11. R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed personal $PM_{2.5}$ levels utilizing the stove scale variable (n=58).

*Cp based on model set including interaction terms and, therefore, not comparable to models 1-3

PM, particulate matter

	N	R-square
Total population: final model	58	0.54
Second-hand smoke exposure		
No	41	0.54
Yes	17	0.68
Outdoor PM _{2.5} levels		
Less than 167 µg/m ³	24	0.54
Greater than 167 µg/m ³	24	0.64

Table 4.11.01. Variation in natural logarithm transformed personal $PM_{2.5}$ explained by the stove scale and the age of the stove and stratified by exposures to second-hand smoke and outdoor $PM_{2.5}$ levels.

PM, particulate matter

Model No.	No. of Effects in Model	R- square	Ср	Variables in Model
1	2	0.40	1.1	Stove type (Justa vs. Traditional)
2	3	0.45	-1.1	Stove type (Justa vs. Traditional) Age of the stove (years)
3	5	0.48	0.2	Stove type (Justa vs. Traditional) Age of the stove (years) Hours the fire burns on a typical day (hours) Number of kitchen walls (2 categories)
4	5	0.50	*	Stove type (Justa vs. Traditional) Age of the stove (years) Hours the fire burns on a typical day (hours) Interaction: Age of stove and Hours the fire burns

Table 4.12. R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed personal $PM_{2.5}$ levels utilizing the stove type variable (n=58).

*Cp based on model set including interaction terms and, therefore, not comparable to models 1-3

PM, particulate matter

Table 4.13. R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed indoor $PM_{2.5}$ levels utilizing the stove scale variable (n=57).

Model No.	No. of Effects in Model	R- square	Ср	Variables in Model
. 1	2	0.40	14.3	Stove scale (4 categories)
2	5	0.57	6.6	Stove scale (4 categories) Total area of the kitchen windows (3 categories) Number of kitchen walls (2 categories) Primary material of the kitchen walls (3 categories)
3	6	0.58	7.6	Stove scale (4 categories) Total area of the kitchen windows (3 categories) Number of kitchen walls (2 categories) Primary material of the kitchen walls (3 categories) Volume of the kitchen (Log of cubic feet)

PM, particulate matter

Table 4.13.01. Variation in natural logarithm transformed indoor $PM_{2.5}$ explained by the stove scale, the total area of the kitchen windows, the number of kitchen walls, and the primary material of the kitchen walls and stratified by exposures to second-hand smoke and outdoor $PM_{2.5}$ levels.

	N	R-square
Total population: final model	57	0.57
Second-hand smoke exposure		
No	41	0.63
Yes	16	0.59
Outdoor PM _{2.5} levels		
Less than 167 µg/m ³	24	0.68
Greater than 167 μ g/m ³	23	0.50

PM, particulate matter

Table 4.14. R-square and Mallow's Cp (Cp) as selection criteria for choosing a
parsimonious model that explains natural logarithm transformed indoor PM _{2.5} levels
utilizing the stove type variable (n=57).

Model No.	No. of Effects in Model	R- square	Ср	Variables in Model
1	2	0.33	8.7	Stove type (Justa vs. Traditional)
2	4	0.45	3.4	Stove type (Justa vs. Traditional) Total area of the kitchen windows (3 categories) Number of kitchen walls (2 categories)
3	6	0.51	4.4	Stove type (Justa vs. Traditional) Total area of the kitchen windows (3 categories) Number of kitchen walls (2 categories) Primary material of the kitchen walls (3 categories) Volume of the kitchen (Log of cubic feet)

PM, particulate matter

Model No.	No. of Effects in Model	R- square	Ср	Variables in Model
1	2	0.44	12.2	Chimney condition (4 categories)
2	6	0.60	6.6	Chimney condition (4 categories) Total area of the kitchen windows (3 categories) Number of kitchen walls (2 categories) Primary material of the kitchen walls (3 categories) Volume of the kitchen (Log of cubic feet)
3	7	0.61	7.7	Chimney condition (4 categories) Total area of the kitchen windows (3 categories) Number of kitchen walls (2 categories) Primary material of the kitchen walls (3 categories) Volume of the kitchen (Log of cubic feet) Age of the stove (years)

Table 4.15. R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed indoor $PM_{2.5}$ levels utilizing the chimney condition variable (n=57).

PM, particulate matter

Model No.	No. of Effects in Model	R- square	Ср	Variables in Model
1	2	0.79	15.0	Stove scale (4 categories)
2	4	0.85	2.4	Stove scale (4 categories) Volume of the kitchen (Log of cubic feet) Number of walls with eave spaces (3 categories)
3	5	0.86	3.5	Stove scale (4 categories) Volume of the kitchen (Log of cubic feet) Number of walls with eave spaces (3 categories) Hours the fire burns on a typical day (hours)

Table 4.16. R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed indoor carbon monoxide 1-hr maximum levels utilizing the stove scale variable (n=54).

Table 4.16.01. Variation in natural logarithm transformed indoor carbon monoxide 1-hr maximum explained by the stove scale, the natural log transformed volume of the kitchen, and the number of kitchen walls with eave spaces and stratified by exposure to second-hand smoke.

	N	R-square
Total population: final model	54	0.85
Second-hand smoke exposure		
No	39	0.85
Yes	15	0.89

Model No.	No. of Effects in Model	R- square	Ср	Variables in Model
1	2	0.54	7.7	Stove type (Justa vs. Traditional)
2	3	0.63	-1.4	Stove type (Justa vs. Traditional) Volume of the kitchen (Log of cubic feet)
3	4	0.65	-0.3	Stove type (Justa vs. Traditional) Volume of the kitchen (Log of cubic feet) Number of walls with eave spaces (3 categories)

Table 4.17. R-square and Mallow's Cp (Cp) as selection criteria for choosing a parsimonious model that explains natural logarithm transformed indoor carbon monoxide 1-hr maximum levels utilizing the stove type variable (n=54).

Table 4.18. Means and standard deviations (SL)) of health endpoints for the study population;	total population and stratified by stove
type.		

		1	TOTAL				Impr	oved S	toves			Tradi	tional S	toves	
Health Endpoint	N	Mean*	SD*	Min	Max	N	Mean*	SD*	Min	Max	N	Mean*	SD*	Min	Max
PEF, highest maneuver (L/min)	52	259.8	59.4	159.0	395.0	28	249.2	56.2	159.0	378.0	24	272.1	61.8	164.0	395.0
PEF, average of up to 3 maneuvers (L/min)	52	239.6	49.7	159.0	395.0	28	234.5	46.4	159.0	318.7	24	245.5	53.6	161.5	395.0
FEV ₁ , highest maneuver (L)	52	2.03	0.43	1.10	2.75	28	1.93	0.44	1.15	2.65	24	2.14	0.40	1.10	2.75
FEV ₁ , average of up to 3 maneuvers (L)	52	1.96	0.42	1.09	2.62	28	1.87	0.42	1.15	2.53	24	2.07	0.40	1.09	2.62
FEV ₁ , percent predicted	52	81.4	10.7	55.8	100.1	28	80.1	10.6	56.0	97.6	24	83.0	10.9	55.8	100.1
C-reactive protein (mg/L; dried blood spot scale)	71	4.2	4.4	0.02	41.0	36	5.2	4.0	0.2	41.0	35	3.3	4.8	0.02	40.6

*Geometric mean and geometric standard deviation used for C-reactive protein SD, standard deviation

(OD

1.1

m

Stove scale:		<u>High qua</u>	ality	E	ligh-mid g	uality		ow-mid q	<u>uality</u>		Low qual	ity
Health endpoint	N	Mean*	SD*	N	Mean*	SD*	N	Mean*	SD*	N	Mean*	SD*
PEF, highest maneuver (L/min)	14	261.6	44.6	13	226.0	52.9	8	289.8	70.1	17	270.1	62.3
FEV ₁ , highest maneuver (L)	14	2.03	0.42	13	1.82	0.46	8	2.09	0.33	17	2.17	0.43
C-reactive protein (mg/L; dried blood spot scale)	23	5.6	3.9	13	4.6	4.3	13	4.5	2.7	22	2.8	6.3

Table 4.19. Lung function and CRP means and standard deviations for the study population across levels of the stove scale.

*Geometric mean and geometric standard deviation used for C-reactive protein SD, standard deviation

	FEV ₁	PEF	CRP
FEV ₁ , highest maneuver (L)	1.00		
PEF, highest maneuver (L/min)	0.63	1.00	
C-reactive protein* (mg/L)	-0.32	-0.15	1.00

Table 4.20. Spearman correlation coefficients for three health endpoints among the study population.

	TC	TAL	Improv	ed Stoves	Traditio	nal Stoves
Symptoms	N=79	Percent	N=41	Percent	N=38	Percent
Do you usually have a cough?						
Yes	13	16.46	2	4.88	11	28.95
No	66	83.54	39	95.12	27	71.05
Do you usually bring up phlegm						
from your chest?						
Yes	10	12.66	3	7.32	7	18.42
No	69	87.34	38	92.68	31	81.58
Does your chest usually sound wheezy or whistling?						
Yes	9	11.39	0	0.00	9	23.68
No	70	88.61	41	100.00	29	76.32
Are you troubled by shortness of						
breath?						
Yes	53	67.09	25	60.98	28	73.68
No	26	32.91	16	39.02	10	26.32
Do you currently have nasal						
stuffiness, runny nose, sneezing and/or nasal itchiness?						
Yes	22	27.85	11	26.83	11	28.95
No	57	72.15	30	73.17	27	71.05
Do you usually develop a headache during cooking?						
Yes	21	26.58	5	12.20	16	42.11
No	58	73.42	36	87.80	22	57.89

Table 4.21. Reported symptoms among the study population; total population and stratified by stove type.

Stove scale:	High of Ste	quality ove	High quality	-mid Stove	Low quality	-mid Stove	Low qu Stov	ality 'e
Symptoms	N=23	Percent	N=17	Percent	N=15	Percent	N=24	Percent
Do you usually have a								
cough?								
Yes	1	4.35	1	5.88	4	26.67	7	29.17
No	22	95.65	16	94.12	11	73.33	17	70.83
Do you usually bring up phlegm from your chest?								
Yes	0	0.00	3	17.65	2	13.33	5	20.83
No	23	100.00	14	82.35	13	86.67	19	79.17
Does your chest usually sound wheezy or whistling?								
Yes	0	0.00	0	0.00	4	26.67	5	20.83
No	23	100.00	17	100.00	11	73.33	19	79.17
Are you troubled by shortness of breath?								
Yes	10	43.48	14	82.35	11	73.33	18	75.00
No	13	56.52	3	17.65	4	26.67	6	25.00
Do you currently have nasal stuffiness, runny								
nose, sneezing and/or nasal itchiness?								
Yes	5	21.74	6	35.29	5	33.33	6	25.00
No	18	78.26	11	64.71	10	66.67	18	75.00
Do you usually develop a headache during cooking?								
Yes	5	21.74	0	0.00	3	20.00	13	54.17
No	18	78.26	17	100.00	12	80.00	11	45.83

Table 4.22. Reported symptoms among the study population stratified by stove scale.

	N	Estimate	95% CI	P-value
Air Quality*				
$PM_{2.5}$, indoor (µg/m ³ ; 8-hr time-weighted average)	43	0.097	-0.017 to 0.210	0.0936
$PM_{2.5}$, personal ($\mu g/m^3$; 8-hr time-weighted average)	44	0.076	-0.039 to 0.190	0.1895
Carbon monoxide, indoor 1-hr maximum (ppm)	40	0.059	-0.092 to 0.209	0.4353
Stove (Traditional vs. Improved)	52	0.210	-0.026 to 0.446	0.0804
Stove Scale				
High quality	14	REF		
High-mid quality	13	-0.207	-0.534 to 0.120	0.2102
Low-mid quality	8	0.065	-0.311 to 0.441	0.7299
Low quality	17	0.146	-0.160 to 0.453	0.3423
Potential confounders				
Age (estimate per 10 year increase)	52	-0.213	-0.270 to -0.155	<.0001
Height (estimate per 3 inch increase)	52	0.252	0.117 to 0.386	0.0004
Waist Circumference (estimate per 5.5 inch increase)	52	-0.164	-0.302 to -0.027	0.0204
Second-hand smoke exposure (yes vs. no)	52	-0.208	-0.464 to 0.049	0.1105
Education level				
>5 yrs	10	REF		
0.5 – 5 yrs	22	-0.177	-0.422 to 0.069	0.1549
0 yrs	20	-0.526	-0.834 to -0.218	0.0012

Table 4.23. Univariate linear regression estimates and 95% confidence intervals (CI) for the association of air quality measures and Forced Expiratory Volume in 1 second (FEV_1 ; Liters) and potential confounders.

*Estimates for PM_{2.5} and carbon monoxide are per IQR increase: personal PM_{2.5} (106.1 μ g/m³), indoor PM_{2.5} (572.3 μ g/m³), and indoor carbon monoxide 1-hr max (4.62 ppm)

PM, particulate matter; REF, reference category

Table 4.24. Selected models of the association between personal $PM_{2.5}$ and Forced Expiratory Volume in 1 second (FEV₁; Liters): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (n=44)

Model	Personal PM _{2.5}	95% CI	P-value
$FEV_1 = Personal PM_{25}$	0.076	-0.039 to 0.190	0.1895
FEV ₁ = Personal PM _{2.5} , Age, Height, Waist circumference, Second-hand smoke, Education level	0.066	0.008 to 0.124	0.0264
Reduced model: FEV_1 = Personal PM _{2.5} , Age, Height	0.068	0.010 to 0.126	0.0220
\rightarrow Addition of Waist circumference	0.064	0.006 to 0.122	0.0302
\rightarrow Addition of Second-hand smoke exposure	0.068	0.011 to 0.126	0.0216
\rightarrow Addition of Education level	0.070	0.011 to 0.130	0.0214
\rightarrow Addition of Outdoor average temperature (n=41)	0.070	0.016 to 0.125	0.0132

*Estimates for personal PM_{2.5} are per IQR increase (106.1 μ g/m³); PM, particulate matter

	N	Beta coefficient	95% CI	P-value
Personal PM _{2.5}				
Low (reference)	13			
Medium	15	-0.008	-0.179 to 0.164	0.9292
High	16	0.194	0.029 to 0.359	0.0223

Table 4.24.01. Adjusted association between Personal $PM_{2.5}$ assessed as a categorical variable and Forced Expiratory Volume in 1 second (FEV₁; Liters) (adjusted for age and height) (N=44).

PM, particulate matter

	Pers	djusted estimate:	.*
· · · · · · · · · · · · · · · · · · ·	Beta	95% CI	P-value
	<u>coefficient</u>	0.010 0.100	0.0000
Total population $(N = 44)$	0.068	0.010 to 0.126	0.0220
Age**			
Less than 40 yrs (N=17)	0.057	-0.056 to 0.171	0.2956
40 yrs and older (N=27)	0.091	-0.028 to 0.209	0.1286
Second-hand smoke exposure			
No $(N=31)$	0.045	-0.013 to 0.103	0.1194
Yes (N=13)	0.145	-0.017 to 0.308	0.0732
Outdoor PM _{2.5} levels			
Less than 167 μ g/m ³ (N=15)	0.128	-0.249 to 0.506	0.4697
Greater than 167 μ g/m ³ (N=20)	0.038	-0.033 to 0.109	0.2702
Village of residence			
Santa Lucia (N=24)	0.067	0.013 to 0.121	0.0170
Suyapa (N=20)	0.068	-0.066 to 0.203	0.2967
Any medication intake	0.046	0.002 to 0.006	0.0660
No Medication $(N=26)$	0.040	-0.003 to 0.096	0.0000
Medication (N=18)	0.165	-0.015 10 0.382	0.0079
Outdoor average afternoon temperature			
Low Outdoor Temp. (N=21)	0.070	0.010 to 0.130	0.0244
High Outdoor Temp. (N=20)	0.030	-0.157 to 0.216	0.7414
Concern that stove smoke causes health problems			
No $(N = 10)$	0.243	-0.347 to 0.834	0.3520
$Y_{es}(N = 34)$	0.065	0.007 to 0.123	0.0303
	0.000		0.00000
Stove type			
Improved $(N = 23)$	-0.083	-0.434 to 0.268	0.6264
Traditional $(N = 21)$	0.060	-0.010 to 0.130	0.0881
Any current respiratory symptoms			
No symptoms (N=22)	0.084	-0.027 to 0.196	0.1298
Symptoms (N=22)	0.052	-0.022 to 0.126	0.1601
Length of time with current stove			
Less than 3 years (N=20)	0.070	-0.025 to 0.165	0.1363
3 or more years (N=24)	0.068	-0.025 to 0.161	0.1402

Table 4.24.02. Adjusted association between Personal $PM_{2.5}$ and Forced Expiratory Volume in 1 second (FEV₁; Liters) stratified by various factors.

*Adjusted for age and height; Estimates for personal $PM_{2.5}$ are per IQR increase (106.1 $\mu g/m^3$); **Adjusted for height; PM, particulate matter

	A Pers	djusted estimate: onal PM _{2.5} & FEV	·*
-	Beta coefficient	95% CI	P-value
Total population (N = 44)	0.068	0.010 to 0.126	0.0220
Women performing at least 2 successful lung function maneuvers (N=36)	0.061	-0.000 to 0.123	0.0517
Women having their current stove longer than 6 months (N=39)	0.080	0.025 to 0.134	0.0056
Women without a history of smoking (N=41)	0.064	0.005 to 0.123	0.0352
Women indicating that their day would not have been different without monitoring (N=36)	0.079	0.000 to 0.157	0.0487
Women not taking bronchodilator medication (N=43)	0.068	0.010 to 0.127	0.0231

Table 4.24.03. Adjusted association between Personal $PM_{2.5}$ and Forced Expiratory Volume in 1 second (FEV₁; Liters) among subgroups of participants.

*Adjusted for age and height; Estimates for personal $PM_{2.5}$ are per IQR increase (106.1 $\mu g/m^3$); PM, particulate matter

Table 4.25. Selected models of the association between indoor $PM_{2.5}$ and Forced Expiratory Volume in 1 second (FEV₁; Liters): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=43)

Model	Indoor	95% CI	P-value
	PM _{2.5}		
	<u>Coefficient*</u>		
FEV ₁ = Indoor PM _{2.5}	0.097	-0.017 to 0.210	0.0936
FEV ₁ = Indoor PM _{2.5} , Age, Height, Waist circumference, Second-hand smoke, Education level	0.009	-0.058 to 0.076	0.7890
Reduced model: FEV ₁ = Indoor PM _{2.5} , Age, Height	-0.0002	-0.066 to 0.066	0.9943
\rightarrow Addition of Waist circumference	-0.001	-0.066 to 0.065	0.9815
\rightarrow Addition of Second-hand smoke exposure	0.009	-0.057 to 0.075	0.7768
\rightarrow Addition of Education level	-0.0003	-0.069 to 0.069	0.9924
\rightarrow Addition of Outdoor average temperature (N= 41)	0.002	-0.062 to 0.065	0.9554

*Estimates for indoor PM_{2.5} are per IQR increase (572.3 μ g/m³); PM, particulate matter

	N	Beta coefficient	95% CI	P-value
Indoor PM _{2.5}				.
Low (reference)	13			
Medium	14	-0.006	-0.190 to 0.177	0.9446
High	16	0.076	-0.102 to 0.254	0.3945

Table 4.25.01. Adjusted association between indoor $PM_{2.5}$ assessed as a categorical variable and Forced Expiratory Volume in 1 second (FEV₁; Liters) (adjusted for age and height) (N=43).

PM, particulate matter

	A Ind	Adjusted estimate: loor PM25 & FEV1	*
	Beta coefficient	95% CI	P-value
Total population (N = 43)	-0.0002	-0.066 to 0.066	0.9943
Age**			
Less than 40 yrs (N=16)	-0.014	-0.076 to 0.048	0.6314
40 yrs and older (N=27)	0.265	0.020 to 0.509	0.0350
Second-hand smoke exposure			
No (N=31)	0.035	-0.045 to 0.116	0.3782
Yes (N=12)	-0.011	-0.168 to 0.145	0.8732
Outdoor PM _{2.5} levels			
Less than 167 μ g/m ³ (N=15)	0.057	-0.131 to 0.244	0.5204
Greater than 167 µg/m ³ (N=19)	-0.034	-0.106 to 0.038	0.3256
Village of residence			
Santa Lucia (N=24)	-0.005	-0.070 to 0.060	0.8832
Suyapa (N=19)	0.056	-0.139 to 0.250	0.5498
Amount of time typically spent in the room with the	fire burning		
Less than 3 hours (N=6)	-0.052	-0.285 to 0.181	0.4372
3 or more hours (N=37)	0.035	-0.053 to 0.122	0.4242
Any medication intake			
No Medication (N=25)	-0.012	-0.069 to 0.045	0.6704
Medication (N=18)	0.046	-0.141 to 0.234	0.6049
Outdoor average afternoon temperature			
Low Outdoor Temp. (N=21)	0.001	-0.071 to 0.073	0.9810
High Outdoor Temp. (N=20)	0.092	-0.168 to 0.352	0.4646
Concern that stove smoke causes health problems			
No $(N = 10)$	-0.010	-0.769 to 0.749	0.9753
Yes $(N = 33)$	-0.003	-0.069 to 0.063	0.9380
Stove type			
Improved $(N = 23)$	0.142	-0.105 to 0.389	0.2434
Traditional $(N = 21)$	-0.032	-0.110 to 0.046	0.4002
Any current respiratory symptoms			
No symptoms (N=21)	-0.007	-0.092 to 0.078	0.8705
Symptoms (N=22)	0.006	-0.112 to 0.125	0.9098
Length of time with current stove			
Less than 3 years (N=19)	-0.012	-0.114 to 0.090	0.8066
3 or more years (N=24)	0.036	-0.075 to 0.147	0.5078

Table 4.25.02. Adjusted association between indoor $PM_{2.5}$ and Forced Expiratory Volume in 1 second (FEV₁; Liters) stratified by various factors.

*Adjusted for age and height; Estimates for indoor $PM_{2.5}$ are per IQR increase (572.3 $\mu g/m^3$); **Adjusted for height; PM, particulate matter

	Adjusted estimate: Indoor PM _{2.5} & FEV ₁ *			
	Beta coefficient	95% CI	P-value	
Total population (N = 43)	-0.0002	-0.066 to 0.066	0.9943	
Women performing at least 2 successful lung function maneuvers (N=35)	-0.008	-0.078 to 0.062	0.8130	
Women having their current stove longer than 6 months (N=38)	0.008	-0.061 to 0.077	0.8159	
Women without a history of smoking (N=40)	-0.002	-0.069 to 0.064	0.9481	
Women indicating that their day would not have been different without monitoring (N=35)	-0.016	-0.081 to 0.050	0.6283	
Women not taking bronchodilator medication (N=42)	0.001	-0.066 to 0.069	0.9676	

Table 4.25.03. Adjusted association between Indoor $PM_{2.5}$ and Forced Expiratory Volume in 1 second (FEV₁; Liters) among subgroups of participants.

*Adjusted for age and height; Estimates for indoor $PM_{2.5}$ are per IQR increase (572.3 $\mu g/m^3$); PM, particulate matter

Table 4.26. Selected models of the association between indoor carbon monoxide 1-hr
maximum and Forced Expiratory Volume in 1 second (FEV1; Liters): crude association,
full model, reduced model, and reduced model with the addition of each variable
individually to evaluate potential confounding (N=40).

Model	Indoor Carbon Monoxide Coefficient*	95% CI	P-value
FEV ₁ = Indoor CO 1-hr max	0.059	-0.092 to 0.209	0.4353
FEV ₁ = Indoor CO 1-hr max, Age, Height, Waist circumference, Second-hand smoke, Education level	0.047	-0.023 to 0.116	0.1 79 4
Reduced model: FEV_1 = Indoor CO 1-hr max, Age, Height	0.024	-0.052 to 0.100	0.5222
\rightarrow Addition of Waist circumference	0.025	-0.046 to 0.097	0.4796
\rightarrow Addition of Second-hand smoke exposure	0.042	-0.033 to 0.116	0.2640
\rightarrow Addition of Education level	0.025	-0.053 to 0.103	0.5198
\rightarrow Addition of Outdoor average temperature (N= 38)	0.017	-0.056 to 0.090	0.6380

*Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

	N	Beta coefficient	95% CI	P-value
Indoor carbon monoxide 1-hr max				
Low (reference)	12			
Medium	13	-0.102	-0.286 to 0.082	0.2680
High	15	0.034	-0.137 to 0.206	0.6859

Table 4.26.01. Adjusted association between indoor carbon monoxide 1-hr maximum assessed as a categorical variable and Forced Expiratory Volume in 1 second (FEV₁; Liters) (adjusted for age and height) (N=40).

	Adjusted estimate: Indoor carbon monoxide 1-hr max & FFV.*			
	Beta	<u>8 FEV1</u> * 95% CI	P-value	
Total population (N = 40)	0.024	-0.052 to 0.100	0.5222	
Age**				
Less than 40 yrs (N=16)	-0.041	-0.132 to 0.049	0.3444	
40 yrs and older (N=24)	0.063	-0.119 to 0.245	0.4795	
Second-hand smoke exposure				
No (N=29)	0.013	-0.091 to 0.118	0.7955	
Yes (N=11)	0.075	-0.074 to 0.223	0.2735	
Outdoor PM _{2.5} levels				
Less than 167 μ g/m ³ (N=14)	0.100	-0.116 to 0.315	0.3280	
Greater than 167 μ g/m ³ (N=17)	-0.032	-0.116 to 0.052	0.4292	
Village of residence				
Santa Lucia (N=21)	-0.023	-0.120 to 0.073	0.6140	
Suyapa (N=19)	0.047	-0.074 to 0.169	0.4151	
Amount of time typically spent in the room with th	e fire burning			
Less than 3 hours (N=5)	-0.039	-1.653 to 1.576	0.8112	
3 or more hours (N=35)	0.058	-0.032 to 0.148	0.2009	
Any medication intake				
No Medication (N=23)	-0.003	-0.090 to 0.084	0.9413	
Medication (N=17)	0.053	-0.084 to 0.189	0.4213	
Outdoor average afternoon temperature				
Low Outdoor Temp. (N=18)	0.013	-0.091 to 0.117	0.7939	
High Outdoor Temp. (N=20)	0.012	-0.102 to 0.125	0.8332	
Concern that stove smoke causes health problems				
No $(N = 9)$	0.329	0.034 to 0.625	0.0350	
Yes $(N = 31)$	0.008	-0.084 to 0.100	0.8552	
Stove type				
Improved $(N = 21)$	-0.069	-0.393 to 0.254	0.6558	
Traditional ($N = 19$)	0.001	-0.101 to 0.103	0.9817	
Any current respiratory symptoms				
No symptoms (N=19)	0.021	-0.089 to 0.131	0.6884	
Symptoms (N=21)	-0.004	-0.128 to 0.120	0.9488	
Length of time with current stove				
Less than 3 years (N=18)	-0.056	-0.225 to 0.113	0.4904	
3 or more years (N=22)	0.063	-0.019 to 0.146	0.1225	

Table 4.26.02. Adjusted association between indoor carbon monoxide 1-hr maximum and Forced Expiratory Volume in 1 second (FEV₁; Liters) stratified by various factors.

*Adjusted for age and height; Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm); **Adjusted for height; PM, particulate matter

	Adjusted estimate: Indoor carbon monoxide 1-hr max & FEV ₁ *			
	Beta coefficient	95% CI	P-value	
Total population (N = 40)	0.024	-0.052 to 0.100	0.5222	
Women performing at least 2 successful lung function maneuvers (N=32)	0.011	-0.070 to 0.093	0.7789	
Women having their current stove longer than 6 months (N=36)	0.036	-0.041 to 0.112	0.3517	
Women without a history of smoking (N=38)	0.013	-0.074 to 0.100	0.7572	
Women indicating that their day would not have been different without monitoring (N=33)	0.0003	-0.082 to 0.082	0.9945	
Women not taking bronchodilator medication (N=39)	0.026	-0.052 to 0.103	0.5052	

Table 4.26.03. Adjusted association between Indoor carbon monoxide 1-hr maximum and Forced Expiratory Volume in 1 second (FEV₁; Liters) among subgroups of participants.

*Adjusted for age and height; Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

Model	Stove (Traditional vs. Improved) Coefficient	95% CI	P-value
FEV ₁ =Stove	0.210	-0.026 to 0.446	0.0804
FEV ₁ =Stove, Age, Height, Waist circumference, Second-hand smoke, Education level	0.066	-0.091 to 0.223	0.4031
Reduced model: FEV ₁ = Stove, Age, Height	0.046	-0.106 to 0.198	0.5434
\rightarrow Addition of Waist circumference	0.049	-0.102 to 0.200	0.5189
\rightarrow Addition of Second-hand smoke exposure	0.047	-0.107 to 0.200	0.5437
\rightarrow Addition of Education level	0.062	-0.094 to 0.219	0.4255

Table 4.27. Selected models of the association between stove type (Traditional vs. Improved) and Forced Expiratory Volume in 1 second (FEV₁; Liters): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=52).

	Ś	Adjusted estimate: tove type & FEV ₁ *		
	Beta coefficient	95% CI	P-value	
Total population (N = 52)	0.046	-0.106 to 0.198	0.5434	
Age**				
Less than 40 yrs (N=23)	0.007	-0.229 to 0.242	0.9532	
40 yrs and older (N=29)	0.151	-0.155 to 0.458	0.3188	
Second-hand smoke exposure				
No (N=36)	0.066	-0.103 to 0.235	0.4293	
Yes (N=16)	0.060	-0.322 to 0.442	0.7371	
Outdoor PM _{2.5} levels				
Less than 167 μ g/m ³ (N=15)	-0.032	-0.557 to 0.493	0.8958	
Greater than 167 µg/m ³ (N=21)	0.110	-0.087 to 0.306	0.2547	
Village of residence				
Santa Lucia (N=31)	-0.025	-0.233 to 0.184	0.8107	
Suyapa (N=21)	0.136	-0.082 to 0.354	0.2056	
Amount of time typically spent in the room with the fire burning				
Less than 3 hours (N=8)	0.056	-0.695 to 0.806	0.8471	
3 or more hours (N=44)	0.040	-0.130 to 0.210	0.6384	
Any medication intake				
No Medication (N=31)	0.042	-0.128 to 0.212	0.6169	
Medication (N=21)	0.030	-0.264 to 0.324	0.8340	
Outdoor average afternoon temperature				
Low Outdoor Temp. (N=21)	0.179	-0.012 to 0.369	0.0644	
High Outdoor Temp. (N=21)	0.070	-0.162 to 0.302	0.5316	
Concern that stove smoke causes health problems				
No (N = 12)	0.371	-0.120 to 0.862	0.1197	
Yes $(N = 40)$	0.006	-0.174 to 0.186	0.9494	
Any current respiratory symptoms				
No symptoms (N=26)	0.090	-0.139 to 0.318	0.4247	
Symptoms (N=26)	-0.049	-0.285 to 0.186	0.6686	
Length of time with current stove				
Less than 3 years (N=24)	0.155	-0.108 to 0.419	0.2322	
3 or more years (N=28)	-0.001	-0.208 to 0.205	0.9889	

Table 4.27.01. Adjusted association between stove type (Traditional vs. Improved) and Forced Expiratory Volume in 1 second (FEV₁; Liters) stratified by various factors.

*Adjusted for age and height; **Adjusted for height; PM, particulate matter

	Adjusted estimate: Stove type & FEV ₁ *			
	Beta coefficient	95% CI	P-value	
Total population (N = 52)	0.046	-0.106 to 0.198	0.5434	
Women performing at least 2 successful lung function maneuvers (N=43)	-0.025	-0.196 to 0.145	0.7651	
Women having their current stove longer than 6 months (N=47)	0.042	-0.121 to 0.205	0.6025	
Women without a history of smoking (N=49)	0.029	-0.128 to 0.186	0.7134	
Women indicating that their day would not have been different without monitoring (N=37)	0.090	-0.061 to 0.241	0.2347	
Women not taking bronchodilator medication (N=51)	0.044	-0.110 to 0.198	0.5654	

Table 4.27.02. Adjusted association between stove type (Traditional vs. Improved) and Forced Expiratory Volume in 1 second (FEV₁; Liters) among subgroups of participants.

*Adjusted for age and height

	Adjusted estimate: Stove type & FEV ₁ *			
	Beta coefficient	95% CI	P-value	
Total population (N = 52)	0.046	-0.106 to 0.198	0.5434	
Presence of kitchen windows				
Kitchens with windows (N=27)	-0.067	-0.294 to 0.160	0.5456	
Kitchens without windows (N=25)	0.047	-0.144 to 0.238	0.6164	
Kitchen volume				
Greater than 700 cu. ft. (N=30)	0.000	-0.213 to 0.214	0.9991	
Less than 700 cu. ft. (N=22)	0.184	-0.079 to 0.447	0.1583	
Women cooking in kitchens with eave spaces (N=41)	0.066	-0.108 to 0.241	0.4465	
Women cooking in kitchens with 4 walls (N=49)	0.067	-0.091 to 0.225	0.3961	
Women cooking in kitchens attached to or part of the main living area (N=46)	0.056	-0.109 to 0.221	0.4955	

Table 4.27.03. Adjusted association between stove type (Traditional vs. Improved) and Forced Expiratory Volume in 1 second (FEV_1 ; Liters) among various ventilation subgroups.

*Adjusted for age and height

.

Model	N	Stove Scale	Stove Scale	95% CI	P-value
		Variable	Coefficient		
FEV ₁ =Stove scale	14	High quality*			
	13	High-mid quality	-0.207	-0.534 to 0.120	0.2102
	8	Low-mid quality	0.065	-0.311 to 0.441	0.7299
	17	Low quality	0.146	-0.160 to 0.453	0.3423
FEV_1 = Stove scale, Age, Height,	14	High quality*			
Waist circumference, Second-hand	13	High-mid quality	-0.027	-0.257 to 0.203	0.8144
smoke, Education level	8	Low-mid quality	-0.041	-0.284 to 0.202	0.7344
	17	Low quality	0.129	-0.069 to 0.326	0.1960
Reduced model: FEV ₁ = Stove	14	High quality*			
scale, Age, Height	13	High-mid quality	-0.027	-0.232 to 0.178	0.7885
	8	Low-mid quality	-0.064	-0.297 to 0.169	0.5843
	17	Low quality	0.115	-0.075 to 0.306	0.2289
\rightarrow Addition of Waist	14	High quality*			
circumference	13	High-mid quality	-0.001	-0.212 to 0.211	0.9948
	8	Low-mid quality	-0.039	-0.277 to 0.198	0.7400
	17	Low quality	0.125	-0.067 to 0.316	0.1962
\rightarrow Addition of Second-hand	14	High quality*			
smoke exposure	13	High-mid quality	-0.019	-0.228 to 0.190	0.8553
	8	Low-mid quality	-0.067	-0.302 to 0.169	0.5703
	17	Low quality	0.121	-0.072 to 0.315	0.2130
\rightarrow Addition of Education level	14	High quality*			
	13	High-mid quality	-0.066	-0.283 to 0.151	0.5438
	8	Low-mid quality	-0.060	-0.296 to 0.177	0.6130
	17	Low quality	0.111	-0.082 to 0.304	0.2526

Table 4.28. Selected models of the association between stove scale and Forced Expiratory Volume in 1 second (FEV₁; Liters): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=52).

*Reference category

	<u> </u>	M	Adjusted estimate: Stove type & FEV,**		
	N	Stove Scale Variable	Beta coefficient	95% CI	P-value
Total population $(N = 52)$	14	High quality*			
	13	High-mid quality	-0.027	-0.232 to 0.178	0.7885
	8	Low-mid quality	-0.064	-0.297 to 0.169	0.5843
	17	Low quality	0.115	-0.075 to 0.306	0.2289
Age***					
Less than 40 yrs (N=23)	7	High quality*			~=
	4	High-mid quality	-0.094	-0.429 to 0.242	0.5647
	5	Low-mid quality	-0.182	-0.495 to 0.130	0.2364
	7	Low quality	0.098	-0.195 to 0.390	0.4915
40 yrs and older (N=29)	7	High quality*			
•	9	High-mid quality	-0.124	-0.526 to 0.279	0.5320
	3	Low-mid quality	0.115	-0.458 to 0.687	0.6832
	10	Low quality	0.134	-0.266 to 0.535	0.4952
Second-hand smoke exposure					
No (N=36)	11	High quality*			
	7	High-mid quality	-0.037	-0.268 to 0.193	0.7423
	7	Low-mid quality	-0.037	-0.271 to 0.197	0.7465
	11	Low quality	0.159	-0.045 to 0.362	0.1220
Yes (N=16)	3	High quality*			
	6	High-mid quality	0.114	-0.516 to 0.745	0.6945
	1	Low-mid quality	-0.059	-0.999 to 0.881	0.8917
	6	Low quality	0.178	-0.454 to 0.811	0.5440
Outdoor PM ₂₅ levels					
Less than 167 μ g/m ³ (N=15)	3	High quality*			
	5	High-mid quality	-0.152	-0.617 to 0.313	0.4779
	2	Low-mid quality	0.174	-0.404 to 0.752	0.5122
	5	Low quality	-0.040	-0.793 to 0.712	0.9065
Greater than 167 μ g/m ³ (N=21)	7	High quality*			
	4	High-mid quality	-0.008	-0.322 to 0.307	0.9586
	. 1	Low-mid quality	0.010	-0.518 to 0.537	0.9697
	9	Low quality	0.115	-0.124 to 0.353	0.3210

Table 4.28.01. Adjusted association between stove scale and Forced Expiratory Volume in 1 second (FEV₁; Liters) stratified by various factors.

*Reference category; **Adjusted for age and height; ***Adjusted for height; PM, particulate matter

Table 4.28.01. (continued)

<u>,</u>			Adjusted estimate: Stove scale & FEV ₁ **				
	N	Stove Scale Variable	Beta coefficient	95% CI	P-value		
Village of residence							
Santa Lucia (N=31)	7	High quality*					
	9	High-mid quality	0.031	-0.268 to 0.329	0.8340		
	6	Low-mid quality	-0.125	-0.424 to 0.173	0.3956		
	9	Low quality	0.078	-0.205 to 0.362	0.5754		
Suyapa (N=21)	7	High quality*					
	4	High-mid quality	-0.016	-0.334 to 0.301	0.9139		
	2	Low-mid quality	0.203	-0.230 to 0.636	0.3333		
	8	Low quality	0.166	-0.095 to 0.428	0.1959		
Amount of time typically spent in the room with the fire burning							
Less than 3 hours (N=8)	2	High quality*					
`` ,	1	High-mid quality	-0.360	-2.982 to 2.262	0.6144		
	1	Low-mid quality	-0.304	-2.807 to 2.200	0.6537		
	4	Low quality	-0.007	-1.730 to 1.716	0.9874		
3 or more hours (N=44)	12	High quality*					
	12	High-mid quality	0.015	-0.203 to 0.233	0.8895		
	7	Low-mid quality	-0.020	-0.274 to 0.235	0.8765		
	13	Low quality	0.133	-0.083 to 0.348	0.2199		
Any medication intake							
No Medication (N=31)	9	High quality*					
	6	High-mid quality	0.002	-0.243 to 0.246	0.9898		
	7	Low-mid quality	-0.054	-0.296 to 0.187	0.6457		
	9	Low quality	0.156	-0.060 to 0.372	0.1503		
Medication (N=21)	5	High quality*					
	7	High-mid quality	-0.031	-0.409 to 0.347	0.8642		
	1	Low-mid quality	-0.272	-0.979 to 0.435	0.4254		
	8	Low quality	0.069	-0.309 to 0.447	0.7009		
Outdoor average afternoon tem	pera	ture					
Low Outdoor Temp. (N=21)	8	High quality*					
	3	High-mid quality	0.057	-0.308 to 0.422	0.7438		
	1	Low-mid quality	0.013	-0.467 to 0.492	0.9553		
	9	Low quality	0.212	-0.008 to 0.432	0.0579		
High Outdoor Temp. (N=21)	6	High quality*					
	6	High-mid quality	-0.156	-0.428 to 0.116	0.2410		
	3	Low-mid quality	0.081	-0.264 to 0.425	0.6245		
	6	Low quality	0.016	-0.278 to 0.311	0.9071		

*Reference category **Adjusted for age and height

Table 4.28.01. (continued)

· · · · · · · · · · · · · · · · · · ·	•••••	· · · · · · · · · · · · · · · · · · ·	Adjusted estimate: Stove scale & FEV ₁ **		
		-			
	N	Stove Scale	Beta	95% CI	P-value
		Variable	coefficient		
Concern that stove smoke					
causes health problems					
No $(N = 12)$	6	High quality*			
	4	High-mid quality	0.150	-0.254 to 0.554	0.4090
	0	Low-mid quality	NA	NA	NA
	2	Low quality	0.457	-0.104 to 1.017	0.0955
Yes $(N = 40)$	8	High quality*			
	9	High-mid quality	-0.132	-0.400 to 0.136	0.3241
	8	Low-mid quality	-0.138	-0.408 to 0.131	0.3042
	15	Low quality	0.028	-0.210 to 0.267	0.8118
Any append pagning to my appendix	-				
Any current respiratory sympto No symptoms $(N-26)$	0 0	Uigh quality*			
No symptoms (N=20)	07	High mid quality	0.007	0 201 to 0 107	0 4074
	2	Low mid quality	-0.097	$-0.391 \ 10 \ 0.197$	0.4974
	с о	Low-mu quanty	0.009	-0.373 ± 0.390	0.9020
	0	Low quality	0.144	-0.140 10 0.434	0.3121
Symptoms (N=26)	6	High quality*			
	6	High-mid quality	0.027	-0.294 to 0.347	0.8641
	5	Low-mid quality	-0.190	-0.512 to 0.133	0.2345
	9	Low quality	0.047	-0.237 to 0.331	0.7319
Length of time with current stov	e o	TT! 1 11. de			
Less than 3 years (N=24)	9	High quality*			
	7	High-mid quality	-0.083	-0.392 to 0.225	0.5789
	0	Low-mid quality	NA	NA	NA
	8	Low quality	0.119	-0.181 to 0.420	0.4169
3 or more years (N=28)	5	High quality*			
-	6	High-mid quality	0.045	-0.292 to 0.382	0.7860
	8	Low-mid quality	-0.014	-0.305 to 0.277	0.9221
	9	Low quality	0.153	-0.144 to 0.277	0.2967

*Reference category **Adjusted for age and height
······································		• • • • •	· A	djusted estimate:	
		-	Sta	ve scale & FEV ₁ **	*
	N	Stove Scale	Beta	95% CI	P-value
		Variable	coefficient		
Total population $(N = 52)$	14	High quality*			
	13	High-mid quality	-0.027	-0.232 to 0.178	0.7885
	8	Low-mid quality	-0.064	-0.297 to 0.169	0.5843
	17	Low quality	0.115	-0.075 to 0.306	0.2289
Women performing at least 2	12	High quality*			
successful lung function	8	High-mid quality	0.097	-0.145 to 0.339	0.4208
maneuvers (N=43)	7	Low-mid quality	-0.088	-0.338 to 0.162	0.4792
	16	Low quality	0.106	-0.101 to 0.312	0.3062
Women having their current	12	High quality*		-	
stove longer than 6 months	13	High-mid quality	0.038	-0.175 to 0.250	0.7227
(N=47)	8	Low-mid quality	-0.010	-0.248 to 0.227	0.9309
	14	Low quality	0.157	-0.053 to 0.367	0.1392
Women having their current	5	High quality*			
stove at least 3 years (N=28)	6	High-mid quality	0.045	-0.292 to 0.382	0.7860
···· · · · · · · · · · · · · · · · · ·	8	Low-mid quality	-0.014	-0.305 to 0.277	0.9221
	9	Low quality	0.153	-0.144 to 0.277	0.2967
Women without a history of	12	High quality*			
smoking (N=49)	13	High-mid quality	-0.062	-0.280 to 0.155	0.5666
5g (1	8	Low-mid quality	-0.092	-0.335 to 0.151	0.4489
	16	Low quality	0.080	-0.124 to 0.283	0.4342
Women indicating that their day	13	High quality*			
would not have been different	7	High_mid quality	0.035	-0.184 to 0.255	0.7461
without monitoring (N-37)	4	Low-mid quality	0.095	-0.173 to 0.363	0.7401
without monitoring (14–57)	13	Low-mu quanty	0.093	-0.175 to 0.305	0.4742
	15	Low quanty	0.102	-0.077 10 0.282	0.2333
Women not taking	14	High quality*			
bronchodilator medication	13	High-mid quality	-0.028	-0.235 to 0.180	0.7895
(N=51)	8	Low-mid quality	-0.064	-0.300 to 0.173	0.5906
	16	Low quality	0.115	-0.080 to 0.309	0.2414

Table 4.28.02. Adjusted association between stove scale and Forced Expiratory Volume in 1 second (FEV₁; Liters) among subgroups of participants.

*Reference category

**Adjusted for age and height

- <u>112 - 122 - 122 - 122 - 123 - 125 - 125 - 125 - 125 - 125 - 125 - 125 - 125 - 125 - 125 - 125 - 125 - 125 - 1</u> 25 - 12			A	djusted estimate:	
			Sto	ve scale & FEV1**	k
	N	Stove Scale	Beta	95% CI	P-value
<u></u>		Variable	<i>coefficient</i>		
Total population $(N = 52)$	14	High quality*		-÷	
	13	High-mid quality	-0.027	-0.232 to 0.178	0.7885
	8	Low-mid quality	-0.064	-0.297 to 0.169	0.5843
	17	Low quality	0.115	-0.075 to 0.306	0.2289
Presence of kitchen windows					
Kitchens with windows (N=27)	10	High quality*			
	7	High-mid quality	0.019	-0.221 to 0.259	0.8680
	3	Low-mid quality	-0.386	-0.717 to -0.055	0.0245
	7	Low quality	0.060	-0.182 to 0.303	0.6099
Kitchens without windows (N=25)	4	High quality*			
	6	High-mid quality	-0.201	-0.517 to 0.115	0.1979
	5	Low-mid quality	-0.082	-0.400 to 0.236	0.5963
	10	Low quality	-0.017	-0.302 to 0.268	0.9013
Kitchen volume					
Greater than 700 cu. ft. (N=30)	12	High quality*			
	7	High-mid quality	0.223	-0.028 to 0.474	0.0785
	3	Low-mid quality	0.013	-0.342 to 0.368	0.9402
	8	Low quality	0.101	-0.139 to 0.400	0.3940
Less than 700 cu. ft. (N=22)	2	High quality*			
	6	High-mid quality	-0.298	-0.710 to 0.113	0.1442
	5	Low-mid quality	-0.119	-0.548 to 0.309	0.5630
	9	Low quality	0.117	-0.292 to 0.527	0.5521
Women cooking in kitchens with	8	High quality*			
eave spaces (N=41)	11	High-mid quality	0.049	-0.217 to 0.315	0.7115
	6	Low-mid quality	-0.007	-0.307 to 0.292	0.9599
	16	Low quality	0.173	-0.067 to 0.413	0.1518
Women cooking in kitchens with	14	High quality*			
4 walls (N=49)	13	High-mid quality	-0.022	-0.230 to 0.185	0.8299
	7	Low-mid quality	-0.052	-0.299 to 0.195	0.6724
	15	Low quality	0.141	-0.056 to 0.339	0.1559
Women cooking in kitchens	13	High quality*			
attached to or part of the main	10	High-mid quality	-0.016	-0.233 to 0.201	0.8844
living area (N=46)	7	Low-mid quality	-0.118	-0.363 to 0.126	0.3341
	16	Low quality	0.117	-0.078 to 0.312	0.2312

Table 4.28.03. Adjusted association between stove scale and Forced Expiratory Volume in 1 second (FEV_1 ; Liters) among various ventilation subgroups.

*Reference category

**Adjusted for age and height

	N	OR	95% CI
Air Quality*			
$PM_{2.5}$, indoor ($\mu g/m^3$; 8-hr time-weighted average)	43	0.81	0.44 to 1.50
$PM_{2.5}$, personal ($\mu g/m^3$; 8-hr time-weighted average)	44	0.29	0.08 to 1.01
Carbon monoxide, indoor 1-hr maximum (ppm)	40	0.64	0.28 to 1.45
Stove (Traditional vs. Improved)	52	0.41	0.13 to 1.30
Stove Scale			
High quality	14	REF	
High-mid quality	13	2.13	0.46 to 9.94
Low-mid quality	8	1.33	0.23 to 7.63
Low quality	17	0.29	0.06 to 1.47
Potential confounders			
Age (estimate per 10 year increase)	52	1.09	0.75 to 1.60
Height (estimate per 3 inch increase)	52	0.81	0.40 to 1.63
Waist Circumference (estimate per 5.5 inch increase)	52	1.78	0.88 to 3.61
Second-hand smoke exposure (yes vs. no)	52	1.77	0.54 to 5.83
Education level			
>5 yrs	10	REF	
0.5 - 5 yrs	22	1.55	0.45 to 5.37
0 yrs	20	1.24	0.26 to 5.91

Table 4.29. Univariate Odds Ratios (OR) and 95% confidence intervals (CI) for the association of air quality measures and dichotomized percent predicted Forced Expiratory Volume in 1 second (FEV₁; Liters) (less than 80% versus 80% or more) and potential confounders.

*Estimates for PM_{2.5} and carbon monoxide are per IQR increase: personal PM_{2.5} (106.1 μ g/m³), indoor PM_{2.5} (572.3 μ g/m³), and indoor carbon monoxide 1-hr max (4.62 ppm)

PM, particulate matter; REF, reference category

Table 4.30. Selected models for the association between personal $PM_{2.5}$ and percent
predicted Forced Expiratory Volume in 1 second (FEV1; less than 80% versus 80% or
more): crude association, full model, crude model with the addition of each variable
individually to evaluate potential confounding (n=44)

Model	Personal PM _{2.5} OR*	95% CI
FEV ₁ = Personal PM _{2.5}	0.29	0.08 to 1.01
FEV ₁ = Personal PM _{2.5} , Age, Height, Waist circumference, Second-hand smoke, Education level	0.17	0.03 to 1.05
Crude model: FEV ₁ = Personal PM _{2.5}		
\rightarrow Addition of Age	0.29	0.08 to 1.02
\rightarrow Addition of Height	0.29	0.08 to 1.00
\rightarrow Addition of Waist circumference	0.29	0.08 to 1.11
\rightarrow Addition of Second-hand smoke exposure	0.26	0.08 to 0.91
\rightarrow Addition of Education level	0.22	0.05 to 1.05
\rightarrow Addition of Outdoor average temperature (n=41)	0.33	0.10 to 1.14

*Estimates for personal $PM_{2.5}$ are per IQR increase (106.1 µg/m³) PM, particulate matter

Table 4.30.01. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of Personal $PM_{2.5}$ assessed as a categorical variable and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) (N=44).

	N	OR	95% CI
Personal PM _{2.5}			
Low (reference)	13		
Medium	15	1.33	0.30 to 5.91
High	16	0.17	0.03 to 1.05

PM, particulate matter

Table 4.30.02. Odds Ratios (OR) and 95% confidence intervals (CI) for the association
of Personal PM _{2.5} and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ;
less than 80% versus 80% or more) stratified by various factors.

OR* 95% CI	
Total population (N = 44) 0.29 0.08 to 1.01	
Age	
Less than 40 yrs (N=17) 0.45 0.08 to 2.44	
40 yrs and older (N=27) 0.22 0.04 to 1.14	
Second-hand smoke exposure	
$\begin{array}{c} N_{1} = (N_{1} + 12) \\ N_{2} = (N_{1} + 12) \\ N_{2} = (N_{1} + 12) \\ N_{3} = (N_{1} $	
105 (N=15) 0.28 0.00 to 1.51	
Outdoor PM ₂ - levels	
Less than 167 µg/m^3 (N=15) 0.36 0.03 to 3.93	
Greater than $167 \text{ µg/m}^3 (\text{N}=20)$ 0.47 0.11 to 2.04	_
Village of residence	
Santa Lucia (N=24) 0.27 0.04 to 1.71	
Suyapa (N=20) 0.40 0.05 to 3.05	4
Any medication intake	
No Medication (N=26) 0.45 0.11 to 1.78	i
Medication (N=18) 0.10 0.01 to 1.18	i
Outdoor average afternoon temperature	
Low Outdoor Temp. $(N=21)$ 0.19 0.03 to 1.50	1
High Outdoor Temp. $(N=20)$ 0.58 0.10 to 3.39	
Concern that stove smake causes health	
problems	
No $(N = 10)$ 0.65 0.05 to 8.36	,
Yes $(N = 34)$ 0.17 0.03 to 0.98	
Stove type	
Improved (N = 23) 7.86 0.39 to 157.7	4
Traditional (N = 21) 0.04 0.001 to 1.32	9
Any current respiratory symptoms	
No symptoms (N=22) 0.42 0.10 to 1.70)
Symptoms (N=22) 0.11 0.01 to 1.54	,
Length of time with current stove	
Less than 3 years (N=20) 0.49 0.14 to 1.75	i
3 or more years (N=24) 0.11 0.01 to 1.12	

*Estimates for personal PM_{2.5} are per IQR increase (106.1 μ g/m³) PM, particulate matter

127

Table 4.30.03. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of Personal $PM_{2.5}$ and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) among subgroups of participants.

	OR*	95% CI
Total population (N = 44)	0.29	0.08 to 1.01
Women performing at least 2 successful lung function maneuvers (N=36)	0.29	0.08 to 1.10
Women having their current stove longer than 6 months (N=39)	0.27	0.07 to 1.02
Women without a history of smoking (N=41)	0.29	0.08 to 1.06
Women indicating that their day would not have been different without monitoring (N=36)	0.35	0.10 to 1.19
Women not taking bronchodilator medication (N=43)	0.30	0.09 to 1.01

*Estimates for personal PM_{2.5} are per IQR increase (106.1 μ g/m³)

PM, particulate matter

Table 4.31. Selected models for the association between indoor $PM_{2.5}$ and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more): crude association, full model, crude model with the addition of each variable individually to evaluate potential confounding (n=43)

Model	Indoor PM _{2.5} OR*	95% CI
$FEV_1 = Indoor PM_{2.5}$	0.81	0.44 to 1.50
FEV ₁ = Indoor PM _{2.5} , Age, Height, Waist circumference, Second-hand smoke, Education level	0.88	0.45 to 1.72
Crude model: $FEV_1 = Indoor PM_{2.5}$		
\rightarrow Addition of Age	0.86	0.45 to 1.63
\rightarrow Addition of Height	0.81	0.44 to 1.51
\rightarrow Addition of Waist circumference	0.88	0.47 to 1.62
\rightarrow Addition of Second-hand smoke exposure	0.79	0.43 to 1.42
\rightarrow Addition of Education level	0.85	0.45 to 1.60
\rightarrow Addition of Outdoor average temperature (n=41)	0.86	0.48 to 1.55

*Estimates for indoor $PM_{2.5}$ are per IQR increase (572.3 µg/m³) PM, particulate matter

Table 4.31.01. Odds Ratio (OR) and 95% confidence intervals (CI) for the association of indoor $PM_{2.5}$ assessed as a categorical variable and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) (N=44).

	N	OR	95% CI
Indoor PM _{2.5}			
Low (reference)	13		
Medium	14	0.88	0.19 to 4.00
High	16	0.39	0.08 to 1.87

PM, particulate matter

Table 4.31.02. Odds Ratios (OR) and 95% confidence intervals (CI) for the association
of indoor PM _{2.5} and percent predicted Forced Expiratory Volume in 1 second (FEV ₁ ; less
than 80% versus 80% or more) stratified by various factors.

	OR*	95% CI
Total population (N = 43)	0.81	0.44 to 1.50
Age		
Less than 40 yrs (N=16)	1.02	0.56 to 1.87
40 yrs and older (N=27)	0.22	0.04 to 1.22
Second-hand smoke exposure		
No (N=31)	0.20	0.04 to 1.19
Yes (N=12)	1.31	0.50 to 3.42
Outdoor PM _{2.5} levels		
Less than 167 μ g/m ³ (N=15)	0.16	0.01 to 2.43
Greater than 167 µg/m ³ (N=19)	1.26	0.66 to 2.38
Village of residence		
Santa Lucia (N=24)	0.89	0.49 to 1.60
Suyapa (N=19)	NA	
Amount of time typically spent in the room with the fire burning		
Less than 3 hours (N=6)	1.42	0.53 to 3.82
3 or more hours (N=37)	0.29	0.08 to 1.10
Any medication intake		
No Medication (N=25)	1.19	0.63 to 2.24
Medication (N=18)	0.08	0.003 to 2.42
Outdoor average afternoon temperature		
Low Outdoor Temp. (N=21)	0.86	0.48 to 1.56
High Outdoor Temp. (N=20)	0.26	0.02 to 3.72
Concern that stove smoke causes health problems		
No $(N = 10)$	0.34	0.004 to 25.62
Yes $(N = 33)$	0.77	0.40 to 1.50
Stove type		
Improved ($N = 23$)	0.67	0.10 to 4.58
Traditional ($N = 20$)	1.25	0.63 to 2.49
Any current respiratory symptoms		
No symptoms (N=21)	1.17	0.60 to 2.27
Symptoms (N=22)	0.16	0.02 to 1.46
Length of time with current stove		
Less than 3 years (N=19)	0.97	0.52 to 1.82
3 or more years $(N-24)$	0.38	0.08 to 1.91

*Estimates for indoor $PM_{2.5}$ are per IQR increase (572.3 μ g/m³) PM, particulate matter

131

Table 4.31.03. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of indoor $PM_{2.5}$ and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) among subgroups of participants.

	OR*	95% CI
Total population (N = 43)	0.81	0.44 to 1.50
Women having their current stove longer than 6 months (N=38)	0.86	0.47 to 1.58
Women without a history of smoking (N=40)	0.86	0.47 to 1.54
Women indicating that their day would not have been different without monitoring (N=35)	0.93	0.53 to 1.64
Women not taking bronchodilator medication (N=42)	0.80	0.43 to 1.49

*Estimates for indoor $PM_{2.5}$ are per IQR increase (572.3 µg/m³) PM, particulate matter

132

Table 4.32. Selected models for the association between indoor carbon monoxide 1-hr maximum and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more): crude association, full model, crude model with the addition of each variable individually to evaluate potential confounding (n=40)

Model	Indoor carbon monoxide OR*	95% CI
$FEV_1 = Indoor carbon monoxide 1-hr max$	0.64	0.28 to 1.45
FEV ₁ = Indoor carbon monoxide 1-hr max, Age, Height, Waist circumference, Second-hand smoke, Education level	0.51	0.18 to 1.42
Crude model: FEV_1 = Indoor carbon monoxide 1-hr max		
\rightarrow Addition of Age	0.64	0.28 to 1.46
\rightarrow Addition of Height	0.64	0.28 to 1.45
\rightarrow Addition of Waist circumference	0.59	0.23 to 1.54
\rightarrow Addition of Second-hand smoke exposure	0.57	0.26 to 1.26
\rightarrow Addition of Education level	0.63	0.27 to 1.47
\rightarrow Addition of Outdoor average temperature (n=41)	0.72	0.31 to 1.65

*Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

Table 4.32.01. Odds Ratio (OR) and 95% confidence intervals (CI) for the association of indoor carbon monoxide 1-hr maximum assessed as a categorical variable and percent predicted Forced Expiratory Volume in 1 second (FEV_1 ; less than 80% versus 80% or more) (N=40).

	N	OR	95% CI
Indoor carbon monoxide 1-hr max			
Low (reference)	12		
Medium	13	2.33	0.46 to 11.81
High	15	0.50	0.09 to 2.86

Table 4.32.02. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of indoor carbon monoxide 1-hr maximum and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) stratified by various factors.

	OR*	95% CI
Total population (N = 40)	0.64	0.28 to 1.45
Age		
Less than 40 yrs $(N=16)$	1 45	0.52 to 4.09
40 vrs and older ($N=24$)	0.05	0.002 to 0.99
	0.00	0.002 10 0.00
Second-hand smoke exposure		
No (N=29)	0.43	0.10 to 1.88
Yes (N=11)	0.65	0.25 to 1.65
Outdoor PM ₂₅ levels		
Less than $167 \mu\text{g/m}^3$ (N=14)	0.45	0.10 to 1.99
Greater than 167 μ g/m ³ (N=17)	1.12	0.39 to 3.22
Village of residence		
Santa Lucia (N-21)	0.84	0.32 to 2.10
Sama Lucia $(N-10)$	0.04 ΝΔ	0.52 10 2.19
Suyapa (14–19)	INA	
Amount of time typically spent in the room with the		
fire burning	1.20	0.24 ± 0.499
Less than 5 hours $(N=3)$	1.29	0.34 10 4.88
5 of more nours (N=55)	0.32	0.08 10 1.29
Any medication intake		
No Medication (N=23)	1.18	0.37 to 3.80
Medication (N=17)	0.35	0.08 to 1.47
Outdoor average afternoon temperature		
Low Outdoor Temp. (N=18)	0.63	0.22 to 1.79
High Outdoor Temp. (N=20)	0.74	0.21 to 2.58
Concern that stays smalle course health problems		
Concern that stove smoke causes health problems N_0 (N = 0)	0.32	0.01 to 13.27
NO(1N-3) Vec (N - 31)	0.52	0.01 to 15.27
1es(N-51)	0.04	0.20 10 1.57
Stove type		
Improved $(N = 21)$	2.04	0.14 to 29.75
Traditional $(N = 19)$	0.86	0.28 to 2.64
Any current respiratory symptoms		
No symptoms (N=19)	0.69	0.25 to 1.93
Symptoms (N=21)	0.50	0.11 to 2.30
Length of time with current stove		
Less than 3 years $(N=18)$	1.78	0.53 to 6.05
3 or more years (N=22)	NA	

*Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm) PM, particulate matter Table 4.32.03. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of indoor carbon monoxide 1-hr maximum and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) among subgroups of participants.

	OR*	95% CI
Total population (N = 40)	0.64	0.28 to 1.45
Women performing at least 2 successful lung function maneuvers (N=32)	0.64	0.27 to 1.52
Women having their current stove longer than 6 months (N=36)	0.63	0.28 to 1.46
Women without a history of smoking (N=38)	0.70	0.30 to 1.65
Women indicating that their day would not have been different without monitoring (N=33)	0.75	0.32 to 1.78
Women not taking bronchodilator medication (N=39)	0.63	0.28 to 1.43

*Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

Model	Stove type (Traditional vs. Improved) OR	95% CI
$FEV_1 = Stove$	0.41	0.13 to 1.30
FEV ₁ = Stove, Age, Height, Waist circumference, Second-hand smoke, Education level	0.38	0.10 to 1.43
Crude model: FEV ₁ = Stove		
\rightarrow Addition of Age	0.42	0.13 to 1.34
\rightarrow Addition of Height	0.43	0.13 to 1.39
→ Addition of Waist circumference	0.39	0.12 to 1.28
\rightarrow Addition of Second-hand smoke exposure	0.41	0.13 to 1.32
\rightarrow Addition of Education level	0.41	0.13 to 1.30

Table 4.33. Selected models for the association between stove type (Traditional vs. Improved) and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more): crude association, full model, crude model with the addition of each variable individually to evaluate potential confounding (n=52)

Table 4.33.01. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove type (Traditional vs. Improved) and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) stratified by various factors.

· · · · · · · · · · · · · · · · · · ·	OR	95% CI
Total population (N = 52)	0.41	0.13 to 1.30
Age		
Less than 40 yrs (N=23)	0.86	0.16 to 4.47
40 yrs and older (N=29)	0.18	0.03 to 1.07
Casend hand smalls are		
Second-nand smoke exposure	0.24	0.08 + 1.44
$N_{0} = 10$ (N-16)	0.54	0.08 to 1.44
105 (11=10)	0.00	0.00 10 4.40
Outdoor PM _{2.5} levels		
Less than 167 μ g/m ³ (N=15)	0.63	0.07 to 5.35
Greater than 167 μ g/m ³ (N=21)	0.13	0.01 to 1.44
Village of residence		
Santa Lucia (N=31)	0.40	0.09 to 1.72
Suyapa (N=21)	NA	
A mount of time tunically grant in the		
Amount of time typically spent in the		
Less than 3 hours (N=8)	0.33	0.02 to 6.66
3 or more hours (N=44)	0.39	0.11 to 1.40
Any medication intake		
No Medication (N=31)	0.61	0.13 to 2.79
Medication (N=21)	0.25	0.04 to 1.56
Low Outdoor Temp (N=21)	0.16	0.02 to 1.07
High Outdoor Temp. $(N=21)$	0.10	0.02 to 1.07
	0.52	0.05 10 5.50
Concern that stove smoke causes health		
problems $N_0 (N - 12)$	NΔ	
Ves(N = 40)	0.37	0 10 to 1 36
10 (11 - 10)	0.57	0.10 10 1.30
Any current respiratory symptoms		
No symptoms (N=26)	0.33	0.06 to 1.78
Symptoms (N=26)	0.56	0.11 to 2.86
Length of time with current stove	0.15	
Less than 3 years (N=24)	0.43	0.07 to 2.81
3 or more years (N=28)	0.33	0.07 to 1.55

PM, particulate matter

Table 4.33.02. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove type (Traditional vs. Improved) and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) among subgroups of participants.

	OR	95% CI
Total population (N = 52)	0.41	0.13 to 1.30
Women performing at least 2 successful lung function maneuvers (N=43)	0.51	0.15 to 1.77
Women having their current stove longer than 6 months (N=47)	0.43	0.13 to 1.41
Women without a history of smoking (N=49)	0.44	0.14 to 1.42
Women indicating that their day would not have been different without monitoring (N=37)	0.31	0.07 to 1.28
Women not taking bronchodilator medication (N=51)	0.44	0.14 to 1.39

Table 4.33.03. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove type (Traditional vs. Improved) and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) among various ventilation subgroups.

	OR	95%CI
Total population (N = 52)	0.41	0.13 to 1.30
Presence of kitchen windows		
Kitchens with windows (N=27)	0.70	0.15 to 3.37
Kitchens without windows (N=25)	0.29	0.04 to 2.02
Kitchen volume		
Greater than 700 cu. ft. (N=30)	0.52	0.10 to 2.58
Less than 700 cu. ft. (N=22)	0.22	0.04 to 1.37
	0.00	
women cooking in kitchens with eave spaces (N=41)	0.33	0.09 to 1.19
Women cooking in kitchens with 4 walls	0.31	0.09 to 1.09
(N=49)		
	0.40	0.12 to 1.24
women cooking in kitchens attached to r_{1}	0.40	0.12 to 1.34
or part of the main nying area (N=40)		

Model	N	Stove scale variable	Stove scale OR	95% CI
$FEV_1 = Stove scale$	14	High quality*		
	13	High-mid quality	2.13	0.46 to 9.94
	8	Low-mid quality	1.33	0.23 to 7.63
	17	Low quality	0.29	0.06 to 1.47
$FEV_1 = Stove scale, Age, Height, Waist$	14	High quality*		
circumference, Second-hand smoke, Education	13	High-mid quality	1.37	0.20 to 9.12
level	8	Low-mid quality	1.22	0.17 to 8.69
	17	Low quality	0.19	0.03 to 1.19
Crude model: FEV ₁ = Stove scale	- <u></u>	· · · · ·		
\rightarrow Addition of Age	14	High quality*		
	13	High-mid quality	2.06	0.42 to 10.05
	8	Low-mid quality	1.35	0.24 to 7.74
	17	Low quality	0.28	0.06 to 1.46
\rightarrow Addition of Height	14	High quality*		
-	13	High-mid quality	2.13	0.46 to 9.94
	8	Low-mid quality	1.39	0.24 to 8.22
	17	Low quality	0.30	0.06 to 1.54
\rightarrow Addition of Waist circumference	14	High quality*		
	13	High-mid quality	1.53	0.30 to 7.88
	8	Low-mid quality	1.04	0.17 to 6.32
	17	Low quality	0.24	0.04 to 1.27
\rightarrow Addition of Second-hand smoke exposure	14	High quality*		
	13	High-mid quality	1.83	0.38 to 8.90
	8	Low-mid quality	1.43	0.24 to 8.31
	17	Low quality	0.25	0.05 to 1.35
\rightarrow Addition of Education level	14	High quality*		
	13	High-mid quality	2.23	0.45 to 11.07
	8	Low-mid quality	1.49	0.25 to 8.86
	17	Low quality	0.26	0.05 to 1.38
\rightarrow Addition of waist circumference and second-	14	High quality*		
hand smoke exposure	13	High-mid quality	1.33	0.25 to 7.15
mand onloke exposure	8	Low-mid quality	1.12	0.18 to 6.97
	17	Low mality	0.21	0.04 to 1.18

Table 4.34. Selected models for the association between stove scale and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more): crude association, full model, crude model with the addition of each variable individually to evaluate potential confounding (n=52)

*Reference category

	N	Stove Scale Variable	OR**	95%CI
Total population $(N = 52)$	14	High quality*		
	13	High-mid quality	1.33	0.25 to 7.15
	8	Low-mid quality	1.12	0.18 to 6.97
	17	Low quality	0.21	0.04 to 1.18
Age***				
Less than 40 yrs (N=23)	7	High quality*		
	4	High-mid quality	1.67	0.08 to 36.59
	5	Low-mid quality	1.77	0.13 to 24.64
	7	Low quality	0.32	0.03 to 3.82
40 yrs and older (N=29)	7	High quality*		
	9	High-mid quality	1.12	0.09 to 13.99
	3	Low-mid quality	0.32	0.01 to 8.71
	10	Low quality	0.09	0.01 to 1.49
Second-hand smoke exposure***				
No $(N=36)$	11	High quality*		
	7	High-mid quality	1.12	0.13 to 9.40
	7	Low-mid quality	0.66	0.09 to 5.01
	11	Low quality	0.08	0.01 to 0.95
Yes (N=16)	3	High quality*	NA	
	6	High-mid quality		
	1	Low-mid quality		
	6	Low quality		
Outdoor PM ₂₅ levels				
Less than 167 μ g/m ³ (N=15)	3	High quality*	NA	
	5	High-mid quality		
	2	Low-mid quality		
	5	Low quality		
Greater than 167 μ g/m ³ (N=21)	7	High quality*	NA	
	4	High-mid quality		
	1	Low-mid quality		
	9	Low quality		

Table 4.34.01. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove scale and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) stratified by various factors.

*Reference category; **Adjusted for waist circumference and second-hand smoke exposure; ***Adjusted for waist circumference; PM, particulate matter

	N	Stove Scale Variable	OR**	95%CI
Village of residence				
Santa Lucia (N=31)	7	High quality*		
	9	High-mid quality	0.92	0.07 to 11.53
	6	Low-mid quality	1.06	0.09 to 12.22
	9	Low quality	0.15	0.01 to 1.74
Suyapa (N=21)	7	High quality*	NA	
	4	High-mid quality		
	2	Low-mid quality		
	8	Low quality		
Amount of time typically spent in	the room	with the fire burning	(. •	
Less than 3 hours (N=8)	2	High quality*	NA	
	1	High-mid quality		
	1	Low-mid quality		
	4	Low quality		
3 or more hours (N=44)	12	High quality*		
	12	High-mid quality	1.33	0.23 to 7.84
	7	Low-mid quality	0.85	0.12 to 6.03
	13	Low quality	0.20	0.03 to 1.44
Any medication inteko				
No Medication (N=31)	0	High quality*		
No Medication (N=31)	9 6	High-mid quality	1.90	0.20 to 18.03
	7	Low-mid quality	1.50	0.20 to 13.03
	9	Low quality	0.25	0.02 to 3.15
Medication (N=21)	5	High quality*	NA	
Medication (11-21)	7	High-mid quality	1111	
	1	Low-mid quality		
	8	Low quality		
Outdoor average afternoon temp	erature			
Low Outdoor Temp. (N=21)	8	High quality*	NA	
	3	High-mid quality		
	1	Low-mid quality		
	9	Low quality		
High Outdoor Temp. (N=21)	6	High quality*	NA	
	6	High-mid quality		
	5	Low-mid quality		
	6	Low quality		

Table 4.34.01. (continued)

*Reference category; **Adjusted for waist circumference and second-hand smoke exposure

Table 4.34.01. (continued)

	1	Stove Scale Variable	OK**	95%CI
Concern that stove smoke causes health problems		,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,		
No $(N = 12)$	6	High quality*	NA	
· · · · ·	4	High-mid quality		
	0	Low-mid quality		
	2	Low quality		
Yes $(N = 40)$	8	High quality*		
	9	High-mid quality	1.31	0.16 to 10.85
	8	Low-mid quality	0.71	0.08 to 6.09
	15	Low quality	0.14	0.02 to 1.11
Any current respiratory symptoms				
No symptoms (N=26)	8	High quality*	NA	
	7	High-mid quality		
	3	Low-mid quality		
	8	Low quality		
Symptoms (N=26)	6	High quality*		
Symptoms (11–20)	6	High-mid quality	1.51	0.09 to 25.99
	5	Low-mid quality	2.11	0.15 to 29.32
	9	Low quality	0.19	0.01 to 3.08
Length of time with current stove				
Less than 3 years $(N=24)$	9	High quality*		
	7	High-mid quality	1.90	0.20 to 18.16
	Ó	Low-mid quality	NA	NA
	8	Low quality	0.61	0.07 to 5.65
3 or more years (N=28)	5	High quality*	NA	
5 or more years (11-20)	6	High-mid quality	1111	
	8	Low-mid quality		
	9 9	Low quality		

*Reference category; **Adjusted for waist circumference and second-hand smoke exposure

	N	Stove Scale Variable	OR**	95%CI
Total population $(N = 52)$	14	High quality*		
	13	High-mid quality	1.33	0.25 to 7.15
	8	Low-mid quality	1.12	0.18 to 6.97
	17	Low quality	0.21	0.04 to 1.18
Women performing at least 2	12	High quality*		
successful lung function maneuvers	8	High-mid quality	0.45	0.05 to 3.97
(N=43)	7	Low-mid quality	0.93	0.12 to 6.87
	16	Low quality	0.17	0.03 to 1.00
Women having their current stove	12	High quality*		
longer than 6 months (N=47)	13	High-mid quality	0.96	0.17 to 5.56
	8	Low-mid quality	0.83	0.13 to 5.38
	14	Low quality	0.19	0.03 to 1.18
Women without a history of smoking	12	High quality*		
(N=49)	13	High-mid quality	1.40	0.24 to 8.04
	8	Low-mid quality	1.21	0.18 to 8.08
	16	Low quality	0.25	0.04 to 1.44
Women indicating that their day	13	High quality*		
would not have been different without	7	High-mid quality	0.67	0.08 to 5.88
monitoring (N=37)	4	Low-mid quality	0.15	0.01 to 3.40
	13	Low quality	0.17	0.02 to 1.27
Women not taking bronchodilator	14	High quality*		
medication $(N=51)$	13	High-mid quality	1.33	0.25 to 7.16
	8	Low-mid quality	1.11	0.18 to 6.90
	16	Low quality	0.23	0.04 to 1.28

Table 4.34.02. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove scale and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) among subgroups of participants.

*Reference category; **Adjusted for waist circumference and second-hand smoke exposure

	N	Stove Scale	OR**	95%CI
		Variable		
Total population $(N = 52)$	14	High quality*		
	13	High-mid quality	1.33	0.25 to 7.15
	8	Low-mid quality	1.12	0.18 to 6.97
	17	Low quality	0.21	0.04 to 1.18
Presence of kitchen windows				
Kitchens with windows (N=27)	10	High quality*	NA	
	7	High-mid quality		
	3	Low-mid quality		
	7	Low quality		
Kitchens without windows (N=25)	4	High quality*		<u> </u>
	6	High-mid quality	2.97	0.15 to 59.02
	5	Low-mid quality	0.80	0.03 to 22.66
	10	Low quality	0.28	0.01 to 7.02
Kitchen volume				
Greater than 700 cu ft $(N=30)$	12	High quality*		
Greater than 700 cd. it. (1(=50)	7	High-mid quality	0.15	0.01 to 1.88
	3	Low-mid quality	0.19	0.01 to 1.00
	8	Low quality	0.22	0.02 to 2.12
Less than 700 cu. ft. (N=22)	2	High quality*	NA	
	6	High-mid quality		
	5	Low-mid quality		
	9	Low quality		
Women cooking in kitchens with	8	High quality*		
eave spaces (N=41)	11	High-mid quality	0.45	0.06 to 3.44
	6	Low-mid quality	0.52	0.05 to 5.07
	16	Low quality	0.10	0.01 to 0.76
Women cooking in kitchens with 4	14	High quality*		
walls (N=49)	13	High-mid quality	1.47	0.27 to 7.90
	7	Low-mid quality	0.86	0.13 to 5.81
	15	Low quality	0.17	0.03 to 1.09
Waman caaking in kitchens	13	High quality*		
attached to or part of the main	10	High-mid quality	1 34	0.23 to 7.85
living area (N=46)	7	Low-mid quality	1 39	0.21 to 9.35
······································	16	Low quality	0.23	0.04 to 1.27

Table 4.34.03. Odds Ratios (OR) and 95% confidence intervals (CI) for the association of stove scale and percent predicted Forced Expiratory Volume in 1 second (FEV₁; less than 80% versus 80% or more) among various ventilation subgroups.

*Reference category; **Adjusted for waist circumference and second-hand smoke exposure

	N	Estimate*	95% CI	P-value
Air Quality*				
$PM_{2.5}$, indoor ($\mu g/m^3$; 8-hr time-weighted average)	43	3.204	-12.958 to 19.366	0.6910
$PM_{2.5}$, personal (µg/m ³ ; 8-hr time-weighted average)	44	16.639	1.540 to 31.738	0.0316
Carbon monoxide, indoor 1-hr maximum (ppm)	40	-1.828	-22.813 to 19.158	0.8610
Stove (Traditional vs. Improved)	52	22.911	-9.960 to 55.782	0.1677
Stove Scale				
High quality	14	REF		
High-mid quality	13	-35.571	-79.718 to 8.575	0.1118
Low-mid quality	8	28.179	-22.620 to 78.977	0.2703
Low quality	17	8.487	-32.878 to 49.853	0.6818
Potential confounders				
Age (estimate per 10 year increase)	52	-13.823	-24.603 to -3.043	0.0130
Height (estimate per 3 inch increase)	52	17.188	-3.160 to 37.536	0.0960
Waist Circumference (estimate per 5.5 inch increase)	52	-5.223	-25.173 to 14.727	0.6013
Second-hand smoke exposure (yes vs. no)	52	-29.306	-64.529 to 5.918	0.1010
Education level				
>5 yrs	10	REF		
0.5 – 5 yrs	22	-8.241	-45.568 to 29.086	0.6592
0 yrs	20	-20.15	-66.941 to 26.641	0.3910

Table 4.35. Univariate linear regression estimates and 95% confidence intervals (CI) for the association of air quality measures and Peak Expiratory Flow (PEF; L/minute) and potential confounders.

*Estimates for PM_{2.5} and carbon monoxide are per IQR increase: personal PM_{2.5} (106.1 μ g/m³), indoor PM_{2.5} (572.3 μ g/m³), and indoor carbon monoxide 1-hr max (4.62 ppm)

PM, particulate matter; REF, reference category

Model	Personal PM _{2.5} Coefficient*	95% CI	P-value
PEF = Personal PM _{2.5}	16.639	1.540 to 31.738	0.1895
PEF = Personal PM _{2.5} , Age, Height, Waist circumference, Second-hand smoke, Education level	15.977	2.212 to 29.742	0.0241
Reduced model: PEF = Personal PM _{2.5} , Age, Height	16.006	2.712 to 29.299	0.0195
\rightarrow Addition of Waist circumference	16.494	3.020 to 29.968	0.0177
\rightarrow Addition of Second-hand smoke exposure	15.997	2.835 to 29.158	0.0185
\rightarrow Addition of Education level	15.653	1.971 to 29.336	0.0261
\rightarrow Addition of Outdoor average temperature (n=41)	17.733	5.110 to 30.355	0.0072

Table 4.36. Selected models for the association between personal $PM_{2.5}$ and Peak Expiratory Flow (PEF; L/minute): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (n=44)

*Estimates for personal $PM_{2.5}$ are per IQR increase (106.1 μ g/m³) PM, particulate matter

Table 4.36.01. Adjusted association between Personal $PM_{2.5}$ assessed as a categorical variable and Peak Expiratory Flow (PEF; L/minute) (adjusted for age and height) (N=44).

	N	Beta coefficient	95% CI	P-value
Personal PM _{2.5}		• • • •		
Low (reference)	13			
Medium	15	8.296	-34.477 to 51.069	0.6970
High	16	23.910	-17.258 to 65.077	0.2472

PM, particulate matter

		Adjusted estimate:	
	F	Personal PM2.5 & PEF*	
	Beta coefficient	95% CI	P-value
Total population (N = 44)	16.006	2.712 to 29.299	0.0195
Age**			
Less than 40 yrs (N=17)	14.067	-12.493 to 40.628	0.2732
40 yrs and older (N=27)	14.632	0.328 to 28.935	0.0454
Second-hand smoke exposure			
No (N=31)	16.255	1.035 to 31.475	0.0372
Yes (N=13)	14.916	-17.650 to 47.482	0.3272
Outdoor PM _{2.5} levels			
Less than 167 μ g/m ³ (N=15)	69.930	1.140 to 138.719	0.0469
Greater than 167 μ g/m ³ (N=20)	16.871	-1.883 to 35.624	0.0746
Village of residence			
Santa Lucia (N=24)	19.358	4.747 to 33.970	0.0120
Suyapa (N=20)	3.202	-28.452 to 34.855	0.8329
Any medication intake			
No Medication (N=26)	13.292	-0.967 to 27.552	0.0662
Medication (N=18)	18.908	-13.134 to 50.950	0.2263
Outdoor average afternoon temperature			
Low Outdoor Temp. (N=21)	21.137	7.710 to 34.624	0.0041
High Outdoor Temp. (N=20)	1.062	-41.853 to 43.977	0.9588
Concern that stove smoke causes health			
problems No $(N = 10)$	-1.493	-28.879 to 25.893	0.8982
Yes $(N = 34)$	16.118	1.282 to 30.955	0.0342
Stove type			
Improved $(N = 23)$	32.116	-40.290 to 104.521	0.3649
Traditional $(N = 21)$	18.173	-0.791 to 37.137	0.0592
Any current respiratory symptoms			
No symptoms (N=22)	11.738	-16.312 to 39.789	0.3909
Symptoms (N=22)	16.485	0.440 to 32.531	0.0446
Length of time with current stove			
Less than 3 years (N=20)	15.092	-2.918 to 33.101	0.0947
3 or more years (N=24)	19.457	-4.261 to 43.174	0.1025

Table 4.36.02. Adjusted association between Personal $PM_{2.5}$ and Peak Expiratory Flow (PEF; L/minute) stratified by various factors.

*Adjusted for age and height; Estimates for personal $PM_{2.5}$ are per IQR increase (106.1 $\mu g/m^3$); **Adjusted for height; PM, particulate matter

	Pe	Adjusted estimate: rsonal PM _{2.5} & PEF*	
	Beta coefficient	95% CI	P-value
Total population (N = 44)	16.006	2.712 to 29.299	0.0195
Women performing at least 2 successful lung function maneuvers (N=36)	14.407	0.502 to 28.312	0.0427
Women having their current stove longer than 6 months (N=39)	18.531	5.203 to 31.859	0.0078
Women without a history of smoking (N=41)	16.087	2.333 to 29.842	0.0231
Women indicating that their day would not have been different without monitoring (N=36)	14.540	-3.715 to 32.795	0.1145
Women not taking bronchodilator medication (N=43)	16.024	2.549 to 29.500	0.0210

Table 4.36.03. Adjusted association between Personal $PM_{2.5}$ and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants.

*Adjusted for age and height; Estimates for personal PM2.5 are per unit increase in IQR $(106.1 \,\mu\text{g/m}^3)$

PM, particulate matter

Table 4.37. Selected models of the association between indoor PM_{2.5} and Peak Expiratory Flow (PEF; L/minute): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=43)

Model	Indoor PM _{2.5} Coofficient*	95% CI	P-value
PEF = Indoor PM _{2.5}	3.204	-12.958 to 19.366	0.0936
PEF = Indoor PM _{2.5} , Age, Height, Waist circumference, Second-hand smoke, Education level	-3.343	-19.298 to 12.312	0.6732
Reduced model: PEF = Indoor PM _{2.5} , Age, Height	-4.785	-20.095 to 10.525	0.5310
\rightarrow Addition of Waist circumference	-4.741	-20.219 to 10.737	0.5389
\rightarrow Addition of Second-hand smoke exposure	-2.655	-17.959 to 12.648	0.7274
\rightarrow Addition of Education level	-5.394	-21.171 to 10.383	0.4928
\rightarrow Addition of Outdoor average temperature (N=41)	-3.461	-18.270 to 11.349	0.6384

*Estimates for indoor PM_{2.5} are per unit increase in IQR (572.3 μ g/m³) PM, particulate matter

.

Table 4.37.01. Adjusted association between indoor $PM_{2.5}$ assessed as a categorical variable and Peak Expiratory Flow (PEF; L/minute) (adjusted for age and height) (N=43).

	N	Beta coefficient	95% CI	P-value
Indoor PM _{2.5}				
Low (reference)	13			
Medium	14	10.953	-30.235 to 52.141	0.5935
High	16	38.060	-1.936 to 78.056	0.0616

PM, particulate matter

	Adjusted estimate: Indoor PM _{2.5} & PEF*		
-	Beta coefficient	95% CI	P-value
Total population (N = 43)	-4.785	-20.095 to 10.525	0.5310
Аде**			
Less than 40 yrs (N=16)	-12.349	-4.535 to 19.245	0.0842
40 yrs and older (N=27)	55.622	16.731 to 94.513	0.0070
Second-hand smoke exposure			
No (N=31)	10.537	-11.351 to 32.424	0.3320
Yes (N=12)	-16.697	-40.174 to 6.781	0.1396
Outdoor PM2 = levels			
Less than 167 $\mu g/m^3$ (N=15)	17.642	-21.618 to 56.901	0.3439
Greater than 167 μ g/m ³ (N=19)	-9.708	-31.336 to 11.920	0.3539
Village of residence			
Santa Lucia (N=24)	-5.973	-23.699 to 11.752	0.4902
Suyapa (N=19)	7.907	-37.080 to 52.895	0.7132
Amount of time typically spent in the room with the	fire burning		
Less than 3 hours (N=6)	-21.202	-52.732 to 10.329	0.1016
3 or more hours (N=37)	11.121	-9.794 to 32.036	0.2872
Any medication intake			
No Medication (N=25)	-7.259	-24.415 to 9.898	0.3889
Medication (N=18)	0.837	-27.700 to 29.374	0.9507
Outdoor average afternoon temperature			
Low Outdoor Temp. (N=21)	-6.212	-23.689 to 11.266	0.4636
High Outdoor Temp. (N=20)	58.443	6.355 to 110.531	0.0302
Concern that stove smoke causes health problems			
No $(N = 10)$	22.764	-0.570 to 46.098	0.0542
Yes $(N = 33)$	-7.998	-24.781 to 8.785	0.3378
Stove type			
Improved (N = 23)	55.216	8.627 to 101.806	0.0226
Traditional ($N = 20$)	-15.600	-36.584 to 5.385	0.1346
Any current respiratory symptoms			
No symptoms (N=21)	-11.419	-31.702 to 8.864	0.2512
Symptoms (N=22)	6.979	-19.887 to 33.845	0.5919
Length of time with current stove			
Less than 3 years (N=19)	-9.532	-28.028 to 8.964	0.2893
3 or more years (N=24)	15.643	-12.377 to 43.664	0.2579

Table 4.37.02. Adjusted association between indoor $PM_{2.5}$ and Peak Expiratory Flow (PEF; L/minute) stratified by various factors.

*Adjusted for age and height; Estimates for indoor $PM_{2.5}$ are per IQR increase (572.3 $\mu g/m^3$); **Adjusted for height; PM, particulate matter

	I	Adjusted estimate: ndoor PM _{2.5} & PEF*	
	Beta coefficient	95% CI	P-value
Total population (N = 43)	-4.785	-20.095 to 10.525	0.5310
Women performing at least 2 successful lung function maneuvers (N=35)	-6.789	-22.660 to 9.083	0.3897
Women having their current stove longer than 6 months (N=38)	-1.111	-18.024 to 15.802	0.8946
Women without a history of smoking (N=40)	-5.328	-21.008 to 10.352	0.4952
Women indicating that their day would not have been different without monitoring (N=35)	-9.175	-23.970 to 5.619	0.2153
Women not taking bronchodilator medication (N=42)	-4.775	-20.449 to 10.899	0.5411

Table 4.37.03. Adjusted association between Indoor $PM_{2.5}$ and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants.

*Adjusted for age and height; Estimates for indoor $PM_{2.5}$ are per IQR increase (572.3 $\mu g/m^3$); PM, particulate matter

Table 4.38. Selected models of the association between indoor carbon monoxide 1-hr maximum and Peak Expiratory Flow (PEF; L/minute): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=40).

Model	Indoor Carbon Monoxide Coefficient*	95% CI	P-value
PEF = Indoor carbon monoxide 1-hr max	-1.828	-22.813 to 19.158	0.8610
PEF = Indoor carbon monoxide 1-hr max, Age, Height, Waist circumference, Second-hand smoke, Education level	-1.375	-21.307 to 18.556	0.8891
Reduced model: PEF = Indoor carbon monoxide 1-hr	-4.355	-23.489 to 14.779	0.6471
max, Age, Height			
\rightarrow Addition of Waist circumference	-4.361	-23.786 to 15.063	0.6513
\rightarrow Addition of Second-hand smoke exposure	-0.876	-20.071 to 18.319	0.9267
\rightarrow Addition of Education level	-4.865	-24.399 to 14.668	0.6160
\rightarrow Addition of Outdoor average temperature (N= 38)	-3.726	-21.960 to 14.508	0.6803

*Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

	N	Beta coefficient	95% CI	P-value
Indoor carbon monoxide 1-hr max				
Low (reference)	12			
Medium	13	17.062	-30.473 to 64.596	0.4711
High	15	7.211	-37.167 to 51.589	0.7435

Table 4.38.01. Adjusted association between indoor carbon monoxide 1-hr maximum assessed as a categorical variable and Peak Expiratory Flow (PEF; L/minute) (adjusted for age and height) (N=40).
	Adjusted estimate:			
	Indoor Carbon Monoxide 1-hr max & PEF			
	Beta	95% CI	P-value	
	<u>coefficient</u>			
Total population $(N = 40)$	-4.355	-23.489 to 14.779	0.6471	
А де* *				
Less than 40 yrs (N=16)	-17.035	-42.230 to 7.160	0.1522	
40 yrs and older (N=24)	6.019	-27.207 to 39.245	0.7102	
Second hand smalle avagues				
Second-hand smoke exposure N_0 (N=29)	0 139	-31 021 to 31 200	0 0027	
NO(N-23) Ves (N-11)	0.159	-30.898 to 31.239	0.9927	
	0.101	-50.070 (0 51.217	0.7700	
Outdoor PM _{2.5} levels				
Less than 167 μ g/m ³ (N=14)	22.970	-25.869 to 71.810	0.3193	
Greater than 167 µg/m ³ (N=17)	-13.503	-46.316 to 19.309	0.3901	
Village of residence				
Santa Lucia (N=21)	14.597	-45.000 to 16.593	0.3442	
Suyapa (N=19)	2.869	-26.947 to 32.685	0.8403	
A mount of time typically spont in the room with the f	ire hurning			
Less than 3 hours (N-5)	-21 505	-285 862 to 242 852	0.4895	
3 or more hours (N=35)	9 187	-14 962 to 33 335	0.4075	
	2.107	11002 10 001000	0.1107	
Any medication intake				
No Medication (N=23)	-11.548	-42.239 to 19.144	0.4407	
Medication (N=17)	6.013	-15.495 to 27.520	0.5563	
Outdoor average afternoon temperature				
Low Outdoor Temp. (N=18)	-9.535	-39.423 to 20.353	0.5050	
High Outdoor Temp. (N=20)	1.996	-26.083 to 30.074	0.8821	
Concern that stove smoke causes health problems				
No $(N = 9)$	0.455	-48 442 to 49 352	0.9818	
Yes (N = 31)	-8.367	-32.936 to 16.203	0.4907	
Stove type	52.002	17.000 (105.50)	0 1210	
Improved (N = 21) The life $= 1 \text{ (N} = 10)$	53.803	-1/.899 to 125.506	0.1318	
1 raditional (N = 19)	-19.173	-47.215 to 8.809	0.1000	
Any current respiratory symptoms				
No symptoms (N=19)	-15.111	-43.962 to 13.740	0.2818	
Symptoms (N=22)	-0.205	-32.035 to 31.626	0.9893	
Length of time with current stove				
Less than 3 years (N=18)	-16.148	-48.379 to 16.084	0.3008	
3 or more years (N=22)	5.955	-17.811 to 29.721	0.6050	

Table 4.38.02. Adjusted association between indoor carbon monoxide 1-hr maximum and Peak Expiratory Flow (PEF; L/minute) stratified by various factors.

*Adjusted for age and height; Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm); **Adjusted for height

		Adjusted estimate:	
	Indoor carb	on monoxide 1-hr ma.	x & PEF*
	Beta	95% CI	P-value
	coefficient		
Total population (N = 40)	-4.355	-23.489 to 14.779	0.6471
Women performing at least 2 successful lung function maneuvers (N=32)	-8.064	-28.310 to 12.182	0.4215
Women having their current stove longer than 6 months (N=36)	-0.610	-21.178 to 19.958	0.9522
Women without a history of smoking (N=38)	-10.033	-31.487 to 11.420	0.3486
Women indicating that their day would not have been different without monitoring (N=33)	-10.651	-31.010 to 9.708	0.2935
Women not taking bronchodilator medication (N=39)	-4.264	-23.741 to 15.214	0.6595

Table 4.38.03. Adjusted association between Indoor carbon monoxide 1-hr maximum and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants.

*Adjusted for age and height; Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

Model	Stove (Traditional vs. Improved) Coefficient	95% CI	P-value
PEF =Stove	22.911	-9.960 to 55.782	0.1677
PEF =Stove, Age, Height, Waist circumference,	11.826	-22.530 to 46.182	0.4915
Second-hand smoke, Education level			
Reduced model: PEF = Stove, Age, Height	12.715	-19.929 to 45.358	0.4374
\rightarrow Addition of Waist circumference	12.599	-20.396 to 45.594	0.4462
\rightarrow Addition of Second-hand smoke exposure	12.970	-19.630 to 45.570	0.4275
\rightarrow Addition of Education level	11.662	-22.312 to 45.636	0.4931

Table 4.39. Selected models of the association between stove type (Traditional vs. Improved) and Peak Expiratory Flow (PEF; L/minute): crude associations, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=52).

		Adjusted estimate: Stove type & PEF*	
	Beta coefficient	95% CI	P-value
Total population (N = 52)	12.715	-19.929 to 45.358	0.4374
Age**			
Less than 40 yrs (N=23)	7.994	-40.323 to 56.311	0.7336
40 yrs and older (N=29)	22.954	-28.106 to 74.014	0.3639
Second-hand smoke exposure			
No (N=36)	15.970	-25.146 to 57.085	0.4347
Yes (N=16)	18.753	-40.096 to 77.603	0.5007
Outdoor PM2 c levels			
Less than 167 $\mu g/m^3$ (N=15)	-12.795	-125.149 to 99.560	0.8067
Greater than $167 \ \mu g/m^3$ (N=21)	27.592	-28.671 to 83.854	0.3153
Village of residence			
Santa Lucia (N=31)	26.439	-18.660 to 71.538	0.2395
Suyapa (N=21)	-1.661	-55.058 to 51.736	0.9484
Amount of time typically spent in the room with the	e fire burning		
Less than 3 hours (N=8)	-9.520	-157.697 to 138.657	0.8671
3 or more hours (N=44)	20.558	-16.455 to 57.571	0.2683
Any medication intake			
No Medication (N=31)	22.849	-18.374 to 64.072	0.2654
Medication (N=21)	-7.850	-52.354 to 36.653	0.7144
Outdoor average afternoon temperature			
Low Outdoor Temp. (N=21)	42.542	-4.967 to 90.051	0.0760
High Outdoor Temp. (N=21)	-10.244	-65.603 to 45.114	0.7011
Concern that stove smoke causes health problems			
No $(N = 12)$	-11.453	-59.274 to 36.368	0.5958
Yes $(N = 40)$	12.744	-27.557 to 53.045	0.5254
Any current respiratory symptoms			
No symptoms (N=26)	-17.293	-69.088 to 34.502	0.4959
Symptoms (N=26)	42.627	-3.932 to 89.186	0.0708
Length of time with current stove			
Less than 3 years (N=24)	17.702	-34.183 to 69.587	0.4849
3 or more years (N=28)	10.020	-37.083 to 57.123	0.6646

Table 4.39.01. Adjusted association between stove type (Traditional vs. Improved) and Peak Expiratory Flow (PEF; L/minute) stratified by various factors.

*Adjusted for age and height; **Adjusted for height

		Adjusted estimate: Stove type & PEF*	
	Beta coefficient	95% CI	P-value
Total population (N = 52)	12.715	-19.929 to 45.358	0.4374
Women performing at least 2 successful lung function maneuvers (N=43)	-5.532	-40.162 to 29.098	0.7484
Women having their current stove longer than 6 months (N=47)	19.262	-16.663 to 55.187	0.2856
Women without a history of smoking (N=49)	11.729	-22.152 to 45.609	0.4892
Women indicating that their day would not have been different without monitoring (N=37)	19.558	-15.311 to 54.427	0.2620
Women not taking bronchodilator medication (N=51)	12.423	-20.666 to 45.511	0.4538

Table 4.39.02. Adjusted association between stove type (Traditional vs. Improved) and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants.

*Adjusted for age and height

		Adjusted estimate: Stove type & PEF*	
	Beta coefficient	95% CI	P-value
Total population (N = 52)	12.715	-19.929 to 45.358	0.4374
Presence of kitchen windows			
Kitchens with windows (N=27)	-19.058	-57.500 to 19.384	0.3158
Kitchens without windows (N=25)	31.176	-26.030 to 88.382	0.2698
Kitchen volume			
Greater than 700 cu. ft. (N=30)	48.591	10.477 to 86.705	0.0145
Less than 700 cu. ft. (N=22)	-13.796	-76.315 to 48.723	0.6485
Women cooking in kitchens with eave spaces (N=41)	23.794	-14.551 to 62.139	0.2165
Women cooking in kitchens with 4 walls (N=49)	16.733	-16.093 to 49.558	0.3101
Women cooking in kitchens attached to or part of the main living area (N=46)	23.643	-11.284 to 58.570	0.1792

Table 4.39.03. Adjusted association between stove type (Traditional vs. Improved) and Peak Expiratory Flow (PEF; L/minute) among various ventilation subgroups.

*Adjusted for age and height

Table 4.40. Selected models of the association between stove scale and Peak Expiratory
Flow (PEF; L/minute): crude association, full model, reduced model, and reduced model
with the addition of each variable individually to test for potential confounding (N=52).

Model	N	Stove Scale	Stove Scale	95% CI	P-value
		Variable	Coefficient		
PEF =Stove scale	14	High quality*			
	13	High-mid quality	-35.571	-79.718 to 8.575	0.1118
	8	Low-mid quality	28.179	-22.620 to 78.977	0.2703
	17	Low quality	8.487	-32.878 to 49.853	0.6818
PEF =Stove scale, Age, Height,	14	High quality*			
Waist circumference, Second-	13	High-mid quality	-27.365	-77.759 to 23.029	0.2794
hand smoke, Education level	8	Low-mid quality	18.327	-35.016 to 71.671	0.4919
	17	Low quality	7.276	-36.039 to 50.590	0.7363
Reduced model: PEF = Stove	14	High quality*			
scale, Age, Height	13	High-mid quality	-26.360	-70.395 to 17.675	0.2344
	8	Low-mid quality	21.208	-28.891 to 71.307	0.3986
	17	Low quality	6.608	-34.318 to 47.534	0.7466
\rightarrow Addition of Waist	14	High quality*			
circumference	13	High-mid quality	-29.108	-74.926 to 16.710	0.2073
	8	Low-mid quality	18.705	-32.861 to 70.270	0.4688
	17	Low quality	5.667	-35.801 to 47.136	0.7844
\rightarrow Addition of Second-hand	14	High quality*			
smoke exposure	13	High-mid quality	-23.627	-68.356 to 21.102	0.2930
*	8	Low-mid quality	20.238	-30.129 to 70.605	0.4226
	17	Low quality	8.508	-32.856 to 49.871	0.6807
\rightarrow Addition of Education level	14	High quality*			
· · · · · · · · · · · · · · · · · · ·	13	High-mid quality	-27.479	-74.811 to 19.853	0.2483
	8	Low-mid quality	21.696	-29.874 to 73.266	0.4011
	17	Low quality	6.258	-35.774 to 48.290	0.7656

*Reference category

<u></u>		- ··· · ·	S	Adjusted estimate: Stove scale & PEF**	
	N	Stove Scale Variable	Beta coefficient	95% CI	P-value
Total population (N = 52)	14	High quality*			
`	13	High-mid quality	-26.360	-70.395 to 17.675	0.2344
	8	Low-mid quality	21.208	-28.891 to 71.307	0.3986
	17	Low quality	6.608	-34.318 to 47.534	0.7466
Age***					
Less than 40 yrs (N=23)	7	High quality*			
- ·	4	High-mid quality	-5.897	-81.334 to 69.539	0.8714
	5	Low-mid quality	1.988	-68.365 to 72.341	0.9533
	7	Low quality	9.019	-56.800 to 74.838	0.7767
40 yrs and older (N=29)	7	High quality*			
	9	High-mid quality	-46.700	-107.050 to 13.650	0.1233
	3	Low-mid quality	60.287	-25.532 to 149.106	0.1600
	10	Low quality	2.688	-57.300 to 62.676	0.9271
Second-hand smoke exposure					
No (N=36)	11	High quality*			
	7	High-mid quality	-29.208	-86.279 to 27.864	0.3043
	7	Low-mid quality	20.070	-37.926 to 78.066	0.4852
	11	Low quality	17.806	-32.678 to 68.290	0.4769
Yes (N=16)	3	High quality*			
	6	High-mid quality	8.025	-89.445 to 105.494	0.8581
	1	Low-mid quality	58.292	-87.128 to 203.712	0.3928
	6	Low quality	18.976	-78.811 to 116.763	0.6746
Outdoor PM levels					
Less than $167 \mu g/m^3$ (N-15)	3	High quality*			
Exist that $107 \ \mu g/m^2 (14-15)$	5	High-mid quality	-24 157	-120 423 to 72 109	0 5841
	2	Low-mid quality	57 562	-62 133 to 177 258	0.3049
	5	Low quality	25.557	-130.347 to 181.462	0.7193
Greater than $167 \text{ us/m}^3 (N=21)$	7	High quality*			
Steater than 107 µg/m (11-21)	4	High-mid quality	-35 619	-118,884 to 47 646	0.3763
	1	Low-mid quality	104.395	-35.246 to 244.037	0.1319
	9	Low quality	8.943	-54.129 to 72.015	0.7666

Table 4.40.01. Adjusted association between stove scale and Peak Expiratory Flow (PEF; L/minute) stratified by various factors.

*Reference category ; **Adjusted for age and height; ***Adjusted for height PM, particulate matter

Table 4.40.01. (continued)

				Adjusted estimate: Stove scale & PEF**	
	N	Stove Scale Variable	Beta coefficient	95% CI	P-value
Village of residence					
Santa Lucia (N=31)	7	High quality*			
	9	High-mid quality	-19.081	-85.607 to 47.445	0.5600
	6	Low-mid quality	9.882	-56.668 to 76.432	0.7623
	9	Low quality	23.417	-39.767 to 86.601	0.4524
Suyapa (N=21)	7	High quality*			
	4	High-mid quality	-35.284	-102.247 to 31.680	0.2791
	2	Low-mid quality	82.951	-8.289 to 174.191	0.0717
	8	Low quality	-6.900	-62.056 to 48.256	0.7934
Amount of time typically spe	nt in	the room with the fi	re burning		
Less than 3 hours (N=8)	2	High quality*			
	1	High-mid quality	-66.946	-625.691 to 491.800	0.6575
	1	Low-mid quality	-41.891	-575.477 to 491.695	0.7677
	4	Low quality	-29.242	-396.460 to 337.977	0.7645
3 or more hours (N=44)	12	High quality*			
× ,	12	High-mid quality	-23.051	-69.737 to 23.634	0.3238
	7	Low-mid quality	31.491	-23.073 to 86.055	0.2499
	13	Low quality	17.178	-28.861 to 63.216	0.4547
Any medication intake					
No Medication (N=31)	9	High quality*			
	6	High-mid quality	-25.698	-86.076 to 34.680	0.3891
	7	Low-mid quality	25.196	-34.284 to 84.675	0.3913
	9	Low quality	24.256	-29.017 to 77.529	0.3573
Medication (N=21)	5	High quality*			
	7	High-mid quality	-23.454	-80.436 to 33.528	0.3941
	1	Low-mid quality	-48.048	-154.572 to 58.476	0.3516
	8	Low quality	-14.146	-71.065 to 42.772	0.6040
Outdoor average afternoon t	empe	erature			
Low Outdoor Temp. (N=21)	8	High quality*			
2011 Concert compt (11 - 1)	3	High-mid quality	-36.120	-127.362 to 55.123	0.4121
	1	Low-mid quality	51.396	-68.537 to 171.329	0.3755
	9	Low quality	32.150	-22.820 to 87.119	0.2317
High Outdoor Temp.	6	High quality*			
(N=21)	6	High-mid quality	-38.627	-99.547 to 22.294	0.1966
	3	Low-mid quality	35.756	-41.337 to 112.850	0.3386
	6	Low quality	-24.610	-90.461 to 41.242	0.4381

*Reference category **Adjusted for age and height

Table 4.40.01. (continued)

			S	Adjusted estimate: Stove scale & PEF**	
	N	Stove Scale Variable	Beta coefficient	95% CI	P-value
Concern that stove smoke					
No $(N = 12)$	6	High quality*			
(11 - 12)	4	High-mid quality	-1.768	-43,194 to 39,689	0.9225
	0	Low-mid quality	NA	NA	NA
	2	Low quality	-12.464	-69.957 to 45.029	0.6240
Yes $(N = 40)$	8	High quality*			
	9	High-mid quality	-53.126	-112.005 to 5.754	0.0755
	8	Low-mid quality	3.523	-55.696 to 62.741	0.9045
	15	Low quality	-4.840	-57.249 to 47.570	0.8523
Any current respiratory symptoms					
No symptoms (N=26)	8	High quality*			
	7	High-mid quality	-27.395	-95.798 to 41.009	0.4314
	3	Low-mid quality	19.488	-69.258 to 108.234	0.6519
	8	Low quality	-19.334	-86.749 to 48.080	0.5564
Symptoms (N=26)	6	High quality*			
	6	High-mid quality	-35.533	-101.360 to 30.293	0.2735
	5	Low-mid quality	23.502	-42.761 to 89.765	0.4680
	9	Low quality	31.013	-27.279 to 89.305	0.2803
Length of time with current stove					
Less than 3 years (N=24)	9	High quality*			
•	7	High-mid quality	-23.464	-83.766 to 36.838	0.4255
	0	Low-mid quality	NA	NA	NA
	8	Low quality	7.470	-51.251 to 66.192	0.7929
3 or more years (N=28)	5	High quality*			
	6	High-mid quality	-14.650	-91.529 to 62.229	0.6965
	8	Low-mid quality	28.624	-37.780 to 95.028	0.3810
	9	Low quality	23.243	-44.419 to 90.906	0.4837

*Reference category **Adjusted for age and height

			S	Adjusted estimate: Stove scale & PEF**	
	N	Stove Scale	Beta	95% CI	P-value
		Variable	coefficient		
Total population $(N = 52)$	14	High quality*			
	13	High-mid quality	-26.360	-70.395 to 17.675	0.2344
	8	Low-mid quality	21.208	-28.891 to 71.307	0.3986
	17	Low quality	6.608	-34.318 to 47.534	0.7466
Women performing at least	12	High quality*			
2 successful lung function	8	High-mid quality	-10.665	-61.632 to 40.302	0.6740
maneuvers (N=43)	7	Low-mid quality	3.233	-49.545 to 56.012	0.9019
	16	Low quality	2.701	-40.863 to 46.266	0.9007
Women having their current	12	High quality*			
stove longer than 6 months	13	High-mid quality	-16.516	-63.332 to 30.299	0.4802
(N=47)	8	Low-mid quality	30.862	-21.558 to 83.282	0.2413
	14	Low quality	19.253	-27.148 to 65.653	0.4069
Women without a history of	12	High quality*			
smoking (N=49)	13	High-mid quality	-27.263	-74.211 to 19.686	0.2480
	8	Low-mid quality	21.149	-31.242 to 73.541	0.4201
	16	Low quality	3.610	-40.309 to 47.529	0.8691
Women indicating that their	13	High quality*			
day would not have been	7	High-mid quality	-18 185	-65.648 to 29 278	0 4405
different without monitoring	4	Low-mid quality	57 712	-0.128 to 115 553	0.0505
(N=37)	13	Low quality	2.302	-36.504 to 41.108	0.9045
Women not taking	14	High quality*			
bronchodilator medication	13	High-mid quality	-26 607	-71,130 to 17,917	0.2350
(N=51)	8	Low-mid quality	21.590	-29.090 to 72.271	0.3954
(/	16	Low quality	5.690	-36.061 to 47.440	0.7850

Table 4.40.02. Adjusted association between stove scale and Peak Expiratory Flow (PEF; L/minute) among subgroups of participants.

*Reference category

**Adjusted for age and height

		<u> </u>	S	Adjusted estimate: Stove scale & PEF**	
	N	Stove Scale Variable	Beta coefficient	95% CI	P-value
Total population (N = 52)	14	High quality*			
	13	High-mid quality	-26.360	-70.395 to 17.675	0.2344
	8	Low-mid quality	21.208	-28.891 to 71.307	0.3986
	17	Low quality	6.608	-34.318 to 47.534	0.7466
Presence of kitchen windows					
Kitchens with windows	10	High quality*			
(N=27)	7	High-mid quality	-0.349	-44.173 to 43.476	0.9870
	3	Low-mid quality	-59.879	-120.401 to 0.642	0.0523
	7	Low quality	-4.152	-48.420 to 40.116	0.8472
Kitchens without windows	А	High quality*			
(N-25)	6	High-mid quality	-67.813	-152 241 to 16 614	0 1091
(1(=23)	5	I ow-mid quality	44 874	-40 108 to 129 855	0.1021
	10	Low quality	-4.809	-80.894 to 71.275	0.8961
Kitchen volume					
Greater than 700 cu ft	12	High quality*		·	
(N=30)	7	High-mid quality	-1 278	-47 346 to 44 790	0 9548
(11-50)	'a	Low-mid quality	83 586	18 465 to 148 708	0.0140
	8	Low quality	38.267	-5.648 to 82.181	0.0847
Less than 700 cu. ft. (N=22)	2	High quality*			
	6	High-mid quality	-82.857	-196.670 to 30.956	0.1423
	5	Low-mid quality	-41.074	-159.648 to 77.500	0.4734
	9	Low quality	-55.522	-168.742 to 57.699	0.3140
Women cooking in	8	High quality*			
kitchens with eave spaces	11	High-mid quality	-24.302	-81.715 to 33.111	0.3960
(N=41)	6	Low-mid quality	44.925	-19.732 to 109.581	0.1672
	16	Low quality	15.987	-35.744 to 37.717	0.5345
Women cooking in	14	High quality*			
kitchens with 4 walls	13	High-mid quality	-24.081	-67.457 to 19.296	0.2691
(N=49)	7	Low-mid quality	18.528	-33.001 to 70.057	0.4723
· /	15	Low quality	15.601	-25.652 to 56.855	0.4498
Women cooking in	13	High quality*			
kitchens attached to or	10	High-mid quality	-39.296	-86.096 to 7.503	0.0975
part of the main living	7	Low-mid quality	5.601	-47.184 to 58.387	0.8313
area (N=46)	16	Low quality	8.639	-33.408 to 50.686	0.6802

Table 4.40.03. Adjusted association between stove scale and Peak Expiratory Flow (PEF; L/minute) among various ventilation subgroups.

*Reference category

**Adjusted for age and height

169

Table 4.41. Univariate linear regression estimates and 95% confidence intervals (CI) for the association of air quality measures and natural logarithm transformed C-reactive protein (CRP; mg/L on the dried blood scale) and potential confounders.

	Ň	Estimate	95% CI	P-value
Air Quality*				
$PM_{2.5}$, indoor (µg/m ³ ; 8-hr time-weighted average)	49	-0.085	-0.309 to 0.139	0.4501
$PM_{2.5}$, personal ($\mu g/m^3$; 8-hr time-weighted average)	50	-0.255	-0.597 to 0.087	0.1399
Carbon monoxide, indoor 1-hr maximum (ppm)	47	-0.020	-0.320 to 0.279	0.8918
Stove (Traditional vs. Improved)	71	-0.463	-1.163 to 0.237	0.1915
Stove Scale				
High quality	23	REF		
High-mid quality	13	-0.200	-1.231 to 0.831	0.6998
Low-mid quality	13	-0.225	-1.256 to 0.806	0.6648
Low quality	22	-0.719	-1.605 to 0.168	0.1103
Potential confounders				
Age (estimate per 10 year increase)	71	0.285	0.047 to 0.522	0.0195
Height (estimate per 3 inch increase)	71	-0.500	-0.905 to -0.094	0.0164
Waist Circumference (estimate per 5.5 inch increase)	71	0.600	0.198 to 1.001	0.0040
Second-hand smoke exposure (yes vs. no)	71	-0.082	-0.858 to 0.695	0.8341
Fish consumption (high vs. low)	71	0.101	-0.747 to 0.949	0.8133
Menopausal status	71	0.066	-0.710 to 0.842	0.8658
Education level				
>5 yrs	12	REF		
0.5 - 5 yrs	32	-0.164	-0.937 to 0.609	0.6732
0 yrs	27	0.606	-0.420 to 1.632	0.2428

*Estimates for PM_{2.5} and carbon monoxide are per IQR increase: personal PM_{2.5} (106.1 μ g/m³), indoor PM_{2.5} (572.3 μ g/m³), and indoor carbon monoxide 1-hr max (4.62 ppm)

PM, particulate matter; REF, reference category

evaluate potential confounding ($N=50$)					
Model	Personal PM _{2.5} Coefficient*	95% CI	P-value		
In(CRP) = Personal PM _{2.5}	-0.255	-0.597 to 0.087	0.1399		
ln(CRP) = Personal PM _{2.5} , Age, Height, Waist circumference, Second-hand smoke, Education level, Menopausal status	-0.237	-0.561 to 0.088	0.1482		

-0.219

-0.223

-0.225

-0.223

-0.222

-0.216

-0.048

-0.533 to 0.095

-0.529 to 0.082

-0.548 to 0.098

-0.540 to 0.095

-0.546 to 0.101

-0.537 to 0.106

-0.332 to 0.236

0.1677

0.1481

0.1675

0.1650

0.1731

0.1828

0.7349

Table 4.42. Selected models of the association between personal $PM_{2.5}$ and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=50)

*Estimates for personal PM _{2.5} are per IQR increase (106.1 μ g/m ³)
PM, particulate matter

Reduced model: $ln(CRP) = Personal PM_{2.5}, Age,$

-> Addition of Second-hand smoke exposure

 \rightarrow Cold/sinus problem during previous week (n=49)

 \rightarrow Addition of Outdoor average temperature (n= 46)

Height, Waist Circumference

 \rightarrow Addition of Education level

 \rightarrow Addition of Fish consumption

 \rightarrow Addition of Menopausal status

	N	Beta coefficient	95% CI	P-value
Personal PM _{2.5}				
Low (reference)	16			
Medium	17	-0.619	-1.494 to 0.255	0.1607
High	17	-0.787	-1.648 to 0.073	0.0720

Table 4.42.01. Adjusted association between Personal $PM_{2.5}$ assessed as a categorical variable and natural logarithm transformed C-reactive protein (adjusted for age, height, and waist circumference) (N=50).

PM, particulate matter

	Adjusted estimate: Personal PM _{2.5} & CRP*		
	Beta coefficient	95% CI	P-value
Total population (N = 50)	-0.219	-0.533 to 0.095	0.1677
Age**			
Less than 40 yrs (N=24)	-0.521	-1.236 to 0.194	0.1437
40 yrs and older (N=26)	-0.123	-0.413 to 0.167	0.3884
Second-hand smoke exposure			
No (N=37)	-0.188	-0.500 to 0.123	0.2265
Yes (N=13)	-0.134	-1.348 to 1.080	0.8054
Outdoor PM2 = levels			
Less than 167 μ g/m ³ (N=19)	-0.277	-1.745 to 1.191	0.6918
Greater than 167 μ g/m ³ (N=22)	-0.213	-0.617 to 0.190	0.2803
Village of residence			
Santa Lucia (N=28)	-0.006	-0.344 to 0.332	0.9710
Suyapa (N=22)	-0.960	-1.650 to -0.270	0.0093
Any mediaction intoka			
No Medication (N=34)	-0 274	-0 646 to 0 098	0 1424
Medication (N=16)	0.352	-0.709 to 1.414	0.4800
Anti-initiammatory and neart medication intake No Medication $(N-42)$	-0.253	-0.593 to 0.087	0 1305
Medication (N=8)	0.698	-0.595 to 0.087	0.4158
	0.020		011100
Infection/illness during prior week	0 129	0.455 to 0.100	0 4227
No liness $(N=38)$	-0.128	-0.455 to 0.199	0.4527
liness (IV-11)	-0.394	-1.940 to 1.139	0.5578
Outdoor average afternoon temperature	0.007		0.0=4.4
Low Outdoor Temp. (N=26)	0.006	-0.349 to 0.361	0.9716
High Outdoor Temp. (N=20)	-0.453	-1.228 to 0.322	0.2316
Concern that stove smoke causes health problems			
No $(N = 12)$	-0.998	-2.259 to 0.263	0.1034
Yes $(N = 38)$	-0.166	-0.508 to 0.177	0.3316
Stove type			
Improved (N = 25)	-1.014	-2.584 to 0.557	0.1933
Traditional ($N = 25$)	-0.249	-0.692 to 0.195	0.2557
Length of time with current stove			
Less than 3 years (N=24)	-0.451	-0.850 to -0.052	0.0286
3 or more years (N=26)	0.045	-0.520 to 0.611	0.8694

Table 4.42.02. Adjusted association between Personal $PM_{2.5}$ and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors.

*Adjusted for age, height, and waist circumference; Estimates for personal PM_{2.5} are per IQR increase (106.1 μ g/m³); **Adjusted for height and waist circumference; PM, particulate matter

	Adjusted estimate: Personal PM _{2.5} & CRP*			
	Beta coefficient	95% CI	P-value	
Total population (N = 50)	-0.219	-0.533 to 0.095	0.1677	
Women having their current stove longer than 6 months (N=42)	-0.196	-0.547 to 0.155	0.2651	
Women without a history of smoking (N=47)	-0.257	-0.575 to 0.062	0.1113	
Women indicating that their day would not have been different without monitoring (N=43)	-0.306	-0.768 to 0.156	0.1878	

Table 4.42.03. Adjusted association between Personal $PM_{2.5}$ and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants.

*Adjusted for age, height, and waist circumference; Estimates for personal $PM_{2.5}$ are per IQR increase (106.1 μ g/m³) PM, particulate matter

Model	Indoor PM _{2.5} Coefficient*	95% CI	P-value
$ln(CRP) = Indoor PM_{2.5}$	-0.085	-0.309 to 0.139	0.4501
ln(CRP) = Indoor PM _{2.5} , Age, Height, Waist circumference, Second-hand smoke, Education level, Menopausal status	0.068	-0.200 to 0.335	0.6130
Reduced model: ln(CRP) = Indoor PM _{2.5} , Age,	0.0003	-0.225 to 0.225	0.9976
Height, Waist circumference			
\rightarrow Addition of Second-hand smoke exposure	0.053	-0.189 to 0.295	0.6598
\rightarrow Addition of Education level	0.001	-0.236 to 0.237	0.9942
\rightarrow Addition of Fish consumption	-0.012	-0.258 to 0.234	0.9225
\rightarrow Addition of Menopausal status	0.001	0.230 to 0.232	0.9925
\rightarrow Cold/sinus problem during previous week (n=48)	0.008	-0.230 to 0.247	0.9436
\rightarrow Addition of Outdoor average temperature (n=46)	0.042	-0.174 to 0.258	0.6975

Table 4.43. Selected models of the association between indoor $PM_{2.5}$ and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=49).

*Estimates for indoor $PM_{2.5}$ are per IQR increase (572.3 μ g/m³) PM, particulate matter

	N	Beta coefficient	95% CI	P-value
Indoor PM _{2.5}				
Low (reference)	17			
Medium	17	-0.251	-1.026 to 0.523	0.5164
High	15	-0.339	-1.151 to 0.472	0.4038

Table 4.43.01. Adjusted association between indoor $PM_{2.5}$ assessed as a categorical variable and natural logarithm transformed C-reactive protein (mg/L) (adjusted for age, height, and waist circumference) (N=49).

PM, particulate matter

	Adjusted estimate: Indoor PM _{2.5} & CR		
	Beta coefficient	95% CI	P-value
Total population (N = 49)	0.0003	-0.225 to 0.225	0.9976
Age**			
Less than 40 yrs (N=23)	0.088	-0.194 to 0.371	0.5212
40 yrs and older (N=26)	-0.414	-1.022 to 0.194	0.1720
Second-hand smoke exposure			
No (N=37)	-0.381	-0.785 to 0.023	0.0634
Yes (N=12)	0.307	0.003 to 0.610	0.0482
Outdoor PMas levels			
Less than 167 μ g/m ³ (N=19)	-0.101	-0.474 to 0.271	0.5692
Greater than 167 μ g/m ³ (N=21)	0.098	-0.273 to 0.469	0.5822
Village of residence			
Santa Lucia (N=28)	0.066	-0.183 to 0.314	0.5885
Suyapa (N= 21)	-0.620	-1.285 to 0.045	0.0657
Amount of time typically spent in the room with the f	ire hurning		
Less than 3 hours (N=5)	NA	NA	NA
3 or more hours (N=44)	-0.072	-0.322 to 0.179	0.5654
Any medication intaka			
No Medication (N=33)	-0.003	-0 280 to 0 274	0 9817
Medication (N=16)	-0.114	-0.777 to 0.548	0.7116
Anti-inflammatory and heart medication intake			
No Medication (N=41)	0.002	-0.246 to 0.250	0.9858
Medication (N=8)	-0.150	-3.029 to 2.729	0.8791
Infaction/illness during prior week			
No Illness (N=38)	-0.051	-0.412 to 0.309	0 7734
Illness (N=10)	0.116	-0.495 to 0.728	0.6461
Outdoor average afternoon temperature	0.077	0.107 (0.240	0.5400
Low Outdoor Temp. (N=26)	0.077	-0.18 / to 0.342	0.5492
High Outdoor Temp. (N=20)	-0.931	-1.994 10 0.152	0.0810
Concern that stove smoke causes health problems	1 102	2 200 - 0 022	0.0050
No $(N = 12)$	-1.183	-3.289 to 0.923	0.2258
Yes(N=57)	0.027	-0.204 to 0.257	0.8132
Stove type	0		0.0007
Improved (N = 25) The line $(N = 24)$	-0.723	-2.092 to 0.646	0.2836
1 raditional (N = 24)	0.032	-0.206 to 0.271	0.7802
Length of time with current stove			
Less than 3 years (N=23)	-0.114	-0.463 to 0.235	0.5017
3 or more years (N=26)	0.075	-0.258 to 0.409	0.6437

Table 4.43.02. Adjusted association between indoor $PM_{2.5}$ and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors.

*Adjusted for age, height, and waist circumference; Estimates are per IQR increase (572.3 μ g/m³); **Adjusted for height and waist circumference; PM, particulate matter

	Adjusted estimate: Indoor PM _{2.5} & CRP*			
	Beta coefficient	95% CI	P-value	
Total population (N = 49)	0.0003	-0.225 to 0.225	0.9976	
Women having their current stove longer than 6 months (N=41)	0.030	-0.219 to 0.280	0.8063	
Women without a history of smoking (N=46)	0.004	-0.222 to 0.231	0.9686	
Women indicating that their day would not have been different without monitoring (N=42)	0.045	-0.193 to 0.284	0.7021	

Table 4.43.03. Adjusted association between Indoor $PM_{2.5}$ and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants.

*Adjusted for age, height, and waist circumference; Estimates for indoor $PM_{2.5}$ are per IQR increase (572.3 µg/m³) PM, particulate matter

178

Table 4.44. Selected models of the association between indoor carbon monoxide 1-hr maximum and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=47).

Model	Indoor Carbon Monoxide Coefficient*	95% CI	P-value
ln(CRP) = Indoor carbon monoxide 1-hr max	-0.020	-0.320 to 0.279	0.8918
In(CRP) = Indoor carbon monoxide 1-hr max, Age, Height, Waist circumference, Second-hand smoke, Education level, Menopausal status	0.097	-0.215 to 0.410	0.5315
Reduced model: ln(CRP) = Indoor carbon monoxide	0.060	-0.223 to 0.343	0.6698
1-hr max, Age, Height, Waist circumference			
\rightarrow Addition of Second-hand smoke exposure	0.081	-0.221 to 0.382	0.5924
\rightarrow Addition of Education level	0.068	-0.222 to 0.357	0.6383
\rightarrow Addition of Fish consumption	0.077	-0.231 to 0.385	0.6173
\rightarrow Addition of Menopausal status	0.063	-0.222 to 0.349	0.6560
\rightarrow Cold/sinus problem during previous week (n=46)	0.096	-0.200 to 0.393	0.5156
\rightarrow Addition of Outdoor average temperature (n=44)	0.022	-0.254 to 0.299	0.8704

*Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

179

Table 4.44.01. Adjusted association between indoor carbon monoxide 1-hr maximum
assessed as a categorical variable and natural logarithm transformed C-reactive protein
(CRP; mg/L) (adjusted for age, height, and waist circumference) (N=47).

	N	Beta coefficient	95% CI	P-value
Indoor carbon monoxide 1-hr max				
Low (reference)	18			
Medium	13	-1.122	-1.828 to -0.416	0.0026
High	16	-0.342	-1.007 to 0.324	0.3057

.

	Adjusted estimate: Indoor carbon monoxide 1-hr max & CRP*				
	Beta coefficient	95% CI	P-value		
Total population (N = 47)	0.060	-0.223 to 0.343	0.6698		
Age**					
Less than 40 yrs (N=23)	0.175	-0.241 to 0.591	0.3892		
40 yrs and older (N=24)	-0.037	-0.447 to 0.373	0.8519		
Second-hand smoke exposure					
No (N=35)	-0.185	-0.748 to 0.379	0.5089		
Yes (N=12)	0.229	-0.113 to 0.571	0.1576		
Outdoor PM _{2.5} levels					
Less than 167 μ g/m ³ (N=19)	0.061	-0.551 to 0.674	0.8330		
Greater than 167 µg/m ³ (N=19)	-0.032	-0.552 to 0.488	0.8982		
Village of residence					
Santa Lucia (N=25)	0.027	-0.381 to 0.434	0.8925		
Suyapa (N=22)	-0.001	-0.496 to 0.493	0.9953		
Amount of time typically spent in the room with the fire burning					
Less than 3 hours (N=4)	NA	NA	NA		
3 or more hours (N=43)	0.071	-0.277 to 0.420	0.6816		
Any medication intake					
No Medication (N=32)	-0.028	-0.432 to 0.377	0.8892		
Medication (N=15)	0.210	-0.265 to 0.685	0.3484		
Anti-inflammatory and heart medication intake					
No Medication (N=40)	0.028	-0.305 to 0.361	0.8654		
Medication (N=7)	0.470	-0.791 to 1.731	0.2502		
Infection/illness during prior week					
No Illness (N=36)	0.157	-0.218 to 0.533	0.3999		
Illness (N=10)	0.391	-0.590 to 1.373	0.3522		

Table 4.44.02. Adjusted association between indoor carbon monoxide 1-hr maximum and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors.

*Adjusted for age, height, and waist circumference; **Adjusted for height and waist circumference; PM, particulate matter; Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

Table 4.44.02. continued.

······································	Adjusted estimate: Indoor carbon monoxide 1-hr max & CRP*			
	Beta coefficient	95% CI	P-value	
Outdoor average afternoon temperature				
Low Outdoor Temp. (N=23)	-0.002	-0.426 to 0.421	0.9912	
High Outdoor Temp. (N=21)	-0.009	-0.447 to 0.429	0.9659	
Concern that stove smoke causes health problems				
No $(N = 12)$	-0.533	-1.790 to 0.724	0.3497	
Yes $(N = 35)$	0.177	-0.141 to 0.494	0.2648	
Stove type				
Improved $(N = 24)$	-0.733	-2.435 to 0.970	0.3791	
Traditional ($N = 23$)	0.163	-0.199 to 0.525	0.3558	
Length of time with current stove				
Less than 3 years (N=22)	-0.254	-0.890 to 0.383	0.4121	
3 or more years (N=26)	0.078	-0.277 to 0.432	0.6524	

*Adjusted for age, height, and waist circumference; PM, particulate matter; Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

	Adjusted estimate: Indoor carbon monoxide 1-hr max & CRP*			
	Beta coefficient	95% CI	P-value	
Total population (N = 47)	0.060	-0.223 to 0.343	0.6698	
Women having their current stove longer than 6 months (N=40)	0.096	-0.209 to 0.401	0.5269	
Women without a history of smoking (N=45)	0.013	-0.288 to 0.315	0.9286	
Women indicating that their day would not have been different without monitoring (N=41)	0.049	-0.260 to 0.357	0.7502	

Table 4.44.03. Adjusted association between Indoor carbon monoxide 1-hr maximum and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants.

*Adjusted for age, height, and waist circumference; Estimates for indoor carbon monoxide 1-hr max are per IQR increase (4.62 ppm)

Model	Stove Coefficient (Traditional vs. Improved)	95% CI	P-value
ln(CRP) = Stove	-0.463	-1.163 to 0.2372	0.1915
In(CRP) = Stove, Age, Height, Waist circumference, Second-hand smoke, Education level, Menopausal status	-0.202	-0.879 to 0.475	0.5526
Reduced model: In(CRP) = Stove, Age, Height, Waist	-0.240	-0.897 to 0.416	0.4675
circumference			
\rightarrow Addition of Second-hand smoke exposure	-0.263	-0.924 to 0.398	0.4300
\rightarrow Addition of Education level	-0.165	-0.847 to 0.517	0.6302
\rightarrow Addition of Fish consumption	-0.226	-0.888 to 0.436	0.4984
\rightarrow Addition of Menopausal status	-0.240	-0.886 to 0.406	0.4601
\rightarrow Cold/sinus problem during previous week (n=70)	-0.248	-0.922 to 0.426	0.4649

Table 4.45. Selected models of the association between stove type (Traditional vs. Improved) and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=71).

	£		
	Beta coefficient	95% CI	P-value
Total population (N = 71)	-0.240	-0.897 to 0.416	0.4675
Age**			
Less than 40 yrs (N=38)	-0.262	-1.304 to 0.781	0.6131
40 yrs and older (N=33)	-0.146	-0.885 to 0.594	0.6898
Second-hand smoke exposure			
No (N=50)	-0.417	-1.151 to 0.317	0.2585
Yes (N=21)	0.090	-1.429 to 1.609	0.9015
Outdoor PM _{2.5} levels			
Less than 167 μ g/m ³ (N=19)	-0.274	-1.729 to 1.182	0.6928
Greater than 167 μ g/m ³ (N=23)	-0.677	-1.805 to 0.452	0.2237
Village of residence			
Santa Lucia (N=48)	-0.219	-1.042 to 0.604	0.5948
Suyapa (N=23)	-0.502	-1.757 to 0.753	0.4120
Amount of time typically spent in the room with the fire burning			
Less than 3 hours (N=9)	-1.041	-6.075 to 3.992	0.5965
3 or more hours (N=62)	-0.130	-0.703 to 0.443	0.6507
Any medication intake			
No Medication (N=44)	-0.530	-1.305 to 0.246	0.1751
Medication (N=27)	0.472	-0.838 to 1.782	0.4632
Anti-inflammatory and heart medication intake			
No Medication (N=62)	-0.370	-1.108 to 0.367	0.3186
Medication (N=9)	0.802	-2.438 to 4.042	0.5295
Infection/illness during prior week			
No Illness (N=53)	-0.229	-0.979 to 0.522	0.5431
Illness (N=17)	-0.265	-2.420 to 1.891	0.7935

Table 4.45.01. Adjusted association between stove type (Traditional vs. Improved) and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors.

.

*Adjusted for age, height, and waist circumference; **Adjusted for height and waist circumference

Table 4.45.01. continuous.

	Adjusted estimate: Stove type & CRP*			
	Beta coefficient	95% CI	P-value	
Outdoor average afternoon temperature				
Low Outdoor Temp. (N=26)	-0.363	-1.398 to 0.673	0.4744	
High Outdoor Temp. (N=21)	-0.366	-1.257 to 0.525	0.3965	
Concern that stove smoke causes health problems				
No $(N = 17)$	-1.052	-2.417 to 0.314	0.1193	
Yes $(N = 54)$	0.093	-0.708 to 0.893	0.8167	
Length of time with current stove				
Less than 3 years (N=32)	-0.626	-1.711 to 0.459	0.2471	
3 or more years (N=39)	-0.144	-1.076 to 0.788	0.7558	

*Adjusted for age, height, and waist circumference

	Adjusted estimate: Stove type & CRP*			
	Beta coefficient	95% CI	P-value	
Total population (N = 71)	-0.240	-0.897 to 0.416	0.4675	
Women having their current stove longer than 6 months (N=62)	-0.162	-0.886 to 0.563	0.6566	
Women without a history of smoking (N=67)	-0.343	-1.018 to 0.331	0.3127	
Women indicating that their day would not have been different without monitoring (N=44)	-0.179	-0.991 to 0.634	0.6587	

Table 4.45.02. Adjusted association between stove type (Traditional vs. Improved) and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants.

*Adjusted for age, height, and waist circumference

		Adjusted estimate: Stove type & CRP*	
	Beta coefficient	95% CI	P-value
Total population (N = 71)	-0.240	-0.897 to 0.416	0.4675
Presence of kitchen windows			
Kitchens with windows (N=38)	0.228	-0.828 to 1.284	0.6638
Kitchens without windows (N=33)	-0.961	-1.795 to -0.128	0.0253
Kitchen volume			
Greater than 700 cu. ft. (N=36)	-0.200	-1.128 to 0.727	0.6625
Less than 700 cu. ft. (N=35)	-0.321	-1.583 to 0.941	0.6074
Women cooking in kitchens with eave spaces (N=56)	-0.198	-0.999 to 0.602	0.6212
Women cooking in kitchens with 4 walls (N=63)	-0.296	-1.011 to 0.418	0.4096
Women cooking in kitchens attached to or part of the main living area (N=61)	-0.410	-1.033 to 0.212	0.1917

Table 4.45.03. Adjusted association between stove type (Traditional vs. Improved) and natural logarithm transformed C-reactive protein (CRP; mg/L) among various ventilation subgroups.

*Adjusted for age, height, and waist circumference

Table 4.46. Selected models of the association between stove scale and natural logarithm transformed C-reactive protein (CRP; mg/L): crude association, full model, reduced model, and reduced model with the addition of each variable individually to evaluate potential confounding (N=71).

Model	N	Stove Scale	Stove Scale	95% CI	P-value
		Variable	Coefficient		· · · · · · · · · · · · · · · · · · ·
ln(CRP) = Stove scale	23	High quality*			
	13	High-mid quality	-0.200	-1.231 to 0.831	0.6998
	13	Low-mid quality	-0.225	-1.256 to 0.806	0.6648
	22	Low quality	-0.719	-1.605 to 0.168	0.1103
ln(CRP) = Stove scale, Age, Height,	23	High quality*			
Waist circumference. Second-hand	13	High-mid quality	-0.008	-0.977 to 0.961	0.9868
smoke. Education level.	13	Low-mid quality	-0.063	-1.027 to 0.900	0.8959
Menopausal status	22	Low quality	-0.287	-1.127 to 0.554	0.4977
Reduced model: ln(CRP) = Stove	23	High quality*			
scale, Age, Height, Waist	13	High-mid quality	-0.109	-1.045 to 0.828	0.8174
circumference	13	Low-mid quality	-0.027	-0.981 to 0.927	0.9553
	22	Low quality	-0.419	-1.239 to 0.400	0.3105
\rightarrow Addition of Second-hand	23	High quality*			
smoke exposure	13	High-mid quality	-0.038	-0.998 to 0.922	0 9371
smoke exposure	13	I ow-mid quality	-0.038	-0.997 to 0.920	0.9364
	22	Low quality	-0.406	-1.229 to 0.418	0.3289
	22	Low quality	-0.400	-1.227 (0 0.410	0.5207
\rightarrow Addition of Education level	23	High quality*			
	13	High-mid quality	-0.147	-1.097 to 0.803	0.7585
	13	Low-mid quality	0.004	-0.963 to 0.972	0.9929
	22	Low quality	-0.346	-1.190 to 0.498	0.4160
Addition of Eich consumption	22	Uich quality*			
\rightarrow Addition of Fish consumption	13	High mid quality	0.117	 1.060 to 0.826	0 8054
	12	Low mid quality	-0.117	-1.000 to 0.820	0.0004
	15	Low-mid quanty	-0.028	-0.966 to 0.952	0.9343
	<i>L L</i>	Low quanty	-0.403	-1.230 to 0.424	0.3343
\rightarrow Addition of Menopausal	23	High quality*			
status	13	High-mid quality	-0.050	-0.977 to 0.877	0.9144
	13	Low-mid quality	-0.078	-1.021 to 0.866	0.8696
	22	Low quality	-0.358	-1.170 to 0.454	0.3811
Cold/ginus problem during	22	Uich qualitest			
\rightarrow condistinus problem during	23 12	Ligh mid quality*	0.006	 1 049 to 0 955	
previous week (n=70)	13	Low mid quality	-0.090	-1.040 0 0.833	0.0401
	13	Low-mid quality	-0.015	-0.984 to 0.954	0.9/5/
	21	Low quality	-0.447	-1.294 to 0.401	0.2962

*Reference category

			Ac	djusted estimate:	
	N	Stove Scale		95% CI	P-value
	14	Variable	coefficient	<i>)) i i i</i>	1 - 14140
Total population (N = 71)	23	High quality*			
• •	13	High-mid quality	-0.109	-1.045 to 0.828	0.8174
	13	Low-mid quality	-0.027	-0.981 to 0.927	0.9553
	22	Low quality	-0.419	-1.239 to 0.400	0.3105
Age***					
Less than 40 yrs (N=38)	9	High quality*			
· · · · ·	6	High-mid quality	-0.244	-1.918 to 1.429	0.7680
	11	Low-mid quality	-0.035	-1.465 to 1.396	0.9610
	12	Low quality	-0.691	-2.119 to 0.737	0.3315
40 yrs and older (N=33)	14	High quality*			
	7	High-mid quality	-0.091	-1.080 to 0.898	0.8519
	2	Low-mid quality	-0.678	-2.392 to 1.037	0.4246
	10	Low quality	-0.099	-0.972 to 0.774	0.8176
Cocond hand smalle superior					
Second-nand smoke exposure N_0 (N-50)	17	High quality*			
100 (11-50)	6	High-mid quality	-0.232	-1 424 to 0.960	0 6962
	11	I ow-mid quality	-0.232	-1.424 to 0.900	0.0902
	16	Low quality	-0.608	-1.509 to 0.293	0.1804
Yes (N=21)	6	High quality*			
	7	High-mid quality	0.520	-1.406 to 2.446	0.5719
	2	Low-mid quality	0.430	-2.365 to 3.224	0.7465
	6	Low quality	0.426	-1.765 to 2.616	0.6833
Outdoor PM _{2.5} levels					
Less than 167 μ g/m ³ (N=19)	7	High quality*			
	3	High-mid quality	-0.751	-2.705 to 1.204	0.4191
	3	Low-mid quality	-0.146	-2.341 to 2.049	0.8870
	6	Low quality	-0.732	-2.938 to 1.471	0.4837
Greater than 167 µg/m ³ (N=23)	8	High quality*		~-	
	3	High-mid quality	-0.378	-2.410 to 1.655	0.6990
	3	Low-mid quality	-0.359	-2.308 to 1.591	0.7015
	9	Low quality	-0.873	-2.231 to 0.486	0.1922

Table 4.46.01. Adjusted association between stove scale and natural logarithm transformed C-reactive protein (CRP; mg/L) stratified by various factors.

*Reference category

**Adjusted for age, height, and waist circumference

***Adjusted for height and waist circumference

PM, particulate matter

190

Table 4.46.01. (continued)

		Stove Scale Variable	Adjusted estimate:			
	N		Beta coefficient	95% CI	P-value	
Village of residence				<u></u> ,		
Santa Lucia (N=48)	12	High quality*				
	11	High-mid quality	0.154	-0.984 to 1.292	0.7861	
	10	Low-mid quality	-0.134	-1.318 to 1.050	0.8205	
	15	Low quality	-0.161	-1.250 to 0.927	0.7660	
Suyapa (N=23)	11	High quality*				
	2	High-mid quality	-0.897	-3.221 to 1.426	0.4249	
	3	Low-mid quality	0.319	-1.650 to 2.287	0.7361	
	7	Low quality	-1.043	-2.483 to 0.397	0.1442	
Amount of time typically spen	nt in the roo	om with the fire bur	ning			
Less than 3 hours (N=9)	3	High quality*	NA	NA	NA	
	0	High-mid quality				
	2	Low-mid quality				
	4	Low quality				
3 or more hours (N=62)	20	High quality*				
	13	High-mid quality	-0.189	-0.973 to 0.596	0.6321	
	11	Low-mid quality	-0.173	-1.007 to 0.661	0.6793	
	18	Low quality	-0.220	-0.948 to 0.508	0.5476	
Any medication intake						
No Medication (N=44)	14	High quality*		·		
	7	High-mid quality	-0.556	-1.765 to 0.654	0.3579	
	11	Low-mid quality	-0.505	-1.564 to 0.555	0.3407	
	12	Low quality	-0.891	-1.899 to 0.117	0.0814	
Medication (N=27)	9	High quality*				
	6	High-mid quality	0.394	-1.270 to 2.058	0.6267	
	2	Low-mid quality	0.996	-1.502 to 3.494	0.4152	
	10	Low quality	0.540	-1.044 to 2.125	0.4852	
Anti-inflammatory and heart	medication	n intake				
No Medication (N=62)	19	High quality*				
	12	High-mid quality	-0.133	-1.198 to 0.931	0.8029	
	13	Low-mid quality	-0.105	-1.139 to 0.928	0.8390	
	18	Low quality	-0.636	-1.578 to 0.306	0.1814	
Medication (N=9)	4	High quality*	NA	NA	NA	
	1	High-mid quality				
	0	Low-mid quality				
	4	Low quality				

*Reference category **Adjusted for age, height, and waist circumference

Table 4.46.01. (continued)

			Adjusted estimate: Stove scale & CRP**			
	N	- Stove Scale Variable	Beta coefficient	95% CI	P-value	
Infection/illness during prior week		·····				
No Illness (N=53)	18	High quality*				
	12	High-mid quality	-0.210	-1 234 to 0 814	0.6819	
	10	Low-mid quality	-0.001	-1 108 to 1 107	0.0012	
	13	Low quality	-0.529	-1.540 to 0.481	0.2972	
Hippess $(N-17)$	5	High quality*				
	1	High mid quality	1 273	A 777 to 7 324	0 6402	
	3	Low-mid quality	0.338	-3.072 to 3.748	0.0492	
	8	Low quality	0.126	-3.622 to 3.873	0.8230	
Outdoor average afternoon tempera	ature					
Low Outdoor Temp. (N=26)	10	High quality*				
F.()	3	High-mid quality	-0.532	-2.434 to 1.369	0.5648	
	3	Low-mid quality	-1.011	-2.724 to 0.702	0.2318	
	10	Low quality	-0.271	-1.413 to 0.872	0.6257	
Hist October Terrer (N. A1)	10	TT: _1,				
High Outdoor Temp. (N=21)	10	High quality*				
	3	High-mid quality	-0.585	-1.800 to 0.629	0.3188	
	5 5	Low-mid quality	-0.168	-2.338 to 0.146 -1.253 to 0.918	0.0792	
Concern that stave smake causes						
health problems						
No $(N = 17)$	8	High quality*				
110 (11 = 17)	3	High-mid quality	0 988	-0 754 to 2 729	0 2349	
	3	Low-mid quality	-1.882	-3.697 to -0.067	0.0435	
	3	Low quality	-0.278	-1.875 to 1.319	0.7066	
V AL CA	15	TT' 1 1'. U				
Y es (N = 54)	15	High quality*				
	10	Hign-mid quality	-0.053	-1.215 to 1.109	0.9209	
	10 19	Low-mid quality	-0.139	-0.700 to 1.633	0.4251	
		1				
Length of time with current stove						
Less than 3 years (N=32)	15	High quality*				
	6	High-mid quality	-0.532	-1.902 to 0.837	0.4309	
	2	Low-mid quality	-0.654	-2.895 to 1.588	0.5536	
a a a	9	Low quality	-0.842	-2.099 to 0.415	0.1800	
3 or more years (N=39)	8	High quality*				
	7	High-mid quality	-0.052	-1.548 to 1.444	0.9440	
	11	Low-mid quality	-0.016	-1.335 to 1.303	0.9803	
	13	Low quality	-0.275	-1.562 to 1.013	0.6666	

*Reference category **Adjusted for age, height, and waist circumference

			Adjusted estimate: Stove scale & CRP**			
		Stove Scale Variable	Beta coefficient	95% CI	P-value	
Total population (N = 71)	23	High quality*				
	13	High-mid quality	-0.109	-1.045 to 0.828	0.8174	
	13	Low-mid quality	-0.027	-0.981 to 0.927	0.9553	
	22	Low quality	-0.419	-1.239 to 0.400	0.3105	
Women having their current stove	19	High quality*				
longer than 6 months (N=62)	13	High-mid quality	-0.096	-1.109 to 0.918	0.8504	
	12	Low-mid quality	0.062	-0.977 to 1.101	0.9054	
	18	Low quality	-0.381	-1.333 to 0.571	0.4262	
Women without a history of	20	High quality*				
smoking (N=67)	13	High-mid quality	-0.200	-1.172 to 0.772	0.6823	
	13	Low-mid quality	-0.121	-1.096 to 0.854	0.8044	
	21	Low quality	-0.591	-1.443 to 0.261	0.1702	
Women indicating that their day	18	High quality*				
would not have been different	6	High-mid quality	-0.687	-1.941 to 0.567	0.2742	
without monitoring (N=44)	7	Low-mid quality	-0.242	-1.413 to 0.928	0.6775	
	13	Low quality	-0.382	-1.359 to 0.594	0.4327	

Table 4.46.02. Adjusted association between stove scale and natural logarithm transformed C-reactive protein (CRP; mg/L) among subgroups of participants.

*Reference category

*Adjusted for age, height, and waist circumference
				Adjusted estimate:	
	N	Stove Seale	SI	050% CT	Dualua
	14	Variable	coefficient	95 % CI	I -value
Total population (N = 71)	23	High quality*			
· ·	13	High-mid quality	-0.109	-1.045 to 0.828	0.8174
	13	Low-mid quality	-0.027	-0.981 to 0.927	0.9553
	22	Low quality	-0.419	-1.239 to 0.400	0.3105
Presence of kitchen windows					
Kitchens with windows (N=38)	16	High quality*			
	7	High-mid quality	-0.110	-1.483 to 1.264	0.8718
	6	Low-mid quality	0.630	-0.896 to 2.155	0.4063
	9	Low quality	-0.087	-1.412 to 1.238	0.8942
Kitchens without windows (N=33)	7	High quality*			
	6	High-mid quality	-0.482	-1.829 to 0.865	0.4683
	7	Low-mid quality	-1.095	-2.417 to 0.226	0.1003
	13	Low quality	-1.241	-2.369 to -0.113	0.0323
Kitchen volume					
Greater than 700 cu. ft. (N=36)	18	High quality*			
	8	High-mid quality	-0.616	-1.698 to 0.467	0.2543
	3	Low-mid quality	-0.712	-2.280 to 0.856	0.3605
	7	Low quality	-0.272	-1.353 to 0.809	0.6109
Less than 700 cu. ft. (N=35)	5	High quality*			
	5	High-mid quality	1.240	-0.940 to 3.421	0.2537
	10	Low-mid quality	0.748	-1.167 to 2.663	0.4303
	15	Low quality	0.085	-1.716 to 1.885	0.9240
Women cooking in kitchens with	15	High quality*			
eave spaces (N=56)	11	High-mid quality	-0.320	-1.490 to 0.850	0.5851
	10	Low-mid quality	-0.110	-1.305 to 1.086	0.8545
	20	Low quality	-0.421	-1.419 to 0.577	0.4006
Women cooking in kitchens with 4	23	High quality*			
walls (N=63)	12	High-mid quality	-0.184	-1.170 to 0.801	0.7093
	10	Low-mid quality	0.061	-1.009 to 1.131	0.9095
	18	Low quality	-0.580	-1.463 to 0.303	0.1936
Women cooking in kitchens	21	High quality*			
attached to or part of the main	9	High-mid quality	-0.00 6	-0.911 to 0.898	0.9886
living area (N=61)	11	Low-mid quality	-0.496	-1.386 to 0.394	0.2686
-	20	Low quality	-0.369	-1.120 to 0.383	0.3302

Table 4.46.03. Adjusted association between stove scale and natural logarithm transformed C-reactive protein (CRP; mg/L) among various ventilation subgroups.

*Reference category

**Adjusted for age, height, and waist circumference

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

Age-adjusted Estimate Adjusted estimate Crude Estimate Adjusted estimate including Outdoor eliminating those taking **Temperature bronchodilators** N **O**R 95% CI N OR 95% CI N OR 95% CI N **OR** 95% CI $PM_{2.5}$, personal ($\mu g/m^3$; 8-hr 58 1.06 0.55 to 2.05 58 1.05 0.54 to 2.06 54 1.32 0.64 to 2.69 57 1.08 0.55 to 2.13 time-weighted average) PM_{25} , indoor (µg/m³; 8-hr 57 1.22 0.81 to 1.82 57 1.49* 0.84 to 2.68 54 1.78 0.89 to 3.57 56 1.69 0.89 to 3.20 time-weighted average) Carbon monoxide (CO), 0.58 to 2.16 1.33* 0.58 to 3.05 51 1.27 0.50 to 3.24 0.60 to 3.41 54 1.11 54 53 1.43 indoor 1-hr maximum (ppm) 55 8.87 0.91 to 86.12 Stove type 7.94 1.63 to 38.75 79 7.99 1.59 to 40.09 1.45 to 36.78 79 77 7.29 (Traditional vs. Improved) Stove scale **REF[‡] REF[‡] REF[‡]** High quality 23 23 NA 22 High-mid quality 17 1.38 0.08 to 23.67 17 1.38 0.08 to 23.98 17 1.28 0.07 to 22.34 Low-mid quality 15 8.00 0.80 to 80.41 15 7.93 0.78 to 80.96 15 7.80 0.77 to 79.41 Low quality 24 1.02 to 80.84 9.02 1.01 to 80.85 23 0.82 to 68.10 9.06 24 7.45

Table 4.47. Odds Ratios (OR) and 95% confidence intervals (CI) for the association between cookstove exposures and the usual presence of a cough as assessed by the question, "Do you usually have a cough?"

*Adjusted for age and second-hand smoke exposure; [‡]P-value for trend < 0.05; NA, Unstable estimates; PM, particulate matter; REF, reference category; Estimates for PM_{2.5} and carbon monoxide are per IQR increase: personal PM_{2.5} (106.1 μ g/m³), indoor PM_{2.5} (572.3 μ g/m³), and indoor carbon monoxide 1 hr max (4.62 ppm)

Table 4.47.01. Odds Ratios (OR) and 95% confidence intervals (CI) for the association between cookstove exposures (including copollutant models) and the usual presence of a cough as assessed by the question, "Do you usually have a cough?"

		Adjusted	Estimate*
	N	ÒR	95% CI
$PM_{2.5}$, indoor (µg/m ³ ; 8-hr time-weighted average)	57	1.49	0.84 to 2.68
Carbon monoxide, indoor 1-hr maximum (ppm)	54	1.33	0.58 to 3.05
Model with both indoor $PM_{2.5}$ and carbon monoxide 1-hr maximum	53		
PM _{2.5}		2.11	0.59 to 7.61
Carbon monoxide 1-hr max		0.53	0.08 to 3.47

*Adjusted for age and second-hand smoke exposure; PM, particulate matter; Estimates for PM_{2.5} and carbon monoxide are per IQR increase: indoor PM_{2.5} (572.3 μ g/m³) and indoor Carbon monoxide 1-hr max (4.62 ppm)

Table 4.48. Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the usual presence of phlegm as assessed by the question, "Do you usually bring up phlegm from your chest?"

	Crude Estimate		Age-adjusted Estimate			Adjusted estimate including Outdoor Temperature			Adjusted estimate eliminating those taking bronchodilators			
	N	OR	95% CI	N	OR	95% CI	N	OR	95% CI	N	OR	95% CI
PM _{2.5} , personal (µg/m ³ ; 8-hr time-weighted average)	58	0.78	0.30 to 2.03	58	0.81	0.32 to 2.05	54	0.83	0.32 to 2.16	57	0.79	0.27 to 2.25
PM _{2.5} , indoor (µg/m ³ ; 8-hr time-weighted average)	57	1.09	0.68 to 1.74	57	1.26	0.74 to 2.15	54	1.46	0.82 to 2.58	56	1.47	0.81 to 2.65
Carbon monoxide, indoor 1- hr maximum (ppm)	54	1.02	0.49 to 2.15	54	1.08	0.49 to 2.39	51	0.89	0.31 to 2.52	53	1.11	0.45 to 2.75
Stove type (Traditional vs. Improved)	79	2.86	0.68 to 11.99	79	3.83	0.86 to 17.14	55	3.05	0.48 to 19.28	77	3.16	0.68 to 14.62
Stove scale High quality High-mid quality Low-mid quality Low quality		NA			NA			NA			NA	

NA, Unstable estimates; PM, particulate matter; Estimates for $PM_{2.5}$ and carbon monoxide are per IQR increase: personal $PM_{2.5}$ (106.1 μ g/m³), indoor $PM_{2.5}$ (572.3 μ g/m³), and indoor carbon monoxide 1-hr max (4.62 ppm)

Table 4.48.01. Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures (including copollutant models) and the usual presence of phlegm as assessed by the question, "Do you usually bring up phlegm from your chest?"

	E	Age-adjus	ted Estimate
	N	OR	95% CI
$PM_{2.5}$, indoor (µg/m ³ ; 8-hr time-weighted average)	57	1.26	0.74 to 2.15
Carbon monoxide, indoor 1-hr maximum (ppm)	54	1.08	0.49 to 2.39
Model with both indoor PM _{2.5} and Carbon monoxide 1-hr maximum	53		
PM _{2.5}		1.40	0.59 to 3.33
Carbon monoxide 1-hr max		0.78	0.23 to 2.60

PM, particulate matter; Estimates for PM_{2.5} and carbon monoxide are per IQR increase: indoor PM_{2.5} (572.3 μ g/m³) and indoor carbon monoxide 1-hr max (4.62 ppm)

Table 4.49. Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the usual presence of wheeze as assessed by the question, "Does your chest usually sound wheezy or whistling?"

	Crude Estimate		Age-adjusted Estimate			Adjusted estimate including Outdoor Temperature			Adjusted estimate eliminating those taking bronchodilators			
	N	OR	95% CI	N	OR	95% CI	N	OR	95% CI	N	OR	95% CI
$PM_{2.5}$, personal ($\mu g/m^3$; 8-hr time-weighted average)	58	0.94	0.45 to 1.97	58	0.94	0.45 to 1.97	54	1.11	0.51 to 2.44	57	0.96	0.45 to 2.06
$PM_{2.5}$, indoor (µg/m ³ ; 8-hr time-weighted average)	57	1.48	0.97 to 2.23	57	1.64	1.02 to 2.62	54	1.79	1.07 to 2.99	56	1.90	1.12 to 3.21
Carbon monoxide, indoor 1- hr maximum (ppm)	54	1.37	0.75 to 2.50	54	1.42	0.76 to 2.63	51	1.62	0.84 to 3.10	53	1.54	0.81 to 2.93
Stove type (Traditional vs. Improved)		NA			NA			NA			NA	
Stove scale High quality High-mid quality Low-mid quality Low quality		NA			NA			NA			NA	

NA, Unstable estimates; PM, particulate matter; Estimates for $PM_{2.5}$ and carbon monoxide are per IQR increase: personal $PM_{2.5}$ (106.1 µg/m³), indoor $PM_{2.5}$ (572.3 µg/m³), and indoor carbon monoxide 1-hr max (4.62 ppm)

Table 4.49.01. Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures (including copollutant models) and the usual presence of wheeze as assessed by the question, "Does your chest usually sound wheezy or whistling?"

		Age-adjus	ted Estimate
	N	OR OR	95% CI
$PM_{2.5}$, indoor (µg/m ³ ; 8-hr time-weighted average)	57	1.64	1.02 to 2.62
Carbon monoxide, indoor 1-hr maximum (ppm)	54	1.42	0.76 to 2.63
Model with both indoor PM _{2.5} and Carbon monoxide 1-hr maximum	53		
PM _{2.5}		2.34	0.85 to 6.39
Carbon monoxide 1-hr max		0.50	0.10 to 2.48

PM, particulate matter; Estimates for PM_{2.5} and carbon monoxide are per IQR increase: indoor PM_{2.5} (572.3 μ g/m³) and indoor carbon monoxide 1-hr max (4.62 ppm)

Table 4.50. Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the presence of nasal irritation as assessed by the question, "Do you have current nasal stuffiness, runny nose, sneezing, and/or nasal itch?"

	Crude Estimate		Age-adjusted Estimate			i	Adjusted estimate including Outdoor Temperature			Adjusted estimate eliminating those taking bronchodilators		
	N OR	95% CI	N	OR	95% CI	N	0R	95% CI	N	OR	95% CI	
$PM_{2.5}$, personal (µg/m ³ ; 8-hr time-weighted average)	58 0.75	0.39 to 1.45	58	0.70	0.34 to 1.44	54	0.72	0.34 to 1.44	57	0.70	0.34 to 1.45	
$PM_{2.5}$, indoor (µg/m ³ ; 8-hr time-weighted average)	57 0.68	0.35 to 1.32	57	0.60	0.30 to 1.16	54	0.45	0.16 to 1.30	56	0.61	0.31 to 1.19	
Carbon monoxide, indoor 1- hr maximum (ppm)	54 0.62	0.28 to 1.38	54	0.52	0.23 to 1.22	51	0.36	0.10 to 1.28	53	0.54	0.23 to 1.26	
Stove type (Traditional vs. Improved)	79 1.11	0.42 to 2.97	79	0.92	0.33 to 2.60	55	1.14	0.33 to 3.89	77	0.85	0.30 to 2.43	
Stove scale												
High quality	23 REF		23	REF		20	REF		22	REF		
High-mid quality	17 1.96	0.48 to 7.99	17	2.28	0.54 to 9.63	10	4.13	0.66 to 25.97	17	2.17	0.51 to 9.18	
Low-mid quality	15 1.80	0.42 to 7.76	15	1.52	0.34 to 6.75	8	3.01	0.44 to 20.64	15	1.51	0.34 to 6.69	
Low quality	24 1.20	0.31 to 4.65	24	1.06	0.27 to 4.25	17	1.39	0.27 to 7.14	23	0.89	0.21 to 3.69	

PM, particulate matter; REF, reference category; Estimates for $PM_{2.5}$ and carbon monoxide are per IQR increase: personal $PM_{2.5}$ (106.1 μ g/m³), indoor $PM_{2.5}$ (572.3 μ g/m³), and indoor carbon monoxide 1-hr max (4.62 ppm)

Table 4.51. Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the usual presence of a headache as assessed by the question, "Do you usually develop a headache during cooking?"

	Crude Estimate		Age-adjusted Estimate				Adjusted estimate including Outdoor Temperature			Adjusted estimate eliminating those taking bronchodilators		
	N	OR	95% CI	N	OR	95% CI	i	N OR	95% CI	N	OR	95% CI
PM _{2.5} , personal (µg/m ³ ; 8-hr time-weighted average)	58	1.19	0.73 to 1.95	58	1.19	0.72 to 1.95	5	4 1.07	0.62 to 1.82	57	1.20	0.73 to 1.97
PM _{2.5} , indoor (µg/m ³ ; 8-hr time-weighted average)	57	1.22	0.84 to 1.76	57	1.26	0.85 to 1.86	5	4 1.22	0.82 to 1.82	56	1.31	0.88 to 1.96
Carbon monoxide, indoor 1- hr maximum (ppm)	54	1.47	0.86 to 2.53	54	1.45	0.84 to 2.52	5	1 1.52	0.87 to 2.65	53	1.50	0.86 to 2.61
Stove type (Traditional vs. Improved)	79	5.24	1.68 to 16.30	79	5.59	1.73 to 18.06	5	5 4.49	1.28 to 15.77	77	5.20	1.60 to 16.84
Stove scale High quality High-mid quality Low-mid quality Low quality		NA			NA			NA			NA	

NA, Unstable estimates; PM, particulate matter; Estimates for $PM_{2.5}$ and carbon monoxide are per IQR increase: personal $PM_{2.5}$ (106.1 µg/m³), indoor $PM_{2.5}$ (572.3 µg/m³), and indoor carbon monoxide 1-hr max (4.62 ppm)

Table 4.51.01. Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures (including copollutant models) and the usual presence of a headache as assessed by the question, "Do you usually develop a headache during cooking?"

	1	Age-adjus	sted Estimate
	N	OR	95% CI
$PM_{2.5}$, indoor (µg/m ³ ; 8-hr time-weighted average)	57	1.26	0.85 to 1.86
Carbon monoxide (CO), indoor 1-hr maximum (ppm)	54	1.45	0.84 to 2.52
Model with both indoor PM _{2.5} and carbon monoxide 1-hr maximum	53		
PM _{2.5}		1.00	0.54 to 1.84
Carbon monoxide 1-hr max		1.56	0.68 to 3.62

PM, particulate matter; Estimates for PM_{2.5} and carbon monoxide are per IQR increase: indoor PM_{2.5} (572.3 μ g/m³) and indoor carbon monoxide 1-hr max (4.62 ppm)

Table 4.52. Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures and the usual presence of shortness of breath as assessed by the question, "Are you troubled by shortness of breath?"*

	Crude Estimate		Age-adjusted Estimate			Adjusted estimate including Outdoor Temperature			Adjusted estimate eliminating those taking bronchodilators			
	N	OR	95% CI	N	OR	95% CI	N	OR	95% CI	N	OR	95% CI
$PM_{2.5}$, personal ($\mu g/m^3$; 8-hr time-weighted average)	58	0.92	0.57 to 1.49	58	0.94	0.58 to 1.54	54	1.04	0.61 to 1.75	57	0.95	0.58 to 1.55
$PM_{2.5}$, indoor ($\mu g/m^3$; 8-hr time-weighted average)	57	1.19	0.77 to 1.85	57	1.33	0.85 to 2.08	54	1.31	0.84 to 2.05	56	1.36	0.86 to 2.14
Carbon monoxide, indoor 1- hr maximum (ppm)	54	1.73	0.84 to 3.57	54	2.05	0.95 to 4.42	51	1.78	0.88 to 3.60	53	2.12	0.98 to 4.57
Stove type (Traditional vs. Improved)	79	1.79	0.69 to 4.66	79	2.33	0.83 to 6.57	55	2.87	0.83 to 9.90	77	2.24	0.80 to 6.29
Stove scale												
High quality	23	$\operatorname{REF}^{\ddagger}$		23	\mathbf{REF}^{\ddagger}		20	\mathbf{REF}^{\ddagger}		22	REF [‡]	
High-mid quality	17	6.07	1.36 to 27.05	17	5.59	1.23 to 25.36	10	7.14	1.03 to 49.46	17	6.02	1.31 to 27.69
Low-mid quality	15	3.58	0.87 to 14.65	15	4.43	1.02 to 19.21	8	3.13	0.51 to 19.30	15	4.61	1.07 to 19.95
Low quality	24	3.90	1.13 to 13.45	24	4.58	1.26 to 16.70	17	7.28	1.49 to 35.54	23	4.45	1.22 to 16.20

*Combined answers for shortness of breath when "hurrying on the level or walking up a slight hill" and "walking at your own pace on the level;" [‡]P-value for trend < 0.05; PM, particulate matter; REF, reference category; Estimates for PM_{2.5} and carbon monoxide are per IQR increase: personal PM_{2.5} (106.1 μ g/m³), indoor PM_{2.5} (572.3 μ g/m³), and indoor carbon monoxide 1-hr max (4.62 ppm)

Table 4.52.01. Odds Ratios (OR) and 95% confidence intervals (CI) for the associations between cookstove exposures (including copollutant models) and the usual presence of shortness of breath as assessed by the question, "Are you troubled by shortness of breath?"*

		Age-adjus	sted Estimate
	N	OR	95% CI
$PM_{2.5}$, indoor (µg/m ³ ; 8-hr time-weighted average)	57	1.33	0.85 to 2.08
Carbon monoxide, indoor 1-hr maximum (ppm)	54	2.05	0.95 to 4.42
Model with both indoor $PM_{2.5}$ and Carbon monoxide 1-hr maximum	53		
PM _{2.5}		0.83	0.37 to 1.86
Carbon monoxide 1-hr max		2.48	0.68 to 8.97

*Combined answers for shortness of breath when "hurrying on the level or walking up a slight hill" and "walking at your own pace on the level;" PM, particulate matter; Estimates for PM_{2.5} and carbon monoxide are per IQR increase: indoor PM_{2.5} (572.3 μ g/m³) and indoor carbon monoxide 1-hr max (4.62 ppm)

Symptoms	FEV_{I}										
	Crude mean	95% CI	P- value**	Adjusted mean*	95% CI	P- value**					
Current Nasal											
Irritation***											
None	1.99	1.86 to 2.12		2.01	1.93 to 2.09						
Mild to Severe	2.23	1.92 to 2.53	0.1573	2.10	1.91 to 2.29	0.3821					
Current Amount of Mucus or Phlegm											
None	2.02	1.89 to 2.15		2.03	1.95 to 2.11						
Mild	2.14	1.75 to 2.54	0.5492	2.03	1.79 to 2.27	0.9926					
Moderate to Severe	2.02	1.51 to 2.53	0.9972	2.04	1.73 to 2.36	0.9454					
Current Shortness of Breath											
None	2.02	1.89 to 2.15		2.02	1.94 to 2.09						
Mild to Severe	2.14	1.75 to 2.53	0.5584	2.17	1.92 to 2.41	0.2448					
Current Chest Wheezing or Whistling***											
None	2.04	1.91 to 2.16		2.02	1.94 to 2.10						
Mild to Severe	2.02	1.57 to 2.46	0.9282	2.19	1.93 to 2.46	0.2130					
Current Throat Irritation											
None	2.07	1.93 to 2.21		2.04	1.95 to 2.13						
Mild	1.82	1.57 to 2.06	0.0719	1.95	1.79 to 2.11	0.3252					
Moderate to Severe	2.29	1.87 to 2.71	0.3331	2.17	1.91 to 2.44	0.3405					
Current Cough											
None	1.96	1.83 to 2.08		2.01	1.93 to 2.09						
Mild	2.39	2.08 to 2.70	0.0131	2.13	1.92 to 2.34	0.2860					
Moderate to Severe	2.33	1.75 to 2.91	0.2163	2.20	1.83 to 2.57	0.3101					

Table 4.53. Differences in mean Forced Expiratory Volume in 1 second (FEV₁; Liters)* for participants with and without current respiratory symptoms (N = 52).

* Mean lung functions adjusted for age and height

P-value comparing the FEV₁ means, symptoms versus no symptoms *N = 51

Symptoms	PEF							
	Crude mean	95% CI	P- value**	Adjusted mean*	95% CI	P- value**		
Current Nasal				<u></u>				
Irritation***								
None	255.8	237.5 to 274.0		257.3	240.0 to 274.6			
Mild to Severe	274.3	231.9 to 316.6	0.4247	266.1	225.6 to 306.6	0.6904		
Current Amount of Mucus or Phlegm								
None	257.6	239.3 to 275.9		258.3	241.0 to 275.5			
Mild	267.6	213.4 to 321.8	0.7272	260.1	208.7 to 311.4	0.9470		
Moderate to Severe	278.7	208.7 to 348.6	0.5613	281.8	214.2 to 349.4	0.5024		
Current Shortness of Breath								
None	256.6	239.3 to 273.9		256.3	240.0 to 272.6			
Mild to Severe	289.6	236.5 to 342.7	0.2414	292.8	241.0 to 344.6	0.1861		
Current Chest Wheezing or Whistling***								
None	259.2	241.5 to 277.0		258.2	241.6 to 274.8			
Mild to Severe	270.8	210.0 to 331.5	0.7161	282.7	224.9 to 340.5	0.4174		
Current Throat Irritation								
None	261.4	242.6 to 280.2		260.0	241.9 to 278.0			
Mild	233.1	200.6 to 265.6	0.1360	239.1	206.6 to 271.6	0.2722		
Moderate to Severe	325.3	268.9 to 381.6	0.0357	320.4	265.8 to 375.0	0.0387		
Current Cough								
None	249.7	232.8 to 266.6		252.5	235.9 to 269.0			
Mild	294.1	252.3 to 336.0	0.0538	279.3	235.9 to 322.8	0.2604		
Moderate to Severe	355.5	277.2 to 433.8	0.0108	348.6	272.3 to 424.9	0.0170		

Table 4.54. Differences in mean Peak Expiratory Flow (PEF; L/minute)* for participants with and without current respiratory symptoms (N = 52).

* Mean lung functions adjusted for age and height

P-value comparing the PEF means, symptoms versus no symptoms *N = 51

Symptoms	C-reactive protein							
	Crude mean	95% CI	P- value**	Adjusted mean*	95% CI	P- value**		
Current Nasal				······································				
Irritation**								
None	4.10	2.75 to 6.11		3.94	2.77 to 5.58			
Mild to Severe	4.44	1.93 to 10.28	0.8554	5.26	2.51 to 11.13	0.4809		
Current Amount of								
Mucus or Phlegm								
None	3.49	2.41 to 5.05		3.56	2.56 to 4.95			
Mild	12.06	3.29 to 43.82	0.0713	9.12	2.83 to 29.08	0.1286		
Moderate to Severe	11.02	3.00 to 40.04	0.0938	11.59	3.53 to 38.09	0.0627		
Current Shortness of Breath								
None	4.06	2.80 to 5.87		4.18	3.00 to 5.75			
Mild to Severe	4.90	1.45 to 16.61	0.7679	3.82	1.26 to 11.70	0.8881		
Current Chest Wheezing or Whistling**								
None	3.71	2.59 to 5.31		3.82	2.75 to 5.26			
Mild to Severe	15.33	4.18 to 56.83	0.0398	10.70	3.22 to 35.16	0.1024		
Current Throat Irritation								
None	3.10	2.05 to 4.71		3.42	2.36 to 5.00			
Mild	8.85	4.14 to 19.11	0.0189	5.64	2.66 to 12.06	0.2479		
Moderate to Severe	6.17	2.25 to 17.12	0.2145	7.39	2.89 to 19.11	0.1324		
Current Cough**								
None	3.67	2.46 to 5.47		3.53	2.48 to 4.95			
Mild	4.26	1.58 to 11.36	0.7794	5.00	2.10 to 11.82	0.4578		
Moderate to Severe	11.82	3.53 to 39.65	0.0697	13.60	4.53 to 40.45	0.0227		

Table 4.55. Differences in geometric mean C-reactive protein (CRP; mg/L on dried blood scale)* for participants with and without current respiratory symptoms (N = 71).

*Geometric mean CRP adjusted for age, height, and waist circumference

**P-value comparing the natural logarithm transformed CRP means, symptoms versus no symptoms

**N = 70

CHAPTER 5

DISCUSSION

<u>General</u>

In this study of Honduran women using traditional or improved Justa stoves, women using traditional stoves reported symptoms of cough, phlegm, wheeze, headache, and shortness of breath more frequently than those using improved stoves. Associations remained elevated, although attenuated compared to those examining stove type, for the association of measured indoor $PM_{2.5}$ and carbon monoxide with symptoms. Associations observed between cookstove exposures and lung function or CRP were consistent with null associations. However, some analyses resulted in wide confidence intervals and associations within the confidence interval ranges cannot be refuted. The use of an improved stove resulted in a 63% decrease in personal PM_{2.5}, a 73% decrease in indoor PM_{2.5}, and a 90% decrease in indoor carbon monoxide levels as compared to the traditional stove. The smaller percent decrease associated with the personal PM2.5 exposure as compared to the indoor kitchen exposure has been demonstrated previously and is expected based on typical time-activity patterns of participants because women will spend different amounts of time in and out of the kitchen (McCracken et al. 2007). Of note, however, is that even the reduced pollutant levels (mean personal $PM_{2.5} = 73.56$ $\mu g/m^3$; mean indoor PM_{2.5} = 266.24 $\mu g/m^3$) for the improved stoves are well above the current World Health Organization guideline for PM2.5 (25 µg/m³: 24-hr mean) as defined in the Global Update for 2005 (WHO 2005) (although the average concentrations would

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

likely have decreased if 24-hr averages would have been measured as opposed to eighthour averages). (The levels for both improved and traditional stoves were below the World Health Organization guideline for carbon monoxide (9 ppm: eight-hour average) (WHO 2002).) Similarly elevated indoor PM_{2.5} levels (as compared to the World Health Organization guidelines) are typical for improved biomass burning cookstoves and have been observed among wood and dung burning cookstoves in India (Chengappa et al. 2007; Dutta et al. 2007), wood burning cookstoves in Mexico (Masera et al. 2007), and wood burning cookstoves in Guatemala (Albalak et al. 2001; McCracken et al. 2007; Nacher et al. 2000a).

The utility of the four-level stove scale appears to be important as a clear trend of decreasing pollutants was observed as the quality of the stove improved. The stove scale was assessed on a scale representing potential for indoor emissions, based on factors such as chimney and plancha (griddle) condition and maintenance. The ability of the stove scale to better predict quantitative exposure measurements as compared to the dichotomous stove type variable (traditional versus improved) indicates the importance of maintaining the condition of the chimney and plancha following the introduction of an improved stove. In a previous study, a non-significant 7% increase in emissions of ten improved stoves in Guatemala was reported over an eight month time period; however, routine repairs to the chimneys and planchas were performed over the study period (Albalak et al. 2001). The authors suggested that deterioration of improved stoves, in general, could be due to improper maintenance as well as building material and construction flaws (Albalak et al. 2001). This information combined with the stove scale

results of the present study (in which no stove maintenance had been provided) stresses the importance of properly training the families as well as local artisans in the practice of stove maintenance and construction.

In multivariate models, the stove scale and ventilation factors combined predicted more than 50% of the variation in personal and indoor PM_{2.5} and 85% of the variation in indoor carbon monoxide. These are well above the 26% variation explaining indoor PM_{10} as reported by Riojas-Rodriguez and colleagues; however, the authors noted a limitation of assessing an improved stove that had been introduced, on average, five years prior to the study and was relatively poorly functioning (Riojas-Rodriguez et al. 2001). Again, this difference stresses the importance of stove maintenance. In addition to the stove scale, other factors predicting exposure measurements were the age of the stove (personal $PM_{2.5}$); the total area of the kitchen windows, the number of kitchen walls, and the primary material of the kitchen walls (indoor $PM_{2.5}$); and the volume of the kitchen and the number of walls with eave spaces (indoor carbon monoxide 1-hr max). Studies performed on improved wood-burning stoves in Guatemala indicated that reductions in indoor pollutant concentrations were influenced by the age of the stove (Naeher et al. 2000a). Other investigators have indicated the importance of ventilation factors similar to those of the present study, such as the number of windows and the type of kitchen (Balakrishnan et al. 2002; Riojas-Rodriguez et al. 2001). The ease with which this information on stove quality and kitchen ventilation can be collected could provide a cost-effective alternative to the more cost and time-intensive pollutant measurements. However, it is likely that these conditions change over cultural and geographic

211

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

boundaries. Therefore, future studies should validate the use of stove and ventilation factors in the same location in which larger epidemiologic investigations utilizing these variables as an exposure assessment technique will occur. To increase the utility of a subjective measure such as a stove scale, future studies should identify clear definitions of stove quality for each level of the scale. For example, photos of plancha and chimney conditions typical for each level may aid in a more accurate and consistent identification process for field investigators. This should decrease both inter- and intra-rater variability which is especially important for larger-scale and longer-term studies.

It is not clear as to why the data were better able to predict carbon monoxide as compared to $PM_{2.5}$ measurements. It is possible that the particulate matter monitors were less accurate than the carbon monoxide monitors. It is also possible that the monitors were measuring unidentified sources of $PM_{2.5}$; for example, burning trash, traffic, or other sources of incomplete combustion (WHO 2007). If such unidentified sources were present and contributing to the measured levels of $PM_{2.5}$, then this may partly explain the observed reductions in model R-square values.

For the most part, associations consistent with the null were observed between cookstove exposures and lung function. Stratification by the age of the stove, ventilation factors, or personal factors such as medication use, did not meaningfully alter the estimates. Due to the small sample size, only first-order stratifications were considered. It is possible that second or even third-order stratifications are necessary to elucidate the true relationship between the exposures and health endpoints. Another limitation of the sample size is that the influence of the presence of usual symptoms or history of symptoms on the relationship between cookstove exposure and lung function could not be assessed. An increased risk of airflow obstruction due to ambient air pollution has been described among susceptible populations with respiratory disease, such as chronic obstructive pulmonary disease (Brauer et al. 2001; Pope and Kanner 1993; Pope et al. 1995b). Only four women reported having cough or phlegm on most days for three consecutive months or more during the year; therefore, similar associations limited to a potentially more susceptible group could not be assessed. Additionally, only 68% of the study population was able to provide a successful lung function maneuver. This may have been a result of difficulties experienced in translating the procedure to participants. The women that did not provide successful maneuvers were, however, evenly distributed across the stove scale. Therefore, if women unable to perform the maneuver were, in fact, those with reduced lung function, then this missing information is not expected to result in a substantial bias although associations may be attenuated.

Another potential limitation of the health results is that our use of education level as an indicator of socio-economic status may not have been a valid proxy, thus resulting in residual confounding. Studies in Guatemala have examined potentially more culturally-appropriate indicators of socio-economic status. These include an asset index incorporating the possession of a radio, a television, and/or a bicycle, as well as owning pigs or cattle (Bruce et al. 1998; Díaz et al. 2007; McCracken et al. 2007). Culturally-appropriate indicators of socio-economic status are likely different across regions of the world and should be assessed accordingly. A method of reducing potential confounding

by factors such as socio-economic status is to conduct a randomized community trial (Smith et al. 2006). This is one of the few methods to adequately control for this type of confounding as the shift to improved stoves or cleaner fuels is usually accompanied by changes in other indicators of socio-economic status that may also influence health. Therefore, it may not be possible to separate these effects from those of the stove or fuel change in observational studies (Bruce et al. 1998; Regalado et al. 2006). Another option that avoids long follow-up periods in households without improved stoves (as controls) is to collect data before and after improved stove installation (without controls) which may require longer sampling periods although fewer houses may need to be assessed (Edwards et al. 2007).

A limitation of the lung function analyses is that values of liters and liters/minute for FEV₁ and PEF, respectively, were used in main analyses instead of the percentage of the predicted lung functions. There may not be an appropriate reference equation to calculate the percent-predicted lung functions for this population. Investigators comparing the height-adjusted forced vital capacity of Quechuan natives (highlanders of South America) and expatriate Europeans and North Americans born and raised at high altitudes reported significantly greater forced vital capacity among the native Quechuan (Greksa 1996). Other investigators examining the relationship between lung function and biomass exposures among Mexican women used Mexican standard reference equations (Pérez-Padilla et al. 2001) but reported that these equations were very similar to the third National Health and Nutrition Examination Survey values for Mexican-Americans (Ramirez-Venegas et al. 2006). In a study among rural Mexican women, Regalado and

colleagues reported FEV_1 in liters and also as percent of the predicted value by internally derived equations obtained using multivariate linear regression among apparently healthy participants (Regalado et al. 2006). Still, it is common in the cookstove literature for investigators to report FEV₁ and PEF in units of liters or liters/minute, respectively, due to the extreme diversity of populations studied. It is not clear if Honduran women could appropriately be compared to another population and the sample size was too small to calculate internally derived prediction equations; therefore, values in liters and liters/minute of lung functions adjusted for age and height were used and relative comparisons within the study population were evaluated in the main analyses. Mean lung function values were comparable to those reported in studies examining similar relationships among Mexican women (FEV₁ mean among women using biomass = 1.98L; SD = 0.53 L) (Regalado et al. 2006); and among Indian women (FEV₁ mean among women burning wood = 1.98 ± 0.63 L) (Saha et al. 2005); but were somewhat lower as compared to Guatemalan women (FEV₁ mean among women burning wood = 2.64 L; SD = 0.34 L) (Smith-Sivertsen et al. 2006). Changes in liters for FEV_1 and liters/minute for PEF may not be as easily interpreted as cut-offs typically used when percentage of the predicted values are reported. In addition, values may not be comparable to other populations as differences in lung function in racial/ethnic groups are well-documented even among a sample of the United States population (Hankinson et al. 1999). In spite of the absence of an appropriate reference population, sensitivity analyses were performed by calculating predicted lung functions based on Mexican-American reference equations (Hankinson et al. 1999). This allowed for the categorization of women into above or below 80% predicted FEV1, a cut-point often used to assess classifications of chronic obstructive pulmonary disease (Pauwels et al. 2001). Interpretations were similar to those of the FEV_1 linear regression analyses; however, results of the sensitivity analyses should be interpreted with caution because it is not clear if Honduran and Mexican-American women have similar lung capacities and therefore, the 80% predicted cut-point may not indicate a disease state in this population.

Personal $PM_{2.5}$ was associated with both FEV_1 and PEF in the direction opposite to that hypothesized (e.g. elevated personal PM_{2.5} levels were associated with a 0.07 liter increase in FEV_1). Similarly, among some subgroups of women (such as those exposed to second-hand smoke and those taking any medications), elevated coefficients were also observed in the direction opposite to that hypothesized. It is likely that the increases in lung function observed when exposures increased are not clinically meaningful and it is also possible that due to the number of analyses, some results may be due to chance. However, this unexpected result may partly be explained by the inability of a crosssectional study design to determine temporality. It is possible that women already experiencing adverse health effects were the women who received the improved stoves. Stratifying analyses on time since receiving the stove was expected to provide an estimate of the association between exposure and health outcome that was not influenced by temporality because women having the stoves for longer periods of time may have already experienced health benefits. However, if adverse heath endpoints are irreversible (as indicated in chronic obstructive pulmonary disease) then stratifying by time since receiving the stove will not influence the estimates. Another limitation of this method is that stratifying by this variable decreased the range of exposures to be compared by

limiting the improved stoves to those that may have deteriorated over time. Estimates did not change upon stratification by time since receiving the stove. Collecting baseline health data before the improved stoves are introduced into the population could overcome this limitation. Additionally, the unexpected results may have been due to exposure misclassification. Personal $PM_{2.5}$ measurements are likely more influenced by day-today variation as compared to indoor pollutant concentrations. However, measurement error is expected to have been random and therefore, would most likely not explain the positive association between personal PM_{2.5} and lung function. Misclassification of the lung function values is also a possibility as lung functions were not different among women with and without current symptoms as has been reported in some occupational epidemiologic studies (Christiani et al. 2001; Musk et al. 2000), although the correlation is poor among asthmatics (Teeter and Bleecker 1998). Measurement error associated with lung function measures is likely random and (if assessed as an isolated misclassification) would likely lead to attenuation of results. It is important to note that increasing age was associated with decrements in lung function and increasing height was associated with increases in lung function, as would be expected (Hankinson et al. 1999); and therefore, some confidence can be placed in the lung function values. In addition, age and height did seem to influence the cookstove exposure and lung function estimates. It is also a possibility that study results are due to chance.

In similar cross-sectional studies, associations between biomass burning and lung function have been variable or small (Malik 1985; Norboo et al. 1991; Pandey et al. 1985; Qureshi 1994; Reddy et al. 2004; Regalado et al. 2006; Rinne et al. 2006). Similar

results were reported in a randomized trial (Smith-Sivertsen et al. 2006). Explanations for the lack of consistent associations despite elevated exposures are unknown (Regalado et al. 2006). Similarly small physiological, although statistically significant, declines in lung function are common in the ambient air pollution literature (Pope et al. 1995a; Pope et al. 1995b) although relatively larger effects have been observed among susceptible populations (Brauer et al. 2001). It is possible that biomass smoke has no or low impact on lung function or that women chronically exposed to biomass have developed reduced susceptibility (Regalado et al. 2006), potentially similar to adaptive mechanisms that have been described in smokers (Jones et al. 1980). Another hypothesis is that the important time frame for exposure is during childhood and all women likely experienced similar exposures due to the use of traditional stoves as children (Rinne et al. 2006). Differences in current exposures (from the use of traditional or improved stoves) may not have an effect on lung functions as it is thought that the adult lung is not as susceptible to air pollution as compared to the lungs of children (Rinne et al. 2006; USEPA 1992). It is also possible that the detection of differences among groups of women was limited by low power. Future studies should continue to utilize improved study designs with repeated measures. Although less ideal study designs may not explain the lack of association entirely as there is strong evidence of an association between solid fuel use and acute lower respiratory infections in children and chronic obstructive pulmonary disease in adult women among cohort, case-control, and cross-sectional studies (Smith et Another potential explanation for the lack of associations involves the al. 2004). difficulty in performing effort-dependent lung function maneuvers; therefore,

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

improvements in culturally acceptable means of coaching participants through the testing may improve the validity of the endpoints.

Although not always statistically significant, a suggestive association between increased cookstove exposures and increased reporting of symptoms was observed. Usual symptoms of cough, phlegm, wheeze, headache, and shortness of breath were associated with cookstove exposures while current nasal irritation was not. For example, adjusted odds ratios for indoor PM_{2.5} ranged from 1.26 for the presence of phlegm and headache to 1.64 for the presence of wheeze. Odds ratios for the low quality as compared to the high quality stove scale were 4.58 for shortness of breath and 9.02 for the presence of cough. There was a dose-response relationship between the odds of exposure and deteriorating stove quality among those reporting cough as compared to those not reporting cough. Small cell frequencies limited the ability to examine this relationship among all symptom types. Interpretations of observed odds ratios are limited due to the instability of the logistic regression models. Another potential limitation of these analyses is the likelihood of reporting or recall bias. Women were aware of the relationship between cookstoves and adverse health with 76% reporting that they were concerned that the smoke from cookstoves adversely effects health. Therefore, those with traditional stoves may have been more likely to report symptoms as compared to those with improved stoves likely leading to a bias away from the null. It was originally thought that associations among women not reporting a concern about cookstove health effects would not be as influenced by reporting bias; however, due to the small number of women reporting no concern, interpretations of results among this subgroup of women are

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

limited. The observed relationships add to the existing literature that has demonstrated a relationship between symptoms (often assessed as chronic bronchitis) and cookstove exposures (Smith et al. 2004). Previous studies have been limited by the use of proxies, such as the use of biofuel for cooking, cooking inside versus outside, and time spent near the fire, to evaluate exposure to indoor air pollution (Smith et al. 2004). Investigators performing quantitative indoor PM_{10} measurements reported no associations between PM₁₀ and respiratory symptoms (Riojas-Rodriguez et al. 2001). A randomized trial reported that the use of an improved stove (median carbon monoxide in exhaled breath was significantly lower in women using improved stoves as compared to traditional stoves) resulted in decreased symptoms of headache and sore eyes (Díaz et al. 2007). One reason for differences in observed relationships between reported symptoms and cookstove exposures is the potential for variation in the understanding of terms used to describe symptoms, particularly because rural areas of developing countries are often characterized by low literacy and poor access to health care (Thompson et al. 2007). Culturally valid surveys utilizing terms familiar to the participants should increase the accuracy of assessing reported symptoms (Thompson et al. 2007).

The laboratory validation for the CRP assay in dried blood was performed in the Human Performance/Clinical Research Laboratory (HPCRL) at CSU. Although only preliminary results are completed, several limitations have been identified (Robinson et al. 2007). The R-square (R-square = 0.75) comparing dried-blood CRP to venous-drawn plasma CRP was considerably lower than that detected by a previously validated method (R-square = 0.92) (McDade et al. 2004). The enzyme linked immunosorbent assay kit used

had high percent coefficients of variation (CV%) for low concentrations of CRP; those which fall within the expected range of most human CRP concentrations. As a result, CRP levels for the present study should be interpreted with caution. Of note, however, is that CRP concentrations were elevated among women reporting more severe current respiratory symptoms. These findings are consistent with investigations reporting associations between elevated CRP and chronic bronchitis or airflow obstruction (Aronson et al. 2006; Kony et al. 2004; Sin and Man 2003). Although a suggestive trend was observed, the clinical value of the changes is unknown. Measurement error is likely random which may lead to an attenuation of the results. Future studies should utilize a more sensitive CRP assay, such as the newly validated protocol by McDade and colleagues with only slight revisions made to the previously validated protocol (McDade et al. 2004; Robinson et al. 2007).

Similar to the lung function results, associations consistent with the null were observed between cookstove exposures and CRP. Again, statistical significance was reported for a couple of stratified subgroups (among women residing in Suyapa and among women cooking in kitchens without windows). In both subgroups, increased cookstove exposures were associated with decreased CRP levels. These changes are likely not clinically meaningful and results should be interpreted carefully as CRP was transformed (natural logarithm) for analyses and the validation study indicated that dried blood CRP was elevated more than four-fold as compared to plasma CRP. The need for additional investigations to determine the magnitude, if any, of the relationship between cookstove exposures and cardiovascular health is great (Smith 2002). To our knowledge, this is only the third study to assess this relationship among women cooking with biomass fuels in a non-controlled setting. As part of a chimney stove intervention study, the use of an improved stove was associated with a 3.7 mm Hg (95% CI: -8.1 to 0.6) reduction in systolic blood pressure and a 3.0 mm Hg (95% CI: -5.7 to -0.4) reduction in diastolic blood pressure as compared to the use of traditional open fires (McCracken et al. 2007). In 2006, Ray and colleagues performed a study among Eastern Indian women and compared those cooking with biomass fuel to those cooking with liquid petroleum gas (Ray et al. 2006). Among biomass users as compared to liquid petroleum gas users, the authors reported increased activation of platelets and formations of platelet-leukocyte complexes, which are risk factors for cardiovascular disease (Ray et al. 2006). A limitation noted by the authors was that they were not able to account for socio-economic factors (Ray et al. 2006). In addition, a controlled study of human exposure to wood smoke and cardiovascular disease-related endpoints has been performed (Barregard et al. 2006). Thirteen participants were exposed to a four-hour session of 200-300 μ g/m³ PM_{2.5} and a four-hour session of "clean" air, spaced one week apart. The authors reported small exposure-related changes in inflammatory mediators, such as Serum Amyloid A and Factor VIIIc and, although not statistically significant, a 10% increase in CRP levels was consistently observed three hours after exposure to wood smoke as compared to the same amount of time following "clean" air exposure (Barregard et al. 2006). It is likely that the global burden of disease due to indoor air

222

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

pollution will be even greater once the cardiovascular disease health impacts are more clearly understood (Smith 2002).

Given the ubiquity of biomass smoke exposures around the world along with the prevalence of cardiovascular disease, future study of this relationship is warranted. Evaluation of this association is limited due to the number of studies as well as lack of a variety of cardiovascular health endpoints assessed. However, several studies of ambient air pollution have been performed. A consistent link between ambient air pollution and cardiovascular mortality and morbidity has been demonstrated through numerous epidemiologic studies (e.g., Dominici et al. 2006; Metzger et al. 2004; Peel et al. 2007; Samet et al. 2000a). The mechanisms underlying these associations are not completely understood, but could involve induction of systemic inflammatory markers leading to increased blood coagulability and/or atherosclerotic plaque progression or disruption (Bai et al. 2007; Brook et al. 2004; Seaton et al. 1999). Several studies suggest that ambient air pollution is associated with increased levels of inflammatory markers in blood (Liao et al. 2005; O'Neill et al. 2007; Pekkanen et al. 2000; Peters et al. 1997; Peters et al. 2001a; Peters et al. 2000; Salvi et al. 1999; Schwartz 2001; Zeka et al. 2006).

Inflammatory mechanisms play a central role in mediating all phases of cardiovascular disease (Blake and Ridker 2003; Fortmann et al. 2004; Vasan 2006). As a result, investigators have focused on identifying biological markers, or biomarkers, of vascular inflammation that may help to identify those at high risk for future cardiovascular events (Blake and Ridker 2003; Ridker et al. 2000; Vasan 2006). In addition to gaining insight

into the pathology of cardiovascular disease, another advantage of collecting biomarkers as outcomes of interest is that they may be gathered in a shorter time frame and with less cost as compared to endpoints such as morbidity or mortality that would require much larger sample sizes (Vasan 2006). A disadvantage of the assessment of biomarkers is the methodological limitations associated with venipuncture blood collection, especially when repeat samples are desired. Venipuncture is a relatively invasive procedure that requires trained phlebotomists. In addition, once blood is collected it often needs to be immediately assayed or centrifuged, separated, and frozen (McDade 2007). The minimally invasive use of dried blood spots, samples of whole blood collected on filter paper following a simple finger stick, provides an alternative method. In addition to the present study, several community-based studies have demonstrated this method to be a convenient and reliable means to blood collection, storage, and transportation (Cook et al. 1998; Erhardt et al. 2002; McDade et al. 2004; McDade and Shell-Duncan 2002; McDade et al. 2000; Worthman and Stallings 1994; Worthman and Stallings 1997). Laboratory methods have been validated for a large number of inflammatory markers in dried-blood spots (Mei et al. 2001; Skogstrand et al. 2005). The ease and convenience of this field method, utilizing finger-stick blood samples, could prove to be a useful tool for larger community-based, epidemiologic investigations especially in settings typical of developing countries (McDade et al. 2004; Mei et al. 2001; Parker and Cubitt 1999).

In summary, the use of dried blood spots collected via finger stick may provide a novel, reliable and convenient method for community-based applications. Subjects may be more likely to participate and also adhere to study protocol with this less invasive

approach to blood collection (Bryant Borders et al. 2007), which could lead to larger sample sizes and/or more repeated samples among participants. Although recent evidence supports the association between ambient air pollution and increased cardiovascular disease risk, several important questions remain: the need for a better understanding of the basic mechanisms and causal pathways leading to disease; the identification of differential toxicities of constituents and sources of air pollutants responsible for effects; and epidemiologic investigations are needed to address limitations of prior research (Brook et al. 2004). In addition, the presence of only three studies examining the relationship between indoor air pollution in developing countries and cardiovascular disease risk necessitates further study if the association is to be elucidated. The ease and convenience of collecting finger-stick dried blood samples to evaluate inflammation, especially in field settings with limited storage capabilities, as well as the ability to assess many inflammatory analytes in a single dried blood spot, may prove invaluable as this area of study is furthered.

Limitations

Although this study was originally planned as a pilot study to determine the feasibility of methodologies, the small sample size limits the interpretation of the results. It is possible that the elevated exposures (above the World Health Organization guidelines) measured even among the improved stove users limits the ability to detect health differences among the women if both improved and traditional stove users experience similar chronic adverse health. There may be an exposure threshold associated with adverse health effects that is below that which was measured for all study participants. As mentioned

previously, it is also possible that women have adapted to life-long elevated cookstove exposures or that the critical exposure time periods occurred during childhood when all women were exposed to similar indoor pollutant concentrations from traditional stoves (Regalado et al. 2006; Rinne et al. 2006). Because improved stove users were older than traditional stove users, another explanation for the lack of health effect associations is the potential for residual confounding by age. In addition, due to the number of statistical tests performed, the possibility that results are due to chance cannot be excluded.

The limitations of the cross-sectional study design include the use of prevalence data, the potential for survivor bias, as well as the inability of investigators to assess temporality. The use of prevalence may not be a limitation in this study as assessments of symptoms, lung function, and CRP are often performed in similar manners especially when the goal of the study is to assess the prevalence of chronic health endpoints. However, repeated measures would improve confidence in identifying chronic conditions as compared to one-time measures. Assessing exposure on one day only may cause measurement error if these measures are not typical of usual longer-term exposures. However, any measurement error is expected to be random which may lead to an attenuation of associations. Survivor bias may be a possibility if women more susceptible to the effects of wood smoke died at an early age. If exposures remained consistent over the lifetime of the women (for example if cookstove exposures did not change from early life through the study period) then this could bias the results toward the null (or even result in associations with exposures appearing protective). However, if more susceptible women died at early ages, it is likely that the present day use of either improved or traditional stoves for these women would have been random. As mentioned previously, the inability to assess temporality may limit study results because women experiencing adverse health effects may have received the improved stoves before apparently healthy women. If these adverse health effects were irreversible, then study results may be biased toward the null or could even explain why exposures appear protective for some analyses. Another limitation of the study design was that outcome measures were not taken at the beginning of the day prior to the start of the cookstove fire. This would have allowed for the analysis of the change in health endpoints from a relatively low exposure due to overnight pollutant concentrations to cookstove exposures experienced throughout the day. The initial study design was to collect pre- and post-exposure health endpoints; however, this was not undertaken once time constraints and desires of participants were more clearly understood. Although limitations of the study design are apparent, for the purposes of a feasibility study, the short time-frame and low cost associated with the cross-sectional study design were ideal and allowed for the validation of finger-stick dried blood spot samples collected in the field.

It is not known that many women refused participation. However, the participants were not randomly selected and therefore, there is a possibility that selection bias exists. For example, if a higher percentage of exposed women with adverse health from the source population participated as compared to the percentage of women from the source population in other categories of exposure and health, then it is possible that estimates are biased away from the null. The use of a health endpoint, such as CRP, that is generally unknown to participants should minimize potential selection bias for CRP estimates, as long as CRP is not related to symptoms. As previously discussed, it is also possible that women using traditional stoves may have recalled or reported symptoms more frequently than women using improved stoves, likely leading to a biased estimate away from the null. In addition, it is possible that carbon monoxide and PM_{2.5} levels on one day do not represent levels typically occurring throughout the year. However, the relative air quality differences among homes may stay consistent and the combination of stove quality, fuel type, and housing parameters may provide an accurate representation of longer-term exposures. Because improved stove designs vary across regions of the world, the four-level subjective stove scale may apply only to stoves similar, in design and use, to the *Justa* stove. However, given the utility of the stove scale as demonstrated in this study, similar methods can be easily adapted to investigations involving other improved stove designs.

CRP is a very general marker of inflammation, and although we attempted to account for factors regularly affecting CRP, it is possible that CRP levels were affected by insults other than air pollutant exposures. It is also possible, due to the elevated pollutant levels measured even among improved stove users, that all participants experienced chronically elevated CRP levels. Future studies should include multiple measures of CRP, and other specific markers of inflammation, in a larger population. However, in follow-up studies, levels of CRP were stable over long periods of time as long as measurements were not taken within two to three weeks of an acute infection (Macy et al. 1997; Ridker et al. 1999). Therefore, given the relative stability of CRP concentrations in individuals over time (Danesh et al. 2004), a single measure of CRP can provide meaningful information

on the level of chronic inflammation (McDade 2006). More importantly, CRP as measured in the present study is limited by the use of an enzyme linked immunosorbent assay that may not have been sensitive enough to detect differences in CRP concentrations within the normal range of human values. Future studies utilizing dried blood spots to assess CRP should use a more highly sensitive assay (McDade et al. 2004; Robinson et al. 2007).

Since associations were identified between pollutants and symptoms and the concentrations of $PM_{2.5}$ and carbon monoxide were moderately correlated (R-square = 0.69), then identifying whether or not the pollutants were acting as confounders of each other may not be possible. Co-pollutant models were assessed for symptom analyses. The odds ratios of indoor $PM_{2.5}$ remained elevated for cough, phlegm, and wheeze while the odds ratios of indoor carbon monoxide remained elevated for headache and shortness of breath. Potential interpretations of co-pollutant model results are that the pollutants with odds ratios remaining elevated are more important, measurement of that pollutant could involve less error, the pollutant could be a better surrogate for the real health damaging pollutant, or results could be random. While the issue of correlated pollutants is a limitation that should be addressed in further study involving larger populations, in practice the inability to identify the most health-damaging pollutant may be of less importance if interventions distributing improved stoves can reduce all pollutants associated with the burning of biofuels.
A limitation of the eight-hour exposure assessment is that, in the presence of large daily variation, observed short-term exposures may not accurately predict long-term exposures (Ezzati et al. 2000; McCracken et al. 2006). A recent validation study performed in Guatemala compared the predictive capabilities of a mixed model, which incorporated within-subject variation in addition to between-subject variation, to more commonly used estimates; which may be important for future studies assessing long-term exposures (McCracken et al. 2006). Another method to assess long-term exposures is to calculate hour-years which may be used as a potential confounder when examining acute health effects (Regalado et al. 2006) or as the exposure of interest when examining chronic health effects. An eight-hour time weighted exposure assessment for PM_{2.5} may also be limited because specific information on exposure peaks during cooking are not assessed. Intense peaks of short duration have been described in an African setting (Ezzati et al. 2000). Temporal patterns of cookstove exposures throughout the day are likely dependent on the climate and available resources. Honduran study participants reported that the fire was burning, on average, 12.7 hours a day which may indicate that most women kept the fire burning for long periods of time without many periods of start and stop. In addition, mean eight-hour carbon monoxide was highly correlated with mean carbon monoxide 1-hr maximum which indicates that the intense peaks of short duration, if present, would most likely not have influenced the exposure classification of the women. Exposure measurement error of $PM_{2.5}$, carbon monoxide, and the stove scale was likely random, potentially leading to an attenuation of the results.

230

Another potential limitation of the exposure measurements is that particulate matter composition and size was not assessed. Even though the use of the improved stove resulted in reductions in time-weighted average PM_{2.5}, there is the possibility that certain health-damaging components or smaller size-fractions of particulate matter were elevated. The composition of the organic carbon fraction of particulate matter varies dramatically based on specific fuels and combustion conditions (Naeher et al. 2007). Emission factors for specific organic compounds are also influenced by wood moisture and combustion conditions, and the relationships may not parallel those observed for fine particles (Guillen and Ibargoitia 1999; Khalil and Rasmussen 2003). In addition, given the substantial number of known health-damaging wood smoke pollutants (Naeher et al. 2007), it is also possible that PM_{2.5} and carbon monoxide were not the appropriate pollutants to measure for assessing the relationship between cookstove exposures and the measured health endpoints.

It is possible that exposure monitoring was limited in that it may not accurately reflect true usual exposures because behaviors may have changed due to the presence of the monitoring equipment (Naeher et al. 2000a). This most likely affected personal exposure monitoring (Naeher et al. 2000a) but could also have influenced the indoor measurements if cooking behaviors such as the amount of time the fire was burning changed. Asking participants whether or not their day would have been different if monitoring had not taken place was one attempt to overcome this limitation. No differences in results were observed when stratified by this information; however, this question is dependent on reporting by the participant so differences cannot be ruled out entirely. Future studies with repeated measures and longer exposure assessment periods may overcome the limitations associated with a one-time assessment, when monitoring is a novel experience for the participant.

Strengths & Summary

Approximately half of the world's population and up to 95% of rural households in developing countries still use biomass fuel for cooking and heating (Bruce et al. 2000; Ezzati and Kammen 2001b; Rehfuess et al. 2006). Modernization has been associated with a shift to cleaner fuels; however, even where cleaner and more sophisticated fuels have become available, households continue to use simple biofuels (Bruce et al. 2000). Even though improved stoves often utilize wood, many are designed to increase combustion efficiency while also meeting cultural needs (Bruce et al. 2000). Therefore, improved wood-burning stoves have the potential to significantly reduce the estimated 1.6 million premature deaths per year associated with biomass and coal smoke (Smith et al. 2004).

Most studies examining the relationship between biomass-derived indoor air pollution and adverse health effects have measured total suspended particulates or PM_{10} as indicators of exposure (Balakrishnan et al. 2002). This study was one of the few to assess personal $PM_{2.5}$, the particulate fraction associated with deposition in the lower airways, and therefore, likely a more relevant exposure when assessing adverse health effects. Stove quality and factors affecting ventilation are easily and inexpensively assessed when compared to measuring air quality concentrations; and results from this study indicate that these factors can account for a substantial amount of the variation in air quality measurements. Further study will be needed to determine whether or not the percent variation explained by stove and ventilation factors is adequate for assessing associated health effects. Large differences in emissions within stove types have been documented (Naeher et al. 2000a). The use of an investigator-assessed stove scale, while subjective, was able to account for a larger percentage of this variation as compared to the dichotomized stove-type variable. Results of this exposure assessment analysis could provide a cost-effective alternative to air quality monitoring for large-scale epidemiologic investigations of indoor air pollution and adverse health endpoints.

Although the CRP laboratory analysis was not well-validated, the field method of collecting a finger-stick blood sample was successful. Dried blood collected on filter paper provides a practical alternative, with regard to sample collection, storage, and transportation, to obtaining venous blood; making it especially appealing for field work in developing countries. Although cookstove exposure and CRP were not associated in this study, the ubiquity of biomass exposures around the world as well as the prevalence of cardiovascular disease necessitates more research on this topic. An association between indoor air pollution and CRP, or other biomarkers of inflammation, could support the biological plausibility of the potential relationship between biomass-derived indoor air pollution and cardiovascular disease endpoints in developing countries, an association that has seldom been examined.

Despite apparently large risks and populations involved, estimates concerning the health effects of biomass burning are associated with large uncertainty due to the lack of information on exposure data and the exposure to health effects associations (Zhang and Smith 2003). This is one of the few studies that have quantitatively assessed both personal and indoor air pollution levels and health effects while adjusting for potential confounders and the first to have examined the association between indoor air pollution and CRP levels in developing countries. In addition, although some studies have assessed the benefits of switching from biomass burning to cleaner fuel types (i.e. gas or electric), poor households currently relying on biomass burning are not likely to make the switch to cleaner fuels in the near future (Albalak et al. 2001; Dutt et al. 1996; Smith 1993). This is one of the few studies that have evaluated the health benefits of improved wood-burning stoves as compared to traditional wood-burning stoves. The dissemination of the Justa stove resulted in substantial reductions in PM2.5 and carbon monoxide and an association between reduced cookstove exposures and a decrease in reported symptoms was consistently demonstrated. Results may help to develop future stove intervention efforts as well as provide the foundation for larger-scale, prospective epidemiologic investigations. Extremely elevated exposures from biomass combustion in developing countries present a unique opportunity to gain insight into the complex mixtures of pollutants of interest to both developing and developed countries and to reduce substantial health risks to nearly three billion people, those representing the poorest populations in the world (Naeher et al. 2007).

234

CHAPTER 6

REFERENCES

- Ackermann-Liebrich U, Leuenberger P, Schwartz J, Schindler C, Monn C, Bolognini G, et al. 1997. Lung function and long term exposure to air pollutants in Switzerland. Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. Am J Respir Crit Care Med 155(1):122-129.
- Albalak R, Bruce N, McCracken JP, Smith KR, De Gallardo T. 2001. Indoor respirable particulate matter concentrations from an open fire, improved cookstove, and LPG/open fire combination in a rural Guatemalan community. Environ Sci Technol 35(13):2650-2655.
- Albalak R, Frisancho AR, Keeler GJ. 1999. Domestic biomass fuel combustion and chronic bronchitis in two rural Bolivian villages. Thorax 54(11):1004-1008.
- American Thoracic Society. 2005. Patient Information Series: Chronic Obstructive Pulmonary Disease. Am J Respir Crit Care Med 171:P3-P4.
- Aronson D, Roterman I, Yigla M, Kerner A, Avizohar O, Sella R, et al. 2006. Inverse association between pulmonary function and C-reactive protein in apparently healthy subjects. Am J Respir Crit Care Med 174(6):626-632.
- Bai N, Khazaei M, van Eeden SF, Laher I. 2007. The pharmacology of particulate matter air pollution-induced cardiovascular dysfunction. Pharmacol Ther 113(1):16-29.
- Baker MD, Henretig FM, Ludwig S. 1988. Carboxyhemoglobin levels in children with nonspecific flu-like symptoms. J Pediatr 113(3):501-504.
- Balakrishnan K, Sankar S, Parikh J, Padmavathi R, Srividya K, Venugopal V, et al. 2002. Daily average exposures to respirable particulate matter from combustion of biomass fuels in rural households of southern India. Environ Health Perspect 110(11):1069-1075.
- Ballard-Tremeer G, Jawurek HH. 1996. Comparison of five rural, wood-burning cooking devices: efficiencies and emissions. Biomass and Bioenergy 11:419-430.

- Barregard L, Sällsten G, Gustafson P, Andersson L, Johansson L, Basu S, et al. 2006. Experimental Exposure to Wood-Smoke Particles in Healthy Humans: Effects on Markers of Inflammation, Coagulation, and Lipid Peroxidation. Inhal Toxicol 18(11):845-853.
- Behera D, Dash S, Malik SK. 1988. Blood carboxyhaemoglobin levels following acute exposure to smoke of biomass fuel. Indian J Med Res 88:522-524.
- Behera D, Dash S, Yadav SP. 1991. Carboxyhaemoglobin in women exposed to different cooking fuels. Thorax 46(5):344-346.
- Behera D, Jindal SK, Malhotra HS. 1994. Ventilatory function in nonsmoking rural Indian women using different cooking fuels. Respiration 61(2):89-92.
- Bellia V, Pistelli F, Giannini D, Scichilone N, Catalano F, Spatafora M, et al. 2003. Questionnaires, spirometry and PEF monitoring in epidemiological studies on elderly respiratory patients. Eur Respir J Suppl 40:21s-27s.
- Blake GJ, Ridker PM. 2003. C-reactive protein and other inflammatory risk markers in acute coronary syndromes. Journal of the American College of Cardiology 41(4):37S-42S.
- Bobak M. 2000. Outdoor air pollution, low birth weight, and prematurity. Environ Health Perspect 108(2):173-176.
- Boy E, Bruce N, Delgado H. 2002. Birth weight and exposure to kitchen wood smoke during pregnancy in rural Guatemala. Environ Health Perspect 110(1):109-114.
- Boy E, Bruce N, Smith KR, Hernandez R. 2000. Fuel efficiency of an improved woodburning stove in rural Guatemala: implications for health, environment and development. Energy for Sustainable Development 4(2):21-29.
- Brandt-Rauf PW, Cosman B, Fallon LF, Jr., Tarantini T, Idema C. 1989. Health hazards of firefighters: acute pulmonary effects after toxic exposures. Br J Ind Med 46(3):209-211.
- Brauer M, Ebelt ST, Fisher TV, Brumm J, Petkau AJ, Vedal S. 2001. Exposure of chronic obstructive pulmonary disease patients to particles: respiratory and cardiovascular health effects. J Expo Anal Environ Epidemiol 11(6):490-500.
- Brody AR, Warheit DB, Chang LY, Roe MW, George G, Hill LH. 1984. Initial deposition pattern of inhaled minerals and consequent pathogenic events at the alveolar level. Ann N Y Acad Sci 428:108-120.
- Brook RD, Brook JR, Rajagopalan S. 2003. Air pollution: the "Heart" of the problem. Curr Hypertens Rep 5(1):32-39.

- Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, et al. 2004. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. Circulation 109(21):2655-2671.
- Bruce N, McCracken J, Albalak R, Schei MA, Smith KR, Lopez V, et al. 2004. Impact of improved stoves, house construction and child location on levels of indoor air pollution exposure in young Guatemalan children. J Expo Anal Environ Epidemiol 14 Suppl 1:S26-33.
- Bruce N, Neufeld L, Boy E, West C. 1998. Indoor biofuel air pollution and respiratory health: the role of confounding factors among women in highland Guatemala. Int J Epidemiol 27(3):454-458.
- Bruce N, Perez-Padilla R, Albalak R. 2000. Indoor air pollution in developing countries: a major environmental and public health challenge. Bull World Health Organ 78(9):1078-1092.
- Bruce N, Perez-Padilla R, Albalak R. 2002. The health effects of indoor air pollution exposure in developing countries WHO/SDE/OEH/02.05. Geneva: World Health Organization.
- Brunekreef B, Forsberg B. 2005. Epidemiological evidence of effects of coarse airborne particles on health. Eur Respir J 26(2):309-318.
- Brunekreef B, Holgate ST. 2002. Air pollution and health. Lancet 360(9341):1233-1242.
- Bryant Borders AE, Grobman WA, Amsden LB, Collins ET, Holl JL. 2007. Factors that influence the acceptability of collecting in-home finger stick blood samples in an urban, low-income population. J Health Care Poor Underserved 18(1):100-115.
- Budds J, Biran A, Rouse J. 2001. What's Cooking: a review of the health impacts of indoor air pollution and technical interventions for its reduction. Leicestershire: Water and Environmental Health at London and Loughborough.
- Burgess JL, Nanson CJ, Bolstad-Johnson DM, Gerkin R, Hysong TA, Lantz RC, et al. 2001. Adverse respiratory effects following overhaul in firefighters. J Occup Environ Med 43(5):467-473.
- Campbell H, Armstrong JR, Byass P. 1989. Indoor air pollution in developing countries and acute respiratory infection in children. Lancet 1(8645):1012.
- Celli BR, Snider GL, Heffner J, Tiep B, Ziment I, Make B, et al. 1995. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. American Thoracic Society. Am J Respir Crit Care Med 152(5 Pt 2):S77-121.
- Chauhan AJ, Johnston SL. 2003. Air pollution and infection in respiratory illness. Br Med Bull 68:95-112.

- Chengappa C, Edwards R, Bajpai R, Naumoff Shields K, Smith KR. 2007. Impact of improved cookstoves on indoor air quality in the Bundelkhand region in India. Energy for Sustainable Development XI(2):33-44.
- Chestnut LG, Schwartz J, Savitz DA, Burchfiel CM. 1991. Pulmonary function and ambient particulate matter: epidemiological evidence from NHANES I. Arch Environ Health 46(3):135-144.
- Christiani DC, Wang XR, Pan LD, Zhang HX, Sun BX, Dai H, et al. 2001. Longitudinal changes in pulmonary function and respiratory symptoms in cotton textile workers. A 15-yr follow-up study. Am J Respir Crit Care Med 163(4):847-853.
- Collings DA, Sithole SD, Martin KS. 1990. Indoor woodsmoke pollution causing lower respiratory disease in children. Trop Doct 20(4):151-155.
- Cook JD, Flowers CH, Skikne BS. 1998. An assessment of dried blood-spot technology for identifying iron deficiency. Blood 92(5):1807-1813.
- Crispen C. 1989. Carbon monoxide and flu-like symptoms. J Pediatr 114(2):342.
- Danesh J, Wheeler JG, Hirschfield GM, Eda S, Eiriksdottir G, Rumley A, et al. 2004. Creactive protein and other circulating markers of inflammation in the prediction of coronary heart disease. N Engl J Med 350(14):1387-1397.
- Dary O, Pineda O, Belizan JM. 1981. Carbon monoxide contamination in dwellings in poor rural areas of Guatemala. Bull Environ Contam Toxicol 26(1):24-30.
- Dasgupta S, Huq M, Khaliquzzaman M, Pandey K, Wheeler DR. 2004. Indoor Air Quality for Poor Families: New Evidence from Bangladesh. Policy Research Working Paper WPS3393 Development Research Group, World Bank.
- de Francisco A, Morris J, Hall AJ, Armstrong Schellenberg JR, Greenwood BM. 1993. Risk factors for mortality from acute lower respiratory tract infections in young Gambian children. Int J Epidemiol 22(6):1174-1182.
- Dennis RJ, Maldonado D, Norman S, Baena E, Martinez G. 1996. Woodsmoke exposure and risk for obstructive airways disease among women. Chest 109(1):115-119.
- Desai MA, Mehta S, Smith KR. 2004. Indoor smoke from solid fuels: Assessing the environmental burden of disease at national and local levels. Geneva: World Health Organization.
- Díaz E, Smith-Sivertsen T, Pope D, Lie RT, Díaz A, McCracken J, et al. 2007. Eye discomfort, headache and back pain among Mayan Guatemalan women taking part in a randomised stove intervention trial. J Epidemiol Community Health 61(1):74-79.

- Diez Roux AV, Auchineloss AH, Astor B, Barr RG, Cushman M, Dvonch T, et al. 2006. Recent exposure to particulate matter and C-reactive protein concentration in the multi-ethnic study of atherosclerosis. Am J Epidemiol 164(5):437-448.
- Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG, Jr. 1989. Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 139(3):587-594.
- Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, et al. 2006. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. Jama 295(10):1127-1134.
- Donaldson K. 2003. The biological effects of coarse and fine particulate matter. Occup Environ Med 60(5):313-314.
- Donaldson K, MacNee W. 2001. Potential mechanisms of adverse pulmonary and cardiovascular effects of particulate air pollution (PM10). Int J Hyg Environ Health 203(5-6):411-415.
- Donaldson K, Stone V, Seaton A, MacNee W. 2001. Ambient particle inhalation and the cardiovascular system: potential mechanisms. Environ Health Perspect 109 Suppl 4:523-527.
- Donaldson K, Tran CL. 2002. Inflammation caused by particles and fibers. Inhal Toxicol 14(1):5-27.
- Dossing M, Khan J, al-Rabiah F. 1994. Risk factors for chronic obstructive lung disease in Saudi Arabia. Respir Med 88(7):519-522.
- Dutt D, Srinivasa DK, Rotti SB, Sahai A, Konar D. 1996. Effect of indoor air pollution on the respiratory system of women using different fuels for cooking in an urban slum of Pondicherry. Natl Med J India 9(3):113-117.
- Dutta K, Naumoff Shields K, Edwards R, Smith KR. 2007. Impact of improved biomass cookstoves on indoor air quality near Pune, India. Energy for Sustainable Development XI(2):19-32.
- Edwards R, Hubbard A, Khalakdina A, Pennise D, Smith KR. 2007. Design considerations for field studies of changes in indoor air pollution due to improved stoves. Energy for Sustainable Development XI(2):71-81.
- Ekici A, Ekici M, Kurtipek E, Akin A, Arslan M, Kara T, et al. 2005. Obstructive airway diseases in women exposed to biomass smoke. Environ Res 99(1):93-98.
- Engel P, Hurtado E, Ruel M. 1997. Smoke exposure of women and young children in highland Guatemala: predictions and recall accuracy. Human Organization 56(4):408-417.

- Erhardt JG, Craft NE, Heinrich F, Biesalski HK. 2002. Rapid and simple measurement of retinol in human dried whole blood spots. J Nutr 132(2):318-321.
- Ezzati M, Kammen D. 2001a. Indoor air pollution from biomass combustion and acute respiratory infections in Kenya: an exposure-response study. Lancet 358(9282):619-624.
- Ezzati M, Kammen DM. 2001b. Quantifying the effects of exposure to indoor air pollution from biomass combustion on acute respiratory infections in developing countries. Environ Health Perspect 109(5):481-488.
- Ezzati M, Kammen DM. 2002. The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps, and data needs. Environ Health Perspect 110(11):1057-1068.
- Ezzati M, Saleh H, Kammen DM. 2000. The contributions of emissions and spatial microenvironments to exposure to indoor air pollution from biomass combustion in Kenya. Environ Health Perspect 108(9):833-839.
- Ferris BG. 1978. Epidemiology Standardization Project (American Thoracic Society). Am Rev Respir Dis 118(6 Pt 2):1-120.
- Fortmann SP, Ford E, Criqui MH, Folsom AR, Harris TB, Hong Y, et al. 2004. CDC/AHA Workshop on Markers of Inflammation and Cardiovascular Disease: Application to Clinical and Public Health Practice: report from the population science discussion group. Circulation 110(25):e554-559.
- Gao YT, Blot WJ, Zheng W, Ershow AG, Hsu CW, Levin LI, et al. 1987. Lung cancer among Chinese women. Int J Cancer 40(5):604-609.
- Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, et al. 2004. The effect of air pollution on lung development from 10 to 18 years of age. N Engl J Med 351(11):1057-1067.
- Gharaibeh NS. 1996. Effects of indoor air pollution on lung function of primary school children in Jordan. Ann Trop Paediatr 16(2):97-102.
- Glinianaia SV, Rankin J, Bell R, Pless-Mulloli T, Howel D. 2004. Particulate air pollution and fetal health: a systematic review of the epidemiologic evidence. Epidemiology 15(1):36-45.
- Greksa LP. 1996. Evidence for a genetic basis to the enhanced total lung capacity of Andean highlanders. Hum Biol 68(1):119-129.
- Guillen MD, Ibargoitia ML. 1999. Influence of the moisture content on the composition of the liquid smoke produced in the pyrolysis process of Fagus sylvatica L. wood. J Agric Food Chem 47(10):4126-4136.

- Guneser S, Atici A, Alparslan N, Cinaz P. 1994. Effects of indoor environmental factors on respiratory systems of children. J Trop Pediatr 40(2):114-116.
- Gupta D, Mathur N. 1997. A study of household environmental risk factors pertaining to respiratory diseases. Energy Environment Monitor 13:61-67.
- Hankinson JL, Odencrantz JR, Fedan KB. 1999. Spirometric reference values from a sample of the general U.S. population. Am J Respir Crit Care Med 159(1):179-187.
- Hannon WH, Boyle J, Davin B, Marsden A, McCabe ERB, Schwartz M, et al. 1997. Blood Collection of Filter Paper for Neonatal Screening Programs, 3rd edition National Committee for Clinical Laboratory Standards Document A4A3. Wayne, PA: National Committee for Clinical Laboratory Standards.
- Hass JD. 1992. Potential mechanisms for the effect of indoor cooking smoke on fetal growth. Invited paper presented at WHO workshop on 'The Impact of Indoor Cooking Smoke on Health' February 26-29.
- Hocking RR. 1976. A Biometrics Invited Paper. The Analysis and Selection of Variables in Linear Regression. Biometrics 32(1):1-49.
- Hole DJ, Watt GC, Davey-Smith G, Hart CL, Gillis CR, Hawthorne VM. 1996. Impaired lung function and mortality risk in men and women: findings from the Renfrew and Paisley prospective population study. Bmj 313(7059):711-715; discussion 715-716.
- Jeffery PK. 1998. Structural and inflammatory changes in COPD: a comparison with asthma. Thorax 53(2):129-136.
- Johnson AW, Aderele WI. 1992. The association of household pollutants and socioeconomic risk factors with the short-term outcome of acute lower respiratory infections in hospitalized pre-school Nigerian children. Ann Trop Paediatr 12(4):421-432.
- Jones JG, Minty BD, Lawler P, Hulands G, Crawley JC, Veall N. 1980. Increased alveolar epithelial permeability in cigarette smokers. Lancet 1(8159):66-68.
- Katsouyanni K. 2003. Ambient air pollution and health. Br Med Bull 68:143-156.
- Khalil MAK, Rasmussen RA. 2003. Tracers of wood smoke. Atmospheric Environment 37(9):1211-1222.
- Khushk WA, Fatmi Z, White F, Kadir MM. 2005. Health and social impacts of improved stoves on rural women: a pilot intervention in Sindh, Pakistan. Indoor Air 15(5):311-316.
- Kiraz K, Kart L, Demir R, Oymak S, Gulmez I, Unalacak M, et al. 2003. Chronic pulmonary disease in rural women exposed to biomass fumes. Clin Invest Med 26(5):243-248.

- Kleinbaum DG, Kupper LL, Muller KE. 1998. Applied regression analysis and other multivariate methods.
- Kony S, Zureik M, Driss F, Neukirch C, Leynaert B, Neukirch F. 2004. Association of bronchial hyperresponsiveness and lung function with C-reactive protein (CRP): a population based study. Thorax 59(10):892-896.
- Lamb D. 1995. Pathology. In: Chronic obstructive pulmonary disease (Calverley PMA, Pride NB, eds). London:Chapman & Hall, 9-34.
- Large AA, Owens GR, Hoffman LA. 1990. The short-term effects of smoke exposure on the pulmonary function of firefighters. Chest 97(4):806-809.
- Larson TV, Koenig JQ. 1994. Wood smoke: emissions and noncancer respiratory effects. Annu Rev Public Health 15:133-156.
- Leroyer C, Perfetti L, Trudeau C, L'Archeveque J, Chan-Yeung M, Malo JL. 1998. Comparison of serial monitoring of peak expiratory flow and FEV1 in the diagnosis of occupational asthma. Am J Respir Crit Care Med 158(3):827-832.
- Liao D, Heiss G, Chinchilli VM, Duan Y, Folsom AR, Lin HM, et al. 2005. Association of criteria pollutants with plasma hemostatic/inflammatory markers: a population-based study. J Expo Anal Environ Epidemiol 15(4):319-328.
- Liu Q, Sasco AJ, Riboli E, Hu MX. 1993. Indoor air pollution and lung cancer in Guangzhou, People's Republic of China. Am J Epidemiol 137(2):145-154.
- Loomis D, Castillejos M, Gold DR, McDonnell W, Borja-Aburto VH. 1999. Air pollution and infant mortality in Mexico City. Epidemiology 10(2):118-123.
- MacNee W, Donaldson K. 1999. Particulate air pollution: injurious and protective mechanisms in the lungs. In: Air Pollution and Health (Holgate ST, Samet JM, Koren HS, Maynard RL, eds). San Diego:Academic Press, 653-672.
- Macy EM, Hayes TE, Tracy RP. 1997. Variability in the measurement of C-reactive protein in healthy subjects: implications for reference intervals and epidemiological applications. Clin Chem 43(1):52-58.
- Maisonet M, Correa A, Misra D, Jaakkola JJ. 2004. A review of the literature on the effects of ambient air pollution on fetal growth. Environ Res 95(1):106-115.
- Malik SK. 1985. Exposure to domestic cooking fuels and chronic bronchitis. Indian J Chest Dis Allied Sci 27(3):171-174.
- Masera O, Edwards R, Armendariz Arnez C, Berrueta V, Johnson M, Rojas Bracho L, et al. 2007. Impact of Patsari improved cookstoves on indoor air quality in Michoacan, Mexico. Energy for Sustainable Development XI(2):45-56.

- Mavalankar DV, Trivedi CR, Gray RH. 1991. Levels and risk factors for perinatal mortality in Ahmedabad, India. Bull World Health Organ 69(4):435-442.
- Maynard RL, Waller R. 1999. Carbon monoxide. In: Air Pollution and Health (Holgate ST, Samet JM, Koren HS, Maynard RL, eds). San Diego:Academic Press, 749-796.
- McCracken J, Ryan L, Díaz A, Mittleman M, Schwartz J, Smith KR. 2006. Estimating long-term woodsmoke exposure when shortterm measurements vary widely: RESPIRE Guatemala. International Society for Environmental Epidemiology: Paris, France (conference poster and presentation available on-line: http://ehs.sph.berkeley.edu/guat/page.asp?id=07).
- McCracken JP, Smith KR, Diaz A, Mittleman MA, Schwartz J. 2007. Chimney stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women. Environ Health Perspect 115(7):996-1001.
- McDade TW. 2006. Measuring immunological markers. In: Measuring Stress in Humans: a practical guide for the field (Ice GH, James GD, eds). Cambridge:Cambridge University Press.
- McDade TW. 2007. Measuring immunological markers. In: Measuring Stress in Humans: a practical guide for the field (Ice GH, James GD, eds). Cambridge:Cambridge University Press.
- McDade TW, Burhop J, Dohnal J. 2004. High-sensitivity enzyme immunoassay for C-reactive protein in dried blood spots. Clin Chem 50(3):652-654.
- McDade TW, Shell-Duncan B. 2002. Whole blood collected on filter paper provides a minimally invasive method for assessing human transferrin receptor level. J Nutr 132(12):3760-3763.
- McDade TW, Stallings JF, Angold A, Costello EJ, Burleson M, Cacioppo JT, et al. 2000. Epstein-Barr virus antibodies in whole blood spots: a minimally invasive method for assessing an aspect of cell-mediated immunity. Psychosom Med 62(4):560-567.
- McGrath JJ. 2000. Biological plausibility for carbon monoxide as a copollutant in PM epidemiologic studies. Inhal Toxicol 12 Suppl 4:91-107.
- Medical Research Council. 1965. Definition and classification of chronic bronchitis for clinical and epidemiological purposes. Lancet 1:775-779.
- Mei JV, Alexander JR, Adam BW, Hannon WH. 2001. Use of filter paper for the collection and analysis of human whole blood specimens. J Nutr 131(5):1631S-1636S.
- Menezes AM, Victora CG, Rigatto M. 1994. Prevalence and risk factors for chronic bronchitis in Pelotas, RS, Brazil: a population-based study. Thorax 49(12):1217-1221.

- Metzger KB, Tolbert PE, Klein M, Peel JL, Flanders WD, Todd K, et al. 2004. Ambient air pollution and cardiovascular emergency department visits. Epidemiology 15(1):46-56.
- Mishra V, Dai X, Smith KR, Mika L. 2004. Maternal exposure to biomass smoke and reduced birth weight in Zimbabwe. Ann Epidemiol 14(10):740-747.
- Mishra V, Retherford RD, Smith KR. 2005. Cooking smoke and tobacco smoke as risk factors for stillbirth. Int J Environ Health Res 15(6):397-410.
- Morris K, Morgenlander M, Coulehan JL, Gahagen S, Arena VC. 1990. Wood-burning stoves and lower respiratory tract infection in American Indian children. Am J Dis Child 144(1):105-108.
- Musk AW, de Klerk NH, Beach JR, Fritschi L, Sim MR, Benke G, et al. 2000. Respiratory symptoms and lung function in alumina refinery employees. Occup Environ Med 57(4):279-283.
- Musk AW, Smith TJ, Peters JM, McLaughlin E. 1979. Pulmonary function in firefighters: acute changes in ventilatory capacity and their correlates. Br J Ind Med 36(1):29-34.
- Naeher LP, Brauer M, Lipsett M, Zelikoff JT, Simpson CD, Koenig JQ, et al. 2007. Woodsmoke health effects: a review. Inhal Toxicol 19(1):67-106.
- Naeher LP, Holford TR, Beckett WS, Belanger K, Triche EW, Bracken MB, et al. 1999. Healthy women's PEF variations with ambient summer concentrations of PM10, PM2.5, SO42-, H+, and O3. Am J Respir Crit Care Med 160(1):117-125.
- Naeher LP, Leaderer BP, Smith KR. 2000a. Particulate matter and carbon monoxide in highland Guatemala: indoor and outdoor levels from traditional and improved wood stoves and gas stoves. Indoor Air 10(3):200-205.
- Naeher LP, Smith KR, Brauer M, Chowdhury Z, Simpson C, Koenig JQ, et al. 2005. Critical Review of the Health Effects of Woodsmoke. Ottawa: Air Health Division, Health Canada.
- Naeher LP, Smith KR, Leaderer BP, Mage D, Grajeda R. 2000b. Indoor and outdoor PM2.5 and CO in high- and low-density Guatemalan villages. J Expo Anal Environ Epidemiol 10(6 Pt 1):544-551.
- Naeher LP, Smith KR, Leaderer BP, Neufeld L, Mage DT. 2001. Carbon monoxide as a tracer for assessing exposures to particulate matter in wood and gas cookstove households of highland Guatemala. Environ Sci Technol 35(3):575-581.
- Norboo T, Yahya M, Bruce NG, Heady JA, Ball KP. 1991. Domestic pollution and respiratory illness in a Himalayan village. Int J Epidemiol 20(3):749-757.

- Nyberg F, Pershagen G. 2000. Epidemiologic studies on the health effects of ambient particulate air pollution. Scand J Work Environ Health 26 Suppl 1:49-89.
- O'Dempsey TJ, McArdle TF, Morris J, Lloyd-Evans N, Baldeh I, Laurence BE, et al. 1996. A study of risk factors for pneumococcal disease among children in a rural area of west Africa. Int J Epidemiol 25(4):885-893.
- O'Neill MS, Veves A, Sarnat JA, Zanobetti A, Gold DR, Economides PA, et al. 2007. Air pollution and inflammation in type 2 diabetes: a mechanism for susceptibility. Occup Environ Med 64(6):373-379.
- Olshan AF. 2007. Workgroup Report: The Use of Newborn Blood Spots in Environmental Research: Opportunities and Challenges Environ Health Perspect doi:10.1289/ehp.10511 (in press).
- Pandey MR. 1984. Domestic smoke pollution and chronic bronchitis in a rural community of the Hill Region of Nepal. Thorax 39(5):337-339.
- Pandey MR, Boleij JS, Smith KR, Wafula EM. 1989. Indoor air pollution in developing countries and acute respiratory infection in children. Lancet 1(8635):427-429.
- Pandey MR, Regmi HN, Neupane RP, Gautam A, Bhandari DP. 1985. Domestic smoke pollution and respiratory function in rural Nepal. Tokai J Exp Clin Med 10(4):471-481.
- Parker SP, Cubitt WD. 1999. The use of the dried blood spot sample in epidemiological studies. J Clin Pathol 52(9):633-639.
- Pauwels RA, Buist AS, Calverley PMA, Jenkins CR, Hurd SS. 2001. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop Summary:Am Thoracic Soc, 1256-1276.
- Peel JL, Metzger KB, Klein M, Flanders WD, Mulholland JA, Tolbert PE. 2007. Ambient air pollution and cardiovascular emergency department visits in potentially sensitive groups. Am J Epidemiol 165(6):625-633.
- Peel JL, Tolbert PE, Klein M, Metzger KB, Flanders WD, Todd K, et al. 2005. Ambient Air Pollution and Respiratory Emergency Department Visits. Epidemiology 16(2):164-174.
- Pekkanen J, Brunner EJ, Anderson HR, Tiittanen P, Atkinson RW. 2000. Daily concentrations of air pollution and plasma fibrinogen in London. Occup Environ Med 57(12):818-822.
- Pereira LA, Loomis D, Conceicao GM, Braga AL, Arcas RM, Kishi HS, et al. 1998. Association between air pollution and intrauterine mortality in Sao Paulo, Brazil. Environ Health Perspect 106(6):325-329.

- Perera FP, Jedrychowski W, Rauh V, Whyatt RM. 1999. Molecular epidemiologic research on the effects of environmental pollutants on the fetus. Environ Health Perspect 107 Suppl 3:451-460.
- Pérez-Padilla JR, Regalado-Pineda J, Vázquez-García JC. 2001. Reproducibilidad de espirometrías en trabajadores mexicanos y valores de referencia internacionales. Salud Publica Mex 43:113-121.
- Perez-Padilla R, Regalado J, Vedal S, Pare P, Chapela R, Sansores R, et al. 1996. Exposure to biomass smoke and chronic airway disease in Mexican women. A casecontrol study. Am J Respir Crit Care Med 154(3 Pt 1):701-706.
- Peters A, Doring A, Wichmann HE, Koenig W. 1997. Increased plasma viscosity during an air pollution episode: a link to mortality? Lancet 349(9065):1582-1587.
- Peters A, Frohlich M, Doring A, Immervoll T, Wichmann HE, Hutchinson WL, et al. 2001a. Particulate air pollution is associated with an acute phase response in men. Results from the MONICA-Augsburg Study. Eur Heart J 22(14):1198-1204.
- Peters A, Frohlich M, Doring A, Immervoll T, Wichmann HE, Hutchinson WL, et al. 2001b. Particulate air pollution is associated with an acute phase response in men; results from the MONICA-Augsburg Study. Eur Heart J 22(14):1198-1204.
- Peters A, Liu E, Verrier RL, Schwartz J, Gold DR, Mittleman M, et al. 2000a. Air pollution and incidence of cardiac arrhythmia. Epidemiology 11(1):11-17.
- Peters A, Perz S, Doring A, Stieber J, Koenig W, Wichmann HE. 2000b. Activation of the autonomic nervous system and blood coagulation in association with an air pollution episode. Inhal Tox 12(Supplement 2):51-61.
- Pope CA, 3rd. 2000. Epidemiology of fine particulate air pollution and human health: biologic mechanisms and who's at risk? Environ Health Perspect 108 Suppl 4:713-723.
- Pope CA, 3rd, Bates DV, Raizenne ME. 1995a. Health effects of particulate air pollution: time for reassessment? Environ Health Perspect 103(5):472-480.
- Pope CA, 3rd, Hansen ML, Long RW, Nielsen KR, Eatough NL, Wilson WE, et al. 2004. Ambient particulate air pollution, heart rate variability, and blood markers of inflammation in a panel of elderly subjects. Environ Health Perspect 112(3):339-345.
- Pope CA, 3rd, Kanner RE. 1993. Acute effects of PM10 pollution on pulmonary function of smokers with mild to moderate chronic obstructive pulmonary disease. The American Review of Respiratory Disease 147(6 Pt 1):1336-1340.

- Pope CA, 3rd, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, et al. 1995b. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. American Journal of Respiratory and Critical Care Medicine 151(3 Pt 1):669-674.
- Quanjer PH, Lebowitz MD, Gregg I, Miller MR, Pedersen OF. 1997. Peak expiratory flow: conclusions and recommendations of a Working Party of the European Respiratory Society. Eur Respir J Suppl 24:2S-8S.
- Qureshi KA. 1994. Domestic smoke pollution and prevalence of chronic bronchitis/asthma in a rural area of Kashmir. Indian J Chest Dis Allied Sci 36(2):61-72.
- Rahman I, Morrison D, Donaldson K, MacNee W. 1996. Systemic oxidative stress in asthma, COPD, and smokers. Am J Respir Crit Care Med 154(4 Pt 1):1055-1060.
- Raizenne M, Neas LM, Damokosh AI, Dockery DW, Spengler JD, Koutrakis P, et al. 1996. Health effects of acid aerosols on North American children: pulmonary function. Environ Health Perspect 104(5):506-514.
- Ramirez-Venegas A, Sansores RH, Perez-Padilla R, Regalado J, Velazquez A, Sanchez C, et al. 2006. Survival of patients with chronic obstructive pulmonary disease due to biomass smoke and tobacco. Am J Respir Crit Care Med 173(4):393-397.
- Ray MR, Mukherjee S, Roychoudhury S, Bhattacharya P, Banerjee M, Siddique S, et al. 2006. Platelet activation, upregulation of CD11b/CD18 expression on leukocytes and increase in circulating leukocyte-platelet aggregates in Indian women chronically exposed to biomass smoke. Human & Experimental Toxicology 25(11):627.
- Reddy TS, Guleria R, Sinha S, Sharma SK, Pande JN. 2004. Domestic cooking fuel and lung functions in healthy non-smoking women. Indian J Chest Dis Allied Sci 46(2):85-90.
- Regalado J, Perez-Padilla R, Samsores R, Vedal S, Brauer M, Pare P. 1996. The effect of biomass burning on respiratory symptoms and lung function in rural Mexican women. Am J Respir Crit Care Med 153:A701.
- Regalado J, Perez-Padilla R, Sansores R, Paramo Ramirez JI, Brauer M, Pare P, et al. 2006. The effect of biomass burning on respiratory symptoms and lung function in rural Mexican women. Am J Respir Crit Care Med 174(8):901-905.
- Rehfuess E, Mehta S, Pruss-Ustun A. 2006. Assessing Household Solid Fuel Use: Multiple Implications for the Millennium Development Goals. Environ Health Perspect 114(3):373-378.

- Ridker PM, Buring JE, Shih J, Matias M, Hennekens CH. 1998. Prospective study of Creactive protein and the risk of future cardiovascular events among apparently healthy women. Circulation 98(8):731-733.
- Ridker PM, Hennekens CH, Buring JE, Rifai N. 2000. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. N Engl J Med 342(12):836-843.
- Ridker PM, Rifai N, Pfeffer MA, Sacks F, Braunwald E. 1999. Long-term effects of pravastatin on plasma concentration of C-reactive protein. The Cholesterol and Recurrent Events (CARE) Investigators. Circulation 100(3):230-235.
- Rifai N, Tracy RP, Ridker PM. 1999. Clinical efficacy of an automated high-sensitivity C-reactive protein assay. Clin Chem 45(12):2136-2141.
- Rinne ST, Rodas EJ, Bender BS, Rinne ML, Simpson JM, Galer-Unti R, et al. 2006. Relationship of pulmonary function among women and children to indoor air pollution from biomass use in rural Ecuador. Respir Med 100(7):1208-1215.
- Riojas-Rodriguez H, Romano-Riquer P, Santos-Burgoa C, Smith KR. 2001. Household firewood use and the health of children and women of Indian communities in Chiapas, Mexico. Int J Occup Environ Health 7(1):44-53.
- Ritz B, Yu F. 1999. The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993. Environ Health Perspect 107(1):17-25.
- Roberts WL, Sedrick R, Moulton L, Spencer A, Rifai N. 2000. Evaluation of four automated high-sensitivity C-reactive protein methods: implications for clinical and epidemiological applications. Clin Chem 46(4):461-468.
- Robin LF, Less PS, Winget M, Steinhoff M, Moulton LH, Santosham M, et al. 1996. Wood-burning stoves and lower respiratory illnesses in Navajo children. Pediatr Infect Dis J 15(10):859-865.
- Robinson LD, Jewell NP. 1991. Some surprising results about covariate adjustment in logistic regression models. International Statistical Review 58:227-240.
- Robinson MM, Hickey MS, Clark ML, Nelson TL. 2007. Analysis of C-reactive protein in dried blood spots using a commercially available enzyme linked immunosorbent assay. *In progress*.
- Rodkey FL, O'Neal JD, Collison HA, Uddin DE. 1974. Relative affinity of hemoglobin S and hemoglobin A for carbon monoxide and oxygen. Clin Chem 20(1):83-84.
- Routledge HC, Ayres JG. 2005. Air pollution and the heart. Occup Med (Oxf) 55(6):439-447.

- Ruckerl R, Ibald-Mulli A, Koenig W, Schneider A, Woelke G, Cyrys J, et al. 2006. Air pollution and markers of inflammation and coagulation in patients with coronary heart disease. Am J Respir Crit Care Med 173(4):432-441.
- Saha A, Rao NM, Kulkarni PK, Majumdar PK, Saiyed HN. 2005. Pulmonary function and fuel use: a population survey. Respir Res 6:127.
- Saksena S, Prasad R, Pal RC, Joshi V. 1992. Patterns of daily exposure to TSP and CO in the Garhwal Himalaya. Atmospheric Environment 26:2125-2134.
- Saksena S, Smith KR. 2003. Indoor Air Pollution. In: Air Pollution and Health in Rapidly Developing Countries (McGranahan G, Murray F, eds):Earthscan Publications Ltd, 129-145.
- Salvi S, Blomberg A, Rudell B, Kelly F, Sandstrom T, Holgate ST, et al. 1999. Acute inflammatory responses in the airways and peripheral blood after short-term exposure to diesel exhaust in healthy human volunteers. Am J Respir Crit Care Med 159(3):702-709.
- Samet JM, Dominici F, Curriero FC, Coursac I, Zeger SL. 2000a. Fine particulate air pollution and mortality in 20 U.S. cities, 1987-1994. N Engl J Med 343(24):1742-1749.
- Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW, et al. 2000b. The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and mortality from air pollution in the United States. Res Rep Health Eff Inst 94(Pt 2):5-70; discussion 71-79.
- Schei MA, Hessen JO, Smith KR, Bruce N, McCracken J, Lopez V. 2004. Childhood asthma and indoor woodsmoke from cooking in Guatemala. J Expo Anal Environ Epidemiol 14 Suppl 1:S110-117.
- Schunemann HJ, Dorn J, Grant BJ, Winkelstein W, Jr., Trevisan M. 2000. Pulmonary function is a long-term predictor of mortality in the general population: 29-year follow-up of the Buffalo Health Study. Chest 118(3):656-664.
- Schwartz J. 1989. Lung function and chronic exposure to air pollution: a cross-sectional analysis of NHANES II. Environ Res 50(2):309-321.
- Schwartz J. 1999. Air pollution and hospital admissions for heart disease in eight U.S. counties. Epidemiology 10(1):17-22.
- Schwartz J. 2001. Air pollution and blood markers of cardiovascular risk. Environ Health Perspect 109 Suppl 3:405-409.
- Schwela D. 2000. Air pollution and health in urban areas. Rev Environ Health 15(1-2):13-42.

- Seaton A, MacNee W, Donaldson K, Godden D. 1995. Particulate air pollution and acute health effects. Lancet 345(8943):176-178.
- Seaton A, Soutar A, Crawford V, Elton R, McNerlan S, Cherrie J, et al. 1999. Particulate air pollution and the blood. Thorax 54(11):1027-1032.
- Sherman CB, Barnhart S, Miller MF, Segal MR, Aitken M, Schoene R, et al. 1989. Firefighting acutely increases airway responsiveness. Am Rev Respir Dis 140(1):185-190.
- Shrestha IL, Shrestha SL. 2005. Indoor air pollution from biomass fuels and respiratory health of the exposed population in Nepalese households. Int J Occup Environ Health 11(2):150-160.
- Siafakas NM, Vermeire P, Pride NB, Paoletti P, Gibson J, Howard P, et al. 1995. Optimal assessment and management of chronic obstructive pulmonary disease (COPD). The European Respiratory Society Task Force. Eur Respir J 8(8):1398-1420.
- Sin DD, Man SFP. 2003. Why are patients with chronic obstructive pulmonary disease at increased risk of cardiovascular diseases? The potential role of systemic inflammation in chronic obstructive pulmonary disease. Circulation 107(11):1514-1519.
- Skogstrand K, Thorsen P, Norgaard-Pedersen B, Schendel DE, Sorensen LC, Hougaard DM. 2005. Simultaneous measurement of 25 inflammatory markers and neurotrophins in neonatal dried blood spots by immunoassay with xMAP technology. Clin Chem 51(10):1854-1866.
- Smith-Sivertsen T, Diaz E, Bruce N, Diaz A, Khalakdina A, Schei MA, et al. 2004. Reducing indoor air pollution with a randomised intervention design - A presentation of the Stove Intervention Study in the Guatemalan Highlands. Norsk Epidemiologi 14(2):137-143.
- Smith-Sivertsen T, Díaz E, Pope D, Arana B, McCracken J, Jenny A, et al. 2006. RESPIRE-the Guatemala Randomised Intervention Trial: Impact of an improved stove on women's lung health in a rural wood-using community. International Society for Environmental Epidemiology: Paris, France (conference presentation available on-line: http://ehs.sph.berkeley.edu/guat/page.asp?id=07).
- Smith KR. 1987. Biofuels, Air Pollution, and Health: A Global Review. New York:Plenum.
- Smith KR. 1988. Air pollution: assessing total exposure in developing countries. Environment 30:16-34.
- Smith KR. 1993. Fuel combustion, air pollution exposure, and health: the situation in developing countries. Annual Review of Energy and the Environment 18:529-566.

- Smith KR. 2002. Indoor air pollution in developing countries: recommendations for research. Indoor Air 12(3):198-207.
- Smith KR, Bruce N, Arana B, Diaz A, Jenny A, Khalakdina A, et al. 2006. RESPIRE the Guatemala Randomized Intervention Trial. International Society for Environmental Epidemiology: Paris, France (conference presentation available online: http://ehs.sph.berkeley.edu/guat/page.asp?id=07)
- Smith KR, Mehta S, Maeusezahl-Feuz M. 2004. Indoor air pollution from household use of solid fuels. In: Comparative quantification of health risks : global and regional burden of disease attributable to selected major risk factors (Ezzati M, Rodgers AD, Lopez AD, Murray CJL, eds). Geneva:World Health Organization, 1435-1493.
- Smith KR, Samet JM, Romieu I, Bruce N. 2000. Indoor air pollution in developing countries and acute lower respiratory infections in children. Thorax 55(6):518-532.
- Sobue T. 1990. Association of indoor air pollution and lifestyle with lung cancer in Osaka, Japan. Int J Epidemiol 19 Suppl 1:S62-66.
- Sram RJ, Binkova BB, Dejmek J, Bobak M. 2005. Ambient air pollution and pregnancy outcomes: A review of the literature. Environ Health Perspect 113(4):375-382.
- Suh HH, Bahadori T, Vallarino J, Spengler JD. 2000. Criteria air pollutants and toxic air pollutants. Environ Health Perspect 108 Suppl 4:625-633.
- Tashkin DP, Detels R, Simmons M, Liu H, Coulson AH, Sayre J, et al. 1994. The UCLA population studies of chronic obstructive respiratory disease: XI. Impact of air pollution and smoking on annual change in forced expiratory volume in one second. Am J Respir Crit Care Med 149(5):1209-1217.
- Teeter JG, Bleecker ER. 1998. Relationship between airway obstruction and respiratory symptoms in adult asthmatics. Chest 113(2):272-277.
- Thomason MJ, Strachan DP. 2000. Which spirometric indices best predict subsequent death from chronic obstructive pulmonary disease? Thorax 55(9):785-788.
- Thompson L, Diaz J, Jenny A, Diaz A, Bruce N, Balmes J. 2007. Nxwisen, ntzarrin or ntzo'lin? Mapping children's respiratory symptoms among indigenous populations in Guatemala. Soc Sci Med 65(7):1337-1350.
- Touloumi G, Samoli E, Katsouyanni K. 1996. Daily mortality and "winter type" air pollution in Athens, Greece--a time series analysis within the APHEA project. J Epidemiol Community Health 50 Suppl 1:s47-51.
- USEPA. 1992. Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders. Washington DC:Office of Research and Development.

- Vasan RS. 2006. Biomarkers of cardiovascular disease: molecular basis and practical considerations. Circulation 113(19):2335-2362.
- Wang B, Peng Z, Zhang X, Xu Y, Wang H, Allen G, et al. 1999. Particulate matter, sulfur dioxide, and pulmonary function in never- smoking adults in Chongqing, China. Int J Occup Environ Health 5(1):14-19.
- WHO. 1998. World health report. Geneva: WHO.
- WHO. 2002. Air quality guidelines for Europe; second edition. Copenhagen: World Health Organization, Regional Office for Europe.
- WHO. 2005. WHO Air Quality Guidelines: Global Update for 2005. Copenhagen: World Health Organization, Regional Office for Europe.
- WHO. 2007. Indoor air pollution and lower respiratory tract infections in children. Geneva, Switzerland: World Health Organization.
- Wilkins J, Gallimore JR, Moore EG, Pepys MB. 1998. Rapid automated high sensitivity enzyme immunoassay of C-reactive protein. Clin Chem 44(6 Pt 1):1358-1361.
- Wilson R, Spengler JD. 1996. Particles in our air: concentrations and health effects. Cambridge, MA:Harvard University Press.
- Woodruff TJ, Grillo J, Schoendorf KC. 1997. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. Environ Health Perspect 105(6):608-612.
- Worthman CM, Stallings JF. 1994. Measurement of gonadotropins in dried blood spots. Clin Chem 40(3):448-453.
- Worthman CM, Stallings JF. 1997. Hormone measures in finger-prick blood spot samples: new field methods for reproductive endocrinology. Am J Phys Anthropol 104(1):1-21.
- Zeka A, Sullivan JR, Vokonas PS, Sparrow D, Schwartz J. 2006. Inflammatory markers and particulate air pollution: characterizing the pathway to disease. Int J Epidemiol 35(5):1347-1354.
- Zhang J, Smith KR. 1999. Emissions of Carbonyl Compounds from Various Cookstoves in China. Environ Sci Technol 33(14):2311-2320.
- Zhang J, Smith KR. 2003. Indoor air pollution: a global health concern. Br Med Bull 68:209-225.

APPENDIX A

QUESTIONNAIRE



Department of Environmental and Radiological Health Sciences

College of Veterinary Medicine and Biomedical Sciences 1681 Campus Delivery Fort Collins, Colorado 80523-1681 (970) 491-7038 FAX: (970) 491-2940

Indoor Air Pollution & Respiratory Health among Honduran Women: Evaluating Intervention Effectiveness

PERSONAL DATA SHEET

Date:	 _	
Participant Name:		
Home Address:	 	
City:	 	
Home Phone: (if applicable)		

QUESTIONNAIRE

Indoor Air Pollution & Respiratory Health among Honduran Women: Evaluating Intervention Effectiveness

CSU Identification Number:

INSTRUCTIONS FOR THE INVESTIGATOR: Administer the questionnaire to each participant and mark each question by circling the answer or by filling in the box. Inform the participant that they do not have to answer a question. If the participant chooses not to answer a question, write "R" for "refuse" after the question.

Body Measurements:

Prior to beginning, ask the participant if she is willing to be weighed and measured for height and waist circumference. If no, write "R" for "refuse" in the space provided. If yes, record the measurements here:

Weight (lbs): _____

Height (in):

Waist Circumference(in):

1.0 GENERAL INFORMATION

1.1 Age: _____

1.2 Do you consider yourself to be of Hispanic or "Ladino" origin?

1. Yes 2. No

1.3 Which group best describes your ethnicity?

- 1. Mestizo
- 2. European
- 3. Arab
- 4. African
- 5. Asian
- 6. Indigenous Indian
- 7. Other: _____
- 9. Don't know

1.4 How long have you attended school? _____ years (enter 0 if none)

1.5 How long have you lived in your current home? ______ years

2.0 WORK

- 2.1 What is your occupation?
- 2.2 How long have you worked at the above job? _____ years (enter 0 if none)

2.3 Do you use your cookstove to prepare food/drink for selling?

3.0 SMOKING

- **3.1** Do you currently smoke cigarettes? (**No** means less than 20 packs, or 400 cigarettes or, less than 1 cigarette a day for a year)
 - 1. Yes 2. No
- **3.2** If no, have you ever smoked cigarettes in the past? (**No** means less than 20 packs, or 400 cigarettes or, less than 1 cigarette a day for a year)

1. Yes 2. No

3.3 If yes, how many years did you smoke?

_____ years 99. never smoked

3.4 If yes, on the average, for the entire time you smoked, how many cigarettes did you smoke per day?

_____ cigs/day 99. never smoked

Yes. Meals prepared per week: ______
No

- **3.5** Do other people smoke in the kitchen?
 - 1. Yes, one or more times per day
 - 2. Occasionally, less than one time per day
 - 3. No
- 3.6 Do other people smoke in your home in places other than the kitchen?
 - 1. Yes, one or more times per day
 - 2. Occasionally, less than one time per day
 - 3. No

4.0 HEALTH

4.1 Please indicate the box that best describes your symptoms AT THIS TIME:

	Symptom	None	Mild	Moderate	Severe	Very Severe
4.1.1	Eye irritation					
4.1.2	Blurred vision					
4.1.3	Nose irritation					
4.1.4	Amount of mucous or phlegm					
4.1.5	Tingling fingers					
4.1.6	Shortness of breath					
4.1.7	Headache					
4.1.8	Chest wheezing or whistling					
4.1.9	Throat irritation					
4.1.10	Cough					

4.2 COUGH

- **4.2.1**. Do you usually have a cough? (Count a cough on first going outside. Exclude clearing of throat.)
 - 1. Yes 2. No
- **4.2.2**. Do you usually cough as much as 4 6 times a day, 4 or more days out of the week?
 - 1. Yes 2. No 99. Does not apply
- **4.2.3** Do you usually cough like this on most days for 3 consecutive months or more during the year?
 - 1. Yes 2. No 99. Does not apply

4.2.4. For how many years have you had this cough?

_____ years 99. Does not apply

4.2.5. Is your cough caused or made worse by exposures to cookstove smoke?

1. Yes 2. No

4.2.6 Is your cough caused or made worse by any other exposure?

1. Yes. Please list: ______2. No

4.3 PHLEGM

4.3.1. Do you usually bring up phlegm from your chest? (Count phlegm on first going outside. Exclude phlegm from the nose, count swallowed phlegm.)

1. Yes 2. No

258

- **4.3.2.** Do you usually bring up phlegm like this as much as twice a day, 4 or more days out of the week?
 - 1. Yes 2. No 99. Does not apply
- **4.3.3.** Do you bring up phlegm like this on most days for 3 consecutive months or more during the year?

1. Yes 2. No 99. Does not apply

4.3.4. For how many years have you had trouble with phlegm?

_____ years 99. Does not apply

4.3.5. Is this problem caused or made worse by exposures to cookstove smoke?

1. Yes 2. No **4.3.6** Is this problem caused or made worse by any other exposure?

> 1. Yes. Please list: ______ 2. No

4.4. WHEEZING

4.4.1. Does your chest usually sound wheezy or whistling?

1. Yes 2. No

4.4.2. For how many years has this been present?

_____years 99. Does not apply

4.4.3. Is your chest wheezing caused or made worse by exposures to cookstove smoke?

1. Yes 2. No

- **4.4.4** Is your chest wheezing caused or made worse by any other exposure?
 - 1. Yes. Please list: _____
 - 2. No

4.5. SHORTNESS OF BREATH

- **4.5.1.** Are you troubled by shortness of breath when: (circle all that apply)
 - 1. Hurrying on the level or walking up a slight hill
 - 2. Walking at your own pace on the level
 - 99. Does not apply
- **4.5.2.** Is your shortness of breath caused or made worse by exposures to cookstove smoke?
 - 1. Yes 2. No
- **4.5.3** Is your shortness of breath caused or made worse by any other exposure?
 - 1. Yes. Please list: ______

4.6. NASAL IRRITATION

- **4.6.1.** Do you currently have nasal stuffiness, runny nose, sneezing and/or nasal itchiness?
 - 1. Yes 2. No
- **4.6.2.** Do these symptoms ever occur: (circle all that apply)
 - 1. Occasionally apart from colds
 - 2. Most days or nights
 - 99. Does not apply

4.6.3. Is your nasal stuffiness, runny nose, sneezing and/or nasal itchiness caused or made worse by exposures to cookstove smoke?

1. Yes 2. No

- **4.6.4** Is your shortness of breath caused or made worse by any other exposure?
 - 1. Yes. Please list: ______ 2. No

4.7 HEADACHE

4.7.1. Do you usually develop a headache during cooking?

1. Yes 2. No

- **4.7.2.** Does the headache get better, worse, or stay the same after cooking?
 - 1. Better
 - 2. Worse
 - 3. Same
 - 99. Does not apply

4.8 Has your doctor ever told you that you have any of the following conditions?

	Condition	Yes	No	Don't know
4.8.1.	Allergies (Hay Fever)			
4.8.2.	Asthma			
4.8.3.	Chronic bronchitis			
4.8.4.	Emphysema			
4.8.5.	5. Lung Cancer			
4.8.6.	Cardiovascular Disease			
4.8.7.	Diabetes			
4.8.8.	Periodontal Disease			
4.8.9.	Rheumatoid arthritis			

4.9 Do any relatives on your side of the family (blood relatives: mother, father, sister, brother, son, daughter) have any of the following?

	Condition	Yes	No	Don't know
4.9.1.	Allergies (Hay Fever)			
4.9.2.	Asthma			
4.9.3.	Chronic bronchitis			
4.9.4.	Emphysema			
4.9.5.	Lung Cancer			

4.10 <u>During the past week</u>, have you had any of the following conditions?

	Condition	Yes	No	Don't know
4.10.1.	Cold or Flu			
4.10.2.	Sinus problem (not a cold)			
4.10.3.	Pneumonia			

4.11 Are you taking any medications including oral contraceptives or noninflammatory medications on a regular basis <u>or</u> as prescribed by your physician?

1. Yes 2. No

If yes, list all medications (if available, ask to see the medicine container to obtain detailed information):

<u> </u>	

262

4.12 Are you taking any vitamins or supplements on a daily or regular basis?

1. Yes 2. No

If yes, list all vitamins (if available, ask to see the vitamin container to obtain detailed information):

 <u></u>

4.13 How many times per week do you eat fish? _____ times/week

4.14 Have you had your menstrual period at any time during the last 6 months?

1. Yes 2. No

4.15 Are you currently experiencing more than the usual amount of stress?

1. Yes 2. No

4.16 Are you concerned that breathing smoke from the fire in your home may cause health problems?

1. Yes 2. No

4.17 In what ways do you feel that smoke from the fire affects:

4.17.1 your health? _____

	4.17.2	the health of your children?
		· · · · · · · · · · · · · · · · · · ·
0	COOKING	PRACTICES
1	What type	of "cookstove" do you use for cooking?
	1 2	Open fire, traditional stove <i>Justa</i> stove
	3	
2	Which type	e of fuel do you use for cooking?
	1 2 3	Wood Gas Other (please specify):
3	How long l	have you used your current stove? years
4	How many meals/wee	r meals per week do you cook, on average? ek
5	How many hours	hours do you typically spend cooking each day?
6	For how m hours	nany hours during a typical day is the fire burning?
7	How much on average	n time do you spend in the room with the fire burning each da e? hours

Can you think of any ways in which your day is different from how it would 5.8 have been if monitoring had not been taking place? Other comments & observations from interviewee: ----Other comments & observations from interviewer:
APPENDIX B

INVESTIGATOR SURVEY SHEET

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.

INTERVIEWER SURVEY

(Adapted ITDG protocol)

Participant ID: _____

Collection Date: _____

AIR SAMPLING:

Interviewer initials: _____

Particulate Matter Sampling (PEM):

PEM personal ID:	
Filter ID:	Pump ID:
Start Time:	End time:
Pre-calibration:	Post-calibration:
Pre-weight:	Post-weight:
PEM Indoor ID:	
Filter ID:	Pump ID:
Start Time:	End time:
Pre-calibration:	Post-calibration:
Pre-weight:	Post-weight:
PEM Outdoor ID:	
Filter ID:	Pump ID:
Start Time:	End time:
Pre-calibration:	Post-calibration:
Pre-weight:	Post-weight:
PEM Blank ID:	
Filter ID:	Pump ID:
Start Time:	End time:
Pre-calibration:	Post-calibration:
Pre-weight:	Post-weight:

Q-Trak Sampling (data logged continuously):

Q-Trak ID:			
Calibration:			
	Location:	Start time:	End time:
Indoor:			
Outdoor 1:			
Outdoor 2:			

ADDITIONAL NOTES:

LUNG FUNCTION:

	(LPM)		(LITERS)	NOTES:
1. PEF		1. FEV1		
2. PEF		2. FEV1		
3. PEF		3. FEV1		

KITCHEN/HOUSE:

1. Kitchen type		
Is the kitchen: 1. Enclosed of	or 2. Semi-open ?	
 Is the kitchen a: Separate building? Separate room attached to read to	est of main house? use?	
 Are there any pets/livestock at 1. Yes, in the kitchen 2. Yes, around the house, but r 3. No 	round the house or in the kitchen?	
2. Roof		
Type of roof in the kitchen:1. Mud or dung2. Ferro-cement3. Iron sheets	4. Thatch 5. Tiles 6. Other	
If 'other' please specify (This box should only be used if previous question)	answer '6' has been given for the	
Permanent ventilation in roof1. None2. Small holes (less than 10cm3. Large holes (more than 10cr4. No roof, or very open roof	of kitchen in diameter) n in diameter)	

3. Walls		
 Type of walls in room with stove 1. Mud or mud blocks 2. Soil/cement blocks 3. Wattle (woven sticks / reeds / bamboo) 4. Iron sheets 5. Bricks 6. Stone 7. Other 	Main type of material used for walls Second type of material for wall <i>(if necessary)</i>	
If 'other' wall material, please give details – this should be answered if the last question had an answer '7' for either main or second type of wall material		

4. Eaves spaces (i.e. spaces between stove	the walls and the roof) in room with
 Depth of eaves spaces (see manual) 1. none 2. less than 10cm in depth 3. 10 - 30cm in depth 4. greater than 30cm in depth 		
 Length of eaves spaces 1. All round room 2. Along outside walls 3. Along walls within house 4. Other (<i>please indicate on sketch at end of que</i>) 	iestionnaire)	
What shape is the eaves space (Type A, Type <i>manual</i>)	B, or Type C – <i>see</i>	
5. Windows & doors		
How many windows are in the room where co	ooking is done?	
What size are the windows in the room with the community (Measure width and enter sizes in table below	he main stove? /)	
Window Sizes		Window size
Size 1 = 2 - 5cm	Window 1	
Size 2 = 6 - 14cm	Window 2	·
Size 3 = 15 - 29cm	Window 3	
Size 4 = 30 - 59cm	Window 4	
Size 5 = >60cm	Window 5	
How many doors are there in the kitchen?		
Are the door (s) usually open or closed?	······································	

6. The stove	an a	
Record main type of stove below, and secondary stove if u	ised	
Type of stove 1. Three-stone or two-stone fire . 2. Shielded mud fire or mud stove (including chimney stove) 3. Wood-burning ceramic stove (made of fired clay) 4. Matel stove	Main type of stove	
 Metal stove Improved charcoal stove Pressurised kerosene stove Non-pressurised kerosene stove Gas stove Solar cooker Grid-powered electric stove Other type of stove 	Secondary stove (if used occasionall y)	
If 'other' type of stove, please describe		
Is a stove used in any other room in the house other than the fouse other	the kitchen?	

7. Smoke extraction		
Is there any type of smoke etc)? 1. Yes 2. No	extraction in the kitchen (chimney stove, hood	
<i>If the answer is 'yes' insert method used to describe c in poor condition would have</i>	number by each type of smoke extraction ondition of hood or chimney (eg a smoke hood a '1' put in the box beside 'smoke hood')	
	Extraction method	
1. Poor condition	Chimney stove	
2. Fainy good condition	Smoke hood	
	Other:	
If 'other' smoke extraction method used, please describe (or sketch) it		

Sketch of house or kitchen: simple outline plan, indicating layout of		
Hooms, identifying kitchen (if part of main house)		
 Position of the fire/stove 		
 Position of door(s) and opening(s) (doorways without doors) Desition of uindex (a) 		
Position of window(s)		
Position of eaves spaces		
• Interior walls • Depition of monitors ($\mathbf{PEM} = \mathbf{V} : \mathbf{O}$ track = \mathbf{O})		
• Position of monitors (PEW = Λ , Q-trak = Q) Skatches please		
Skelones please		
Referring to manual:		
<i>Referring to manual:</i> Please circle correct shape code to describe the shape of the house	A	E
Referring to manual: Please circle correct shape code to describe the shape of the house	A C	E
<i>Referring to manual:</i> Please circle correct shape code to describe the shape of the house Referring to the handbook, in order to determine the volume of the	A C	B
Referring to manual: Please circle correct shape code to describe the shape of the house Referring to the handbook, in order to determine the volume of the kitchen at a later date, please measure dimensions in metres:	A C	8
Referring to manual: Please circle correct shape code to describe the shape of the house Referring to the handbook, in order to determine the volume of the kitchen at a later date, please measure dimensions in metres: (a) =	A C	8
Referring to manual: Please circle correct shape code to describe the shape of the house Referring to the handbook, in order to determine the volume of the kitchen at a later date, please measure dimensions in metres: (a) = (b) =	A C	8