HOUSEHOLD AIR POLLUTION EXPOSURE FROM COOKSTOVE RELATED WOODSMOKE AND BIOMARKERS OF OXIDATIVE STRESS AMONG WOMEN IN SAN MARCOS, PERU

AND

RADIOACTIVITY IN SMOKE PARTICULATES FROM PRESCRIBED BURNS AT THE SAVANNAH RIVER SITE AND AT SELECTED SOUTHEASTERN UNITED STATES FORESTS

by

ADWOA DEDE COMMODORE

(Under the Direction of LUKE P. NAEHER)

ABSTRACT

Objectives: To: (1) compare airborne radionuclide concentrations at the Savannah River Site (SRS) and at select forests in the Southeastern United States during prescribed burns; (2) compare carbon monoxide (CO) exposures and concentrations from cookstove related woodsmoke among intervention and control stove users in San Marcos Province, Cajamarca region, Peru; (3) examine the relationship between household air pollution (HAP) exposure and urinary 8-OHdG and 8-isoprostane among subjects in San Marcos; (4) use $PM_{2.5}$ and CO measurements to characterize HAP exposure in real time.

Methods: Total suspended particulate samples were taken at SRS and at select Southeastern US forests. Spectroscopy was performed to determine radionuclide activity concentrations. Kruskal-Wallis tests and ordinary kriging were used to analyze the data. Time integrated and real-time

$PM_{2.5}$ and CO measurements were taken from subjects using control and intervention stoves in San Marcos, Cajamarca region, Peru. The first morning voids after 48hr exposure assessment were also taken from the subjects. General linear models and correlation analyses were used to estimate associations between the measurements.

Results: Prescribed burns at SRS lead to measurable levels of ^{40}K, gross beta, plutonium, thorium and uranium isotopes compared to offsite burns and nonburn days ($p \leq 0.02$) but these levels were ≤1% of the US Department of Energy's air pathway standard. CO levels did not differ significantly between the two arms of the stove intervention in San Marcos ($p > 0.05$). Real time $PM_{2.5}$ and CO measurements were correlated, particularly during high exposure periods [lunch: $r=0.67$ $p=0.024$ $n=11$ and dinner: $r=0.72$ $p=0.0011$ $n=17$)]. Urinary 8-isoprostane levels reported in the literature are comparable to results reported in the current study. Median urinary 8-OHdG among subjects was 132.6ug/g creatinine while levels reported in the literature, also employing HPLC-ECD, range from 3-10ug/g creatinine.

Conclusion: Radiation exposure from measureable levels of airborne radionuclides during SRS prescribed burns appears insignificant; however there are potential health impacts associated with exposures to forest biomass combustion products. Results of the San Marcos studies show levels of $PM_{2.5}$ and CO mostly higher than WHO air quality guidelines. Results suggest that there is a sustained systemic oxidative stress status among these women.

INDEX WORDS: Carbon monoxide, children, cookstove, exposure assessment, household air pollution, oxidative stress, particulate matter with aerodynamic diameter ≤2.5 μm, Peru, prescribed burns, radionuclides, spatial analysis, total suspended particulates, women, woodsmoke, 8-hydroxy-2'-deoxyguanosine (8-OHdG), 8-isoprostaglandin $F_{2\alpha}$ (8-isoprostane)

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December 2012

DEDICATION

This work is dedicated to my beloved husband, Ken Nii Martey Commodore for being through everything with me. Thank you, God bless you, I love you but remember God loves you best.

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CHAPTER 1

INTRODUCTION

TWO POTENTIAL PUBLIC HEALTH CONCERNS

The relationship between the environment and human health is a complex and challenging one. Indeed there is a cause for celebration when one looks at the great strides in human health from the last century. However, these recent improvements are not evenly distributed globally. Releases of radiation into the atmosphere as a result of forest fires, and exposure to household air pollution are two instances where human health concerns may still exist. The former subject is discussed briefly herein while the latter is in more detail since it constitutes the majority of this body of work.

Forest fires have the potential to release certain constituents, including radioactive material into the atmosphere. The release of these radionuclides raises health concerns depending on the nature of the radionuclides (Peterson et al. 2007). The main concern for exposures that could occur from this airborne radioactivity is for firefighting personnel, nearby communities and the ecology. Studies on radionuclide exposure assessment need to be conducted to fill in the literature and scientific gaps that exist with the question of the fate and bioavailability of radionuclides during forest fires. A study which characterizes the concentrations of manmade and natural radionuclide isotopes in smoke during prescribed burning at the Savannah River Site (SRS) and at select southeastern United States forests is presented in Chapter 4 while a brief background on the topic is presented in Chapter 3.

In the developing world, women together with their young children, have the greatest household air pollution (HAP) exposures due to the use of biomass as cooking fuel (WHO 2011). Household stoves adopted for cooking and heating in the developing world do not burn fuel cleanly leading to incomplete combustion. HAP is responsible for almost 50% of the burden of total diseases resulting from poor household environments (Rehfuess, Mehta, and Prüss-Üstün 2006). Smoke from incomplete biomass combustion contains health-damaging pollutants including particulate matter (PM), Carbon monoxide (CO), volatile organic compounds (VOCs), and polycyclic aromatic hydrocarbons (PAHs). Cardiovascular and respiratory diseases have been associated with woodsmoke exposure (Fullerton, Bruce, and Gordon 2008; Naeher et al. 2007; Orozco-Levi et al. 2006).

There is the need to fill, with focused research, the significant gaps in the scientific literature regarding household air pollution reduction and resulting health impacts. CO and PM are major constituents of woodsmoke exposure and are considered chief inhalation hazards (Naeher et al. 2007). $PM_{2.5}$ has been identified as the best single indicator of the health effects of combustion of biomass such as wood (Naeher et al. 2007; Perez-Padilla, Schilmann, and Riojas-Rodriguez 2010). If there are woodsmoke constituents associated with PM that are easier and cheaper to measure, it may serve well in the adoption of prevention strategies to avoid exposure to the high HAP levels generated during cooking. There is limited data on measurements of HAP surrogates that are correlated in real time (Li et al. 2012a). There is therefore the need to better understand the association between woodsmoke constituents.

Aside experimental studies where the effect of woodsmoke on oxidative stress is well studied, oxidative stress as an adverse health end point has not been widely studied in humans and rarely in women in the developing world. Given the continuous exposure experienced by

these women over a lifetime, studies are needed to better understand the effects of woodsmoke at important stages in their lives, namely with the introduction of improved stoves and during child bearing years. Adequate characterization of exposure to residential biomass combustion is crucial in vulnerable populations such as in rural communities in developing countries such as Peru where biomass fuels are used on a daily basis for cooking and heating (INEI 2007).

The rest of this dissertation presents the results of the exposure assessment component of a series of studies embedded within the framework of a community-randomized controlled trial (c-RCT) in San Marcos, Cajamarca region, Peru (Hartinger et al. 2011). The c-RCT was headed by the Instituto de Investigación Nutricional (IIN) and the Swiss Tropical and Public Health Institute. The c-RCT aimed to deliver an Integrated Home-based-Intervention Package (IHIP) that will reduce acute respiratory infections (through the use of chimney stoves); reduce the incidence of diarrheal diseases (through access to safe drinking water); and reduce the contamination of child- and weaning food (through a hygiene education program).

For the exposure assessment component, HAP exposure due to cookstove generated woodsmoke in a sample of the households participating in the c-RCT was characterized. Recognizing the need to better understand the relationship between exposure to PM and other pollutants in woodsmoke, we used carbon monoxide (CO) to characterize HAP and examine correlations in CO measurements between personal mothers and children's exposures and between personal exposures and kitchen concentrations. This study is presented in Chapter 5.

Secondly, we investigated systemic oxidative stress as a possible health end-point for woodsmoke exposure among the primary household cooks using 8-OHdG and 8-isoprostane as urinary oxidative stress biomarkers. In-vitro and animal studies show that woodsmoke particles are able to induce oxidative stress (Danielsen et al. 2010; Danielsen et al. 2009). Human

experimental studies also reveal that woodsmoke may induce oxidative stress in humans (Barregard et al. 2008; Danielsen et al. 2008). The results of this study are presented in Chapter 6. Finally, we investigated the relationship between particulate matter and carbon monoxide in real time at periods with some of the highest exposure measurements during subjects' cooking activities. This study is presented in Chapter 7.

In the Appendix section of this document, an abstract of a manuscript in preparation is presented (Li et al. 2012b) (Appendix A). The aim of the study was to investigate whether the use of a chimney stove would result in lower urinary hydroxylated polycyclic aromatic hydrocarbons metabolites among intervention stove users when compared to control stove users in San Marcos, Peru. Lastly, a study which assesses 48hr levels of carbon monoxide (CO) and particulate matter ($PM_{2.5}$) in 93 kitchen environments and personal exposure in a sub set of households participating in the c-RCT described above, is presented in Appendix B. Both studies contribute to answering pertinent questions regarding the gaps in knowledge on HAP exposures. The studies shed light on woodsmoke exposure assessment by means of the use of a biomarker of inhalation exposure and the characterization of the chief inhalation hazards of woodsmoke.

As The Global Alliance for Clean Cookstoves (GACC) continues to build momentum in the effort to reduce HAP and the adverse health effects associated with it, this research will add to the limited data on HAP. The GACC, led by the United Nations Foundation, has the goal of 100 million households adopting clean and efficient cookstoves by the year 2020 (GACC 2011). In the advent of national cookstove programs in Peru and other countries, evaluation of new stove models needs to be in conjunction with understanding the health impact of cookstove related woodsmoke exposure (Fitzgerald et al. 2012a) as well as the reductions necessary to improve health (Martin et al. 2011).

REFERENCES

Barregard, L., G. Sällsten, L. Andersson, A. C. Almstrand, P. Gustafson, M. Andersson, and A. C. Olin. 2008. "Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress." *Occupational and Environmental Medicine* 65(5): 319.

Danielsen, P. H., E. V. Bräuner, L. Barregard, G. Sällsten, M. Wallin, R. Olinski, R. Rozalski, P. Møller, and S. Loft. 2008. "Oxidatively damaged DNA and its repair after experimental exposure to wood smoke in healthy humans." *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis* 642(1-2): 37-42.

Danielsen, P. H., S. Loft, N. R. Jacobsen, K. A. Jensen, H. Autrup, J. L. Ravanat, H. Wallin, and P. Møller. 2010. "Oxidative stress, inflammation, and DNA damage in rats after intratracheal instillation or oral exposure to ambient air and wood smoke particulate matter." *Toxicological Sciences* 118(2): 574-85.

Danielsen, P. H., S. Loft, A. Kocbach, P. E. Schwarze, and P. Møller. 2009. "Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines." *Mutation Research/Genetic Toxicology and Environmental Mutagenesis* 674(1-2): 116-22.

Fitzgerald, C., M. Aguilar-Villalobos, A. R. Eppler, S. C. Dorner, S. L. Rathbun, and L. P. Naeher. 2012. "Testing the effectiveness of two improved cookstove interventions in the Santiago de Chuco Province of Peru." *Science of the Total Environment* 420(0): 54-64.

Fullerton, D. G., N. Bruce, and S. B. Gordon. 2008. "Indoor air pollution from biomass fuel smoke is a major health concern in the developing world." *Transactions of the Royal Society of Tropical Medicine and Hygiene* 102(9): 843-51.

GACC. 2011. "The Global Alliance for Clean Cookstoves: Overview [Document on the Internet] [updated 2011; cited 2011 Oct 23]. Available from http://cleancookstoves.org/."

Hartinger, S., C. Lanata, J. Hattendorf, A. Gil, H. Verastegui, T. Ochoa, and D. Mäusezahl. 2011. "A community randomised controlled trial evaluating a home-based environmental intervention package of improved stoves, solar water disinfection and kitchen sinks in rural Peru: Rationale, trial design and baseline findings." *Contemporary Clinical Trials* 32(6): 864-73.

INEI. 2007. "Instituto Nacional de Estadística e Informática[Document on the Internet] [updated 2007; cited 2012 May 10]. Available from http://www.inei.gob.pe."

Li, C., S. Kang, P. Chen, Q. Zhang, J. Guo, J. Mi, P. Basang, Q. Luosang, and K. R. Smith. 2012a. "Personal PM< sub> 2.5</sub> and indoor CO in nomadic tents using open and chimney biomass stoves on the Tibetan Plateau." *Atmospheric Environment*.

Li, Z., A. Commodore, S. Hartinger, E. Pittman, D. Trinidad, R. Ostovar, K. Hubbard, A. Sjodin, C. Lanata, A. Gil, D. Mäusezahl, and L. Naeher. 2012b. "O-046: Assessment of Exposure to

PAHs in Cook Stove Intervention Projects in Peru by Urinary Biomonitoring." *Epidemiology* 23(5S): 10.1097/01.ede.0000416704.62764.3d.

Martin, W. J., R. I. Glass, J. M. Balbus, and F. S. Collins. 2011. "A Major Environmental Cause of Death." *Science* 334(6053): 180-81.

Naeher, L. P., M. Brauer, M. Lipsett, J. T. Zelikoff, C. D. Simpson, J. Q. Koenig, and K. R. Smith. 2007. "Woodsmoke Health Effects: A Review." *Inhalation Toxicology* 19(1): 67-106.

Orozco-Levi, M., J. Garcia-Aymerich, J. Villar, A. Ramirez-Sarmiento, J. Anto, and J. Gea. 2006. "Wood smoke exposure and risk of chronic obstructive pulmonary disease." *European Respiratory Journal* 27(3): 542-46.

Perez-Padilla, R., A. Schilmann, and H. Riojas-Rodriguez. 2010. "Respiratory health effects of indoor air pollution [Review article]." *The International Journal of Tuberculosis and Lung Disease* 14(9): 1079-86.

Peterson, J., M. MacDonell, L. Haroun, F. Monette, R. D. Hildebrand, and A. Taboas. 2007. "Radiological and chemical fact sheets to support health risk analyses for contaminated areas." *Human Health Fact Sheet, Argonne*: 38-39.

Rehfuess, E., S. Mehta, and A. Prüss-Üstün. 2006. "Assessing household solid fuel use: multiple implications for the Millennium Development Goals." *Environmental health perspectives* 114(3): 373.

WHO. 2011. "Indoor air pollution and health; fact sheet 292.".

CHAPTER 2

BACKGROUND ON RADIONUCLIDES IN SMOKE

RADIONUCLIDES IN THE ENVIRONMENT

Ample studies indicate that radioactive contaminants are present within uncultivated soils and ground water which can be transported via biological and physical processes into forest floor and woodland vegetation (Adriano and Pinder, 1977, McLendon et al., 1976, Pinder et al., 1984, Whicker et al., 1999, Yoschenko et al., 2006a, 2006b). Contaminants can incorporate into needles, bark, and leaves that fall and become forest floor materials (Adriano et al., 1981). Radiation accumulated on and in vegetation over time can be released into the air upon combustion (Malilay 1998).

In the western United States, measureable levels of radionuclides have been detected during both the flaming and smoldering phases of wildland fires (Reindhart et al., 2004). Emissions of gross alpha and beta activities ranged from 1.3×10^{-2} to 6.6 pCi/m^3 and 1.1×10^{-1} to 4.8 pCi/m^3 respectively (measured from total suspended particulates and particulate matter with aerodynamic diameter ≤ 10 μm (PM_{10}) samples (Reindhart et al., 2004). The release of radionuclides during forest fires in areas contaminated by nuclear material is also found in the literature. The Chernobyl Nuclear Power Plant accident and the Cerro Grande Fire near the Los Alamos National Laboratory are two major examples of such studies (Kashparov et al., 2000; Pazukhin et al., 2004; Yoschenko et al., 2006a, 2006b; Volkerding, 2004).

Following the Chernobyl accident, ^{137}Cs, ^{90}Sr, and $^{238,239,240}Pu$ were the radionuclides of greatest concern (Yoschenko et al., 2006a) although minute fractions of these radionuclides became airborne. During the Cerro Grande fires, average gross alpha activities were between 1.1 x 10^{-2} and 2.5 x 10^{-2} pCi/m^3 while gross beta activities were between 2.2 x 10^{-2} and 6.5 x 10^{-2} pCi/m^3 (Volkerding, 2004). The Savannah River Site, like the Chernobyl Nuclear Power Plant and the Los Alamos National Laboratory, is another example of a facility with nuclear materials. There is the need to characterize radionuclide emissions during fires at such facilities where nuclear materials exist.

Savannah River Site (SRS) is located near Aiken, in the upper coastal plain of South Carolina and shares a border with the Savannah River (Jannik, 1999). The United States Department of Energy (DOE) established the facility in 1951 for nuclear materials production (Garten et al., 2000). Production of a majority of nuclear materials continued until 1991 (Garten et al., 2000), but prior to this cessation, tritium and various other radionuclides were released into the environment (SCDHEC, 1999).

Today, approximately 12% of the site is still used for nuclear processing purposes whereas a large portion of the remainder is forested and managed for a variety of objectives (USFS-SR, 2005). Potential airborne radiological activities from stack emissions at SRS are due to tritium, plutonium isotopes, and other radionuclide bi-products used in nuclear material processing (SRS Environmental Report, 2008, Ellickson et al., 2002). Plutonium, cesium and uranium have been reported in the forested areas near processing facilities, with some of these areas previously subjected to prescribed burns (Corey et al., 1982). Both prescribed and wild fires at or near SRS therefore have the potential to release radioactivity into the atmosphere.

The following objectives were investigated in an effort to characterize radionuclides released during prescribed fires at SRS since the quantity of radionuclides emitted during burns is significance when considering firefighting personnel and the general public:

1. To characterize concentrations of manmade and natural radionuclide isotopes in smoke during prescribed burns at SRS, and at several regional offsite locations, as well as during days where no prescribed burns were conducted at SRS. This was in an effort to determine whether radionuclide concentrations differed in these three settings.
2. Secondarily, we aimed to examine whether i) airborne radionuclides at SRS prescribed burn areas exhibited spatial trends and ii) high radionuclide activities were located near what we refer to as SRS facilities. These SRS facilities are identified as major nuclear production facilities and reactors at SRS and defined as areas that have released and/or had the potential to release radionuclides. We hypothesize that radionuclides which stem from SRS sources will be concentrated near SRS facilities while radionuclides from natural sources and from fallout will be evenly distributed across sampling areas.

REFERENCES

Adriano, D.C. and Pinder III, J.E., 1977. "Aerial deposition of plutonium in mixed forest stands from nuclear fuel reprocessing." *Journal of Environmental Quality* 6(3): 303-307.

Adriano, D.C., Hoyt, G.D., and Pinder III, J.E., 1981. "Fallout of cesium-137 on a forest ecosystem in the vicinity of a nuclear fuel reprocessing plant." *Health Physics* 40: 369-376.

Agency for Toxic Substances and Disease Registry (ATSDR), 2007. "The ATSDR Public Health Assessment 2007: Evaluation of Off-Site Groundwater and Surface Water Contamination at the Savannah River Site (USDOE)." EPA Facility ID: SC1890008989.

Corey, J.C., Pinder III, J.E., Watts, J.R., Adriano, D.C., Boni, A.L., and McLeod, K.W., 1982. "Stack-released plutonium in the terrestrial environment of a chemical separations facility." *Nuclear Safety* 23, 310-319.

Ellickson, K.M., Schopfer, C.J., Lioy, P.J., 2002. "The bioaccessibility of low level radionuclides from two Savannah River Site soils." *Health Physics* 83, 476-484.

Garten, C.T., Hamby, D.M., Schreckhise, R.G., 2000. "Radiocesium discharges and subsequent environmental transport at the major US weapons production facilities." *Science of the Total Environment* 255, 55-73.

Jannik, G.T., 1999. "Critical radionuclide/critical pathways analysis for the U.S. Department of Energy's Savannah River Site." *Risk Analysis* 19, 417-425.

Kashparov, V.A., Lundin, S.M., Kadygrib, A.M., Protsak, V.P., Levtchuk, S.E, Yoschenko, V.I., Kashpur, V.A., and Talerko, N.N., 2000. "Forest fires in the territory contaminated as a result of the Chernobyl accident: radioactive aerosol resuspension and exposure of firefighters." *Journal of Environmental Radioactivity* 51: 281-298.

Malilay, J., 1998. "A review of factors affecting the human health impacts of air pollutants from forest fires. In: WHO/UNEF/WMO. Health guidelines for vegetation fire events-background papers"; 1999. 258-274.

McLendon, H.R., Stewart, O.M., Boni, A.L., Corey, J.C., McLeod, K.W., and Pinder III, J.E., 1976. "Relationships among plutonium contents of soil, vegetation, and animals collected on and adjacent to an integrated nuclear complex in the humid southeastern United States of America. In transuranium nuclides in the environment." Intl. Atomic Energy Agency Symp., 17-21, pp. 347-363, Nov. 1974, San Francisco. IAEA, Vienna, Austria.

Pazukhin, E.M., Borovoi, A.A., and Ogorodnikov, B.I., 2004. "Forest fire as a factor of environmental redistribution of radionuclides originating from Chernobyl accident." *Radiochemistry* 46(1): 102-106.

Pinder III, J.E., McLeod, K.W., Alberts, J.J., and Adriano, D.C., 1984. "Uptake of ^{244}Cm, ^{238}Pu and other radionuclides by trees inhabiting a contaminated flood plain." *Health Physics* 47(3): 375-384.

Reinhardt, T., Wrobel, C., Eberhart, C., 2004 (LA-14113). Radionuclide emission factors from prescribed burns in Northern New Mexico. Los Alamos, NM: Los Alamos National Laboratory publication. Retrieved from http://www.lanl.gov/environment/air/docs/reports/LA-14113.pdf on June 28, 2010.

South Carolina Department of Health and Environmental Control (SCDHEC) 1999. Environmental Surveillance and Oversight Program (ESOP) Data Report. Columbia, SC, Retrieved from http://www.scdhec.gov/environment/envserv/docs/ESOPDat99.pdf on January 20th 2011.

SRS Environmental Report, 2008 (SRNS-STI-2009-00190). Environmental Surveillance, Chapter 5. Donald Padgett, Monte Steedley, Pete Fledderman, and Teresa Eddy: Regulatory Integration & Environmental Services and Timothy Jannik: Savannah River National Laboratory. Retrieved from http://www.srs.gov/general/pubs/ERsum/er09/08erpdfs/chap5.pdf on January 20, 2011.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), 2000. Sources and effects of ionizing radiation; Annex J. Exposure and effects of Chernobyl accident, report to General Assembly, United Nations, New York, NY.

United States Department of Agriculture Forest Service-Savannah River (USFS-SR), 2005. Natural Resources Management Plan for the Savannah River Site. Prepared for United States Department of Energy (DOE) by (USFS-SR). New Ellenton, SC. Retrieved from http://www.fs.usda.gov/Internet/FSE_DOCUMENTS/stelprdb5208304.pdf on September 11 2010.

Volkerding, J. M., 2004. "Comparison of the radiological dose from the Cerro Grande fire to a natural wildfire." *Environment International* 29, 987-93.

Whicker, F.W., Hinton, T.G., Orlandini, K.A., and Clark, S.B., 1999. "Uptake of natural and anthropogenic actinides in vegetable crops grown on a contaminated lake bed." *Journal of Environmental Radioactivity* 45: 1-12.

Yoschenko, V.I., Kashparov, V.A., Prostak, V.P., Lundin, S.M., Levchuk, S.E., Kadygrib, A.M., Zvarich, S.I., Khomutinin, Y.V., Maloshtan, I.M., Lanshin, V.P., Kovtun, M.V., and Tschiersch, J., 2006a. "Resuspension and redistribution of radionuclides during grassland and forest fires in the Chernobyl exclusion zone: part 1." Fire experiments. *Journal of Environmental Radioactivity* 86: 143-63.

Yoschenko, V.I., Kashparov, V.A., Levchuk, S.E., Glukhovskiy, A.S., Khomutinin, Y.V., Protsak, V.P., Lundin, S.M., and Tschiersch, J., 2006b. "Resuspension and redistribution of radionuclides during grassland and forest fires in the Chernobyl exclusion zone: part II. Modeling the transport process." *Journal of Environmental Radioactivity* 87(3): 260-78.

CHAPTER 3

RADIOACTIVITY IN SMOKE PARTICULATES FROM PRESCRIBED BURNS AT THE SAVANNAH RIVER SITE AND AT SELECTED SOUTHEASTERN UNITED STATES FORESTS[1]

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Abstract

Background: Forest fire events have the potential to release both natural and anthropogenic radionuclides into the atmosphere.

Objectives: We compare airborne radionuclide concentrations at the Savannah River Site (SRS) and a sample of forests in the Southeastern United States during prescribed burns. Moreover the spatial trends of airborne radionuclide concentrations from prescribed burn areas at SRS were characterized.

Methods: Total suspended particulate (TSP) samples were taken at three settings (subsequently termed burn sample populations): during prescribed burns at SRS (n=34), on nonburn days at SRS (n=12) and during prescribed burns at five offsite locations in the Southeastern United States (n=2 per location). Mass concentrations of TSP were calculated and alpha, beta and gamma spectroscopy was performed to determine radionuclide activity concentrations. In order to determine whether spatial trends existed in radionuclide concentrations during SRS prescribed burns, spatial correlation was assessed and ordinary kriging was used to create continuous surface maps across our study area.

Results: Median activity concentrations of natural radionuclides including ^{40}K, thorium and uranium isotopes (n=34) were higher in samples from SRS prescribed fires (p<0.02) compared to offsite locations (n=10) and nonburn days (n=12). Median gross beta activity was also higher at SRS (p<0.0001). Median concentrations of anthropogenic radionuclides did not significantly differ among burn sample populations except for ^{238}Pu (p=0.0022) and ^{239}Pu (p=0.014) with median concentrations of 8.41×10^{-4} and 6.72×10^{-5} pCi/m^3 at SRS compared to 1.55×10^{-4} and -7.07×10^{-6} pCi/m^3 (nonburn days) and 1.46×10^{-4} and 2.78×10^{-6} pCi/m^3 (offsite burns) respectively. Results from our spatial analysis found that only ^{40}K demonstrated significant

spatial correlation (X^2=15.48, p=0.0004) but spatial trends in maps generated from kriging parameters do not appear to directly link areas with higher activity concentrations with SRS facilities.

Conclusion: Results indicate that prescribed burns at SRS lead to measurable levels of ^{40}K, gross beta, plutonium, thorium and uranium isotopes compared to offsite burns and nonburn days, yet there were no apparent spatial trends in relation to SRS facilities and high radionuclide concentrations.

INTRODUCTION

Savannah River Site (SRS) is located near Aiken, in the upper coastal plain of South Carolina and shares a border with the Savannah River (Jannik, 1999). The United States Department of Energy (DOE) established the facility in 1951 for nuclear materials production (Garten et al., 2000). Production of a majority of nuclear materials continued until 1991 (Garten et al., 2000), but prior to this cessation, tritium and various other radionuclides were released into the environment (SCDHEC, 1999). Today, approximately 12% of the site is still used for nuclear processing purposes whereas a large portion of the remainder is forested and managed for a variety of objectives (USFS-SR, 2005). Potential airborne radiological activities from stack emissions at SRS are due to tritium, plutonium isotopes, and other radionuclide bi-products used in nuclear material processing (SRS Environmental Report, 2008, Ellickson et al., 2002). Plutonium, cesium and uranium have been reported in the forested areas near processing facilities, with some of these areas previously subjected to prescribed burns (Corey et al., 1982).

Natural sources of radioactivity also exist. Isotopes of uranium, thorium, radium and ^{40}K are naturally occurring radionuclides in the earth's crust (Ghiassi-Nejad et al., 2001). ^{40}K activity concentration in soil is an order of magnitude higher when compared with ^{238}U or ^{232}Th but all of these radionuclides can become airborne due to resuspension of soil particles (UNSCEAR, 2000). Above ground nuclear testing also contributes to environmental radioactivity (Hu et al., 2010). Various fission products and actinides were released into the environment as a result of nuclear weapons testing, causing worldwide contamination.

Accidental release of radionuclides is another key factor in environmental radioactivity. An estimated 3.2×10^{20} pCi of radioactivity was released from the Chernobyl accident in 1986 (UNSCEAR, 2000). In 1964, a U.S. satellite containing 1kg of ^{238}Pu was accidentally destroyed

over the Indian Ocean resulting in the release of plutonium into the atmosphere (Hardy et al., 1973). Radionuclides from the above mentioned sources can be released into the atmosphere by fire events. In the western United States, measureable levels of radionuclides have been detected during both the flaming and smoldering phases of wildland fires (Reindhart et al., 2004). Emissions of gross alpha and beta activities ranged from 1.3×10^{-2} to 6.6 pCi/m^3 and 1.1×10^{-1} to 4.8 pCi/m^3 respectively (measured from total suspended particulates and particulate matter with aerodynamic diameter $\leq$10 μm (PM_{10}) samples; original results presented in fCi/m^3) (Reindhart et al., 2004). ^{234}U, ^{235}U and ^{238}U and ^{238}Pu and ^{239}Pu were also detected with uranium attributed to natural geologic sources and plutonium to atmospheric fallout. During the Cerro Grande fires, average gross alpha activities were between 1.1×10^{-2} and 2.5×10^{-2} pCi/m^3 while gross beta activities were between 2.2×10^{-2} and 6.5×10^{-2} pCi/m^3 (Volkerding, 2004) (original results in Bq/m^3, converted to pCi/m^3 using 1Bq = 27 pCi). ^{234}U levels were between 1.4×10^{-2} and 2.5×10^{-2} pCi/m^3 whereas plutonium had results of nondetection (Volkerding, 2004).

Given the numerous sources of radioactivity, it is hypothesized that beside radionuclides released in stack emissions, radionuclide concentrations in the atmosphere at SRS may be comparable to airborne concentrations in sampled southeastern United States forests. Objectives of this study were to characterize concentrations of manmade and natural radionuclide isotopes in smoke during prescribed burns at SRS, and at several regional offsite locations, as well as during days where no prescribed burns were conducted at SRS. This was in an effort to determine whether radionuclide concentrations differed in these three settings. Secondarily, we aimed to examine whether i) airborne radionuclides at SRS prescribed burn areas exhibited spatial trends and ii) high radionuclide activities were located near what we refer to in this paper as SRS facilities (Fig. 3.1).

These SRS facilities are identified as major nuclear production facilities and reactors at SRS and defined as areas that have released and/or had the potential to release radionuclides (ATSDR, 2007; Carlton, 1999). We hypothesize that radionuclides which stem from SRS sources will be concentrated near SRS facilities while radionuclides from natural sources and from fallout will be evenly distributed across sampling areas.

METHODS

Prescribed Burns

Prescribed burns at SRS are conducted mainly to improve and maintain red-cockaded woodpecker habitat and reduce hazardous fuel buildup (Kilgo and Blake, 2005). Decisions to burn on any particular day are based on South Carolina Smoke Management guidelines, weather parameters (temperature, wind speed and direction), fuel conditions, and availability of personnel.

Air Sample Collection

Total suspended particulates were collected for 4 years: 2003, 2004, 2006, and 2007 during prescribed burns at SRS (Universal Transverse Mercator (UTM) midpoint: 17 437781 3678807N). High-volume total suspended particle (TSP) samplers (General Metal Works, Model GMWL2000) were used to collect suspended particles on 20.3 x 25.4 cm (8 x 10-inch) glass fiber filters. TSP samplers ran at 1.5 m^3/min for approximately 19, 1.5 and 1.7 hours during nonburns, SRS burns and offsite burns respectively. Table 1 (part 1) presents the burn area, sampling duration, volume, mass and TSP mass concentration of each sample at the three burn sample populations.

Each sampling monitor was placed as close to the fire as possible, but outside the actual fire perimeter. Normal distances from the fire were less than 2 meters at all sampling sites. As

an internal control, air samples were taken during nonburn days at SRS in 2003. In 2003 and 2004, prescribed burns at five different locations were monitored and samples served as offsite controls (Table 3.1).

Offsite areas (UTM midpoint) monitored include Piedmont National Wildlife Refuge (17 243456E 3668541N), Oconee National Forest (17 267066E 3681821N) and Francis Marion National Forest (17 267066E 3681821N), which are designated for recreational purposes; and Fort Benning (16 691971E 3581061N) and Fort Gordon (17 392151E 3698451N) used for field and military training operations. These five offsite locations are similar in that prescribed burns are administered regularly to control wildfires and improve ecology. There are no records of the use of anthropogenic radioactive materials at any of these sites. TSP sampling began at the time of ignition and continued until the end of the burn or as long as battery power lasted. Filters were stored at -20°C, transported to the University of Georgia (UGA), Athens for gravimetric analysis at the Air Quality Laboratory and then sent to the Environmental Bioassay Laboratory at SRS for radiological analysis.

Gravimetric Analysis

Filters were desiccated in climate-controlled conditions (20.6 ± 1.4°C; 31 ± 13% relative humidity) for a 24-hr period prior to the initial weighing of unused filters and the weighing of the sampled filters. Each filter was weighed twice before and after sampling using Fisher Scientific's accu-124 analytical balance (0-120g, 0.1mg). Mass concentrations (weight/m^3 of air sampled) were derived by dividing the average mass of each filter weight by the intake volume of sampled air. Air densities during weighing sessions, nominal densities of calibration masses, and control filters were used to adjust the balance readings for the buoyancy effect of air.

Radiological Analysis

Filters were analyzed for actinides, gross alpha, gross beta and gamma emitting radionuclides. All actinides were counted for 16 hours; gamma emitting nuclides for 5000 seconds; and gross alpha and nonvolatile beta samples for 40 minutes. The 2003 and 2004 filters were received in a Petri dish geometry and counted directly for gamma radionuclides using a Canberra ® /Nuclear Data Genie High Purity Germanium Gamma analysis system with a coaxial germanium detector with a 40% relative efficiency (Canberra® Model GC4019). The 2006 and 2007 filters were folded and placed into calibrated red cap-plug geometry for gamma spectroscopy analysis using the Canberra® Model GC4019. Due to the standard calibrated geometry, there is no expected difference between the 2003/2004 and 2006/2007 samples. The Genie software applies the ANSI N42.14 standard in calculating the quantity of radioactivity in a sample (ANSI, 1999). Radiological activity was measured in pCi/m^3 The radioisotopic concentration results were decay-corrected to the sampling date.

Filter samples were then prepared for actinides and strontium analyses. The preparation protocol included an aggressive hydrofluoric/nitric acid digestion procedure. Nitric and hydrofluoric acids were prepared from reagent-grade acids. To correct for chemical yield, ^{242}Pu tracer, ^{243}Am tracer, ^{232}U tracer, ^{229}Th tracer, and 4.39 mg stable Sr carrier were included in the separation process. The radioisotopic separation utilizes TEVA Resin® (Aliquat ™336), TRU-Resin® (tri-n-butylphosphate (TBP) and octyl (phenyl) N,N diisobutylcarbamoylmethylphosphine oxide (CMPO)), and Sr Resin (4, 4', (5') di-t-butylcyclohexane-18-crown-6) cartridges under vacuum. The radiochemistry separation process is described by Maxwell et al. (2010). Plutonium, americium, and uranium measurements were performed by alpha-particle pulse-height measurements using Passivated Implanted Planar

Silicon (PIPS) detectors. The PIPS detectors have an active surface of 450 mm^2. The nominal counting efficiency for these detectors is 0.30. The distance between the sample and detector surface is ~3mm. Strontium measurements were performed using an Oxford Tennelec LB5100 Gas Flow Proportional Counter which contains 80 $\mu g/cm^2$ mylar detector window with a window to carrier distance of 0.32 cm. The nominal beta counting efficiency for this detector is 0.45. The calculation for the actinides and strontium radionuclide concentrations, *a*, "massic activity" (McCroan, 2007) is as seen in equation (1).

$$a = (R_G - R_B)/\varepsilon_i \Omega \ell s p f D Y m \quad (1)$$

Where R_G = Observed count rate of sample

R_B = Measured background count rate

ε_i = Intrinsic efficiency of the detector

Ω = The geometric relation of sample and detector, or solid angle

ℓ = Factor for attenuation loss in the sample, detector window, and intervening material

s = Factor for scattering into the detector by sample support and shield

p = Factor for pulse losses and discrimination in the pulse processing system

f = The decay fraction of the measured radiation

D = The fractional extent of radioactive decay or ingrowth

Y = The chemical recovery fraction or yield

m = The sample mass or volume.

Radiological activity in pCi/m^3 was considered measurable if activity was above the minimum detection concentration (MDC, also in pCi/m^3) for radioisotopes in each sample. MDCs account for factors such as count time, efficiency of detector, quantity of material analyzed and background levels measured for a given day. The MDCs are calculated at the 95-

percent confidence level using the Curie MDC application (Currie, 1968). Reference MDCs for the four year sampling period can be found in annual SRS Environment Reports for the respective years (SRS Environmental Reports, 2003, 2004, 2006, 2007). Lastly, data (Supplementary Table 3.1) were converted into activity/mass (pCi/mg) by multiplying the activity concentration in pCi/m^3 by the inverse of the mass concentration of TSP (mg/m^3). The data in Supplementary Table 3.1 was generated in an attempt to quantify study results in activity/mass. Only descriptive statistics are provided for data in activity/mass; all other data analyses are based on activity/volume data.

Statistical Analysis

Mean activity concentrations (pCi/m^3) with 95% confidence intervals (CI) were calculated for each radionuclide at each site (SRS, offsite and nonburn days, subsequently referred to as burn sample populations). Bartlett's tests for homogeneity of variance between burn and nonburn days at SRS were performed to analyze for differences in the variances of the radionuclides measured at SRS. To avoid modeling assumptions regarding the statistical distributions of radionuclide concentrations, Kruskal-Wallis one way analysis of variance by ranks was used to test the equality of median radionuclide activity concentrations for all three burn sample populations. Hence for each isotope, the model allowed the determination of differences in the median activity concentrations by site. SAS version 9.1 (Cary, N.C.) was used for all statistical analysis. Results were determined to be statistically significant at $p<0.05$.

Spatial Analysis

The objective of our spatial analysis was to test if radionuclide concentrations at SRS exhibit spatial dependency and to identify if high concentrations are near operating facilities. Since prescribed burns only take place in certain areas at SRS, we expect maps to provide a

visual reference of concentration gradients across burn areas only, allowing the identification of where high concentrations (or vice versa) occur, especially when radiological doses are considered. The data used in this analysis include spatial coordinates for each field sample (collected with global positioning systems (GPS)), locations of SRS operating facilities, forest management compartments and the SRS administrative boundary provided by the United States Forest Service-Savannah River (USFS-SR). All Geographic data were spatially referenced using 1927 North American Datum and then projected using the Universal Transverse Mercator (UTM) zone 17N coordinate system. Depending on the year and prescribed burn location, some locations are sampled more than once (Table 1).

Spatial analysis was conducted in a two-step process. First, spatial dependence among observations at SRS only (n=34) was described by fitting an exponential variogram to the data. This is expressed as follows in equation (2):

$$2\gamma(d) = c_0 + c_1\left(1 - \exp\{-d/\alpha\}\right) \qquad (2)$$

where γ is the variogram,

d is the distance between data points,

c_0 is the nugget effect,

c_1 is the partial sill,

and α is the range parameter.

The nugget effect c_0 describes the small-scale spatial variation, variation at scales shorter than the distances between neighboring samples, the sill $c_0 + c_1$ is equal to twice the population variance of the data, and α is the range parameter. This variogram assumes that the strength of spatial dependence depend only on the distance d between a pair of sampling locations. Pairs of samples further than distance 3α are negligibly correlated. Restricted maximum likelihood

(REML) estimates of model parameters were estimated using SAS *PROC MIXED* (Littell et al. 1996).

Second, ordinary kriging (Webster and Oliver 2001) based on previously described spatial models was used to interpolate smooth surface maps of radionuclide concentrations from our point sample locations over the 4 sampling years. Kriging maps were developed with prediction areas constrained to the minimum and maximum coordinates of our data. Ordinary kriging was performed using the *krigconv* function in the 'geoR' package and final maps were created using the 'map' and 'maptools' packages in R (R: A Language and Environment for Statistical Computing, Vienna, Austria).

RESULTS

Radionuclide Concentrations

At SRS, eight prescribed burns were sampled in 2003, eight in 2004 and 2006 and ten in 2007. Two prescribed burns were sampled at each of the offsite locations: in 2003 (at Francis Marion National Forest only) and in 2004 at the four other offsite locations. Twelve SRS nonburn days were sampled in 2003. Locations of SRS prescribed burns and sampling locations are provided (Figure 3.1). Mean TSP concentrations (95% CI) were 0.93 (0.61, 1.24), 1.70 (1.12, 2.29) and 0.025 (0.019, 0.031) mg/m^3 for burns at SRS, offsite burns and nonburn days respectively (Table 3.1).

Measured radionuclide activity concentrations from the three burn sample populations (SRS burns, offsite burns and SRS nonburn days) along with their means and 95% CI are provided (Table 3.1). Seventeen radionuclides were analyzed in the SRS burn samples, and 16 radionuclides in offsite locations and nonburn day samples. Percentage of radionuclides with measurable activity concentration values ranged from 38-91% at SRS, 0-100% offsite and 0-94%

during nonburn days (Table 3.1). Natural radionuclides detected include ^{40}K, thorium isotopes (^{228}Th, ^{230}Th, ^{232}Th), and uranium isotopes (^{234}U, ^{235}U, ^{238}U). Anthropogenic radionuclides with measurable activity concentrations include ^{241}Am, ^{137}Cs, ^{60}Co, curium isotopes (^{242}Cm, ^{244}Cm), plutonium isotopes (^{238}Pu, $^{239, 240}$Pu) and $^{89, 90}$Sr. Summary statistics are presented, with SRS nonburn day samples referred to as CSRS (Table 3.1 and Fig. 3.2). Comparisons among the three burn sample populations are not possible for $^{89, 90}$Sr as the radionuclide was only analyzed in the 2006 and 2007 filter samples for SRS prescribed burn days (Table 3.1, part 4).

Gross alpha and beta activities in each sample were also determined (Table 3.1 and Fig. 3.3). Measurable activity concentration values of gross alpha and beta were 65% and 100% at SRS; 80% and 100% offsite respectively, and 100% for both during nonburn days (Table 3.1). ^{40}K, ^{137}Cs, $^{89, 90}$Sr and ^{60}Co are the radionuclides expected to contribute the most to the gross beta measurements. However, ^{60}Co and ^{137}Cs had negative mean activity concentrations during burns at SRS. $^{89, 90}$Sr was only measured during burns at SRS and had activity concentration of (mean ± standard deviation, $5.15 \times 10^{-3} \pm 6.92 \times 10^{-3}$ pCi/m^3).

Bartlett's test revealed differences in the variances of radionuclide activity concentrations when burns days are compared with nonburn days at SRS (p value <0.0005 for 16 radionuclides; n=34 and 12 for SRS and CSRS respectively, except for ^{241}Am, ^{242}Cm and ^{244}Cm where n=11, results not shown). Radionuclide activity concentrations differed depending on whether a prescribed burn occurred at SRS; with nonburn days consistently having lower radionuclide activity (refer to observed trends in Fig. 3.2 and Fig. 3.3).

The Kruskal-Wallis test was employed to test for differences in median radionuclide activity concentrations among the three burn sample populations (Table 3.2). Results (chi square and p value) reveal that gross beta (X^2=22.15 p<0.0001), ^{238}Pu (X^2=14.48 p=0.0007), ^{239,240}Pu

(X^2=8.00 p=0.0183), ^{228}Th (X^2=10.91 p=0.0043), ^{230}Th (X^2=7.05 p=0.0294), ^{234}U (X^2=14.71 p=0.0006), ^{235}U (X^2=10.71 p=0.0047), ^{238}U (X^2=11.38 p<0.0034) do not have identically shaped distributions when the ranked scores are examined for each burn sample population. The median activity concentrations of the aforementioned radionuclides differed significantly among the three burn sample populations.

Summary statistics for data in pCi/mg are provided in Supplementary Table 3.1. ^{40}K, gross alpha and beta had increased activity/mass during nonburns at SRS compared with the other burn sample populations. ^{228}Th, ^{230}Th and ^{238}U had increased activity/mass during SRS burns compared to offsite and nonburn samples. ^{238}Pu and ^{234}U activity/mass were of the same order of magnitude (10^{-2}) during burns and nonburns at SRS. The other radionuclides had mean concentrations in pCi/mg close to 0 or were negative.

Spatial Distribution of Radionuclides

Given the limited sample size and sampling locations of TSP samples during prescribed burns at SRS, results from representative radionuclides are presented and subsequently discussed. Parameter estimates used in the spatial models are presented (Table 3.3). Spatial maps (herein referred to as continuous surface maps) for three radionuclides of interest at SRS (^{40}K, ^{137}Cs and ^{238}Pu are examples of radionuclides stemming from natural sources, nuclear fallout and SRS activities respectively) are presented in Fig. 3.4 a, b and c.

Results for ^{40}K revealed a statistically significant spatial pattern (p=0.0004, Table 3.3) across our study area. Our continuous surface map demonstrated high activity concentrations near the center of the site and towards the northern boundary (Fig. 3.4 a). Results for ^{137}Cs showed a statistically insignificant spatial correlation (p=0.3419, Table 3.3) with no noticeable spatial trend in the continuous surface map (Fig. 3.4 b). Likewise, ^{238}Pu had a statistically insignificant

spatial correlation (p=0.9852, Table 3.3) although the continuous surface map appeared to have elevated levels toward the northeast of SRS (Fig. 3.4 c). Spatial trends in the all maps generated from the kriging parameters do not appear to directly link areas with higher activity concentrations with SRS facilities.

DISCUSSION

The primary focus of the discussion is the comparison of natural and anthropogenic radionuclides that differ significantly at the three burn sample populations. We also compare radionuclide measurements from other forest fires in literature. Finally we compare our results with calculated dose data to determine whether the radioactive emissions from SRS prescribed burns contribute to radiological dose.

Natural Radionuclides

^{234}U and ^{235}U were detected in smaller quantities during burns at SRS compared to ^{238}U and concentrations from nonburn days and offsite locations were of the same order of magnitude. This suggests similar airborne levels of these two radionuclides in the Southeastern US. ^{238}U on the other hand is the most abundant, naturally occurring uranium isotope (Peterson et al., 2007) and is present in the soil at SRS (SRS Environmental Report, 2007) and may explain why burn days at SRS had the highest ^{238}U levels compared to offsite samples. SRS prescribed fires contributed to increased ^{238}U airborne activity leading to the trend of higher levels on burn days compared to nonburn days.

^{228}Th, ^{230}Th, ^{232}Th were higher during burns at SRS compared to the other burn sample populations. Thorium levels at SRS may be attributed to natural sources present in the soil compared to other areas in the Southeastern US or past site operations that may have included

the use of thorium in fuel. Akin to uranium, fire activity contributed to the increased activity of the radionuclides, and lead to higher airborne levels on burn days compared to nonburn days.

^{40}K is a major natural beta emitter, found in ample yet varying amounts in soil (El-Arabi, 2005). Stable potassium has been used successfully as a marker for aerosols emitted from forest biomass combustion (Anttila et al., 2008). ^{40}K is usually found where naturally occurring potassium is detected (Peterson et al., 2007). Potential sources of ^{40}K include the application of fertilizers and pesticides. However, there have been no recent fertilizer use at SRS and forest fires do not seem to release pesticides into the atmosphere since most are bound to the vegetation itself or to the soil (Bush et al., 2000).

Elevated levels of ^{40}K at SRS may be due to an accumulation of ash from prescribed fires over time since ^{40}K is found in bottom ash (Hedvall and Erlandsson, 1992). The continuous surface map for ^{40}K (Fig. 4 a) reveals elevated levels particularly toward the center as well as the north of the site but there did not appear to be a direct link between these areas with higher activity concentrations and SRS facilities (Fig. 3.1). Prescribed burns may play an important role in increasing ^{40}K activity in air.

Natural radionuclides including thorium, uranium and ^{40}K can be found in small quantities in coal, and fly ash during coal combustion (McBride et al., 1978; US EPA 2010). A heavy water reprocessing area at SRS, the D-area, constructed in 1952 and scheduled to be shut down in 2011, contains a large coal-fired plant (Ghuman et al., 2006; US EPA, 2011). Both the P-area and A-area also established in the early 1950s at SRS contain coal burning power plants as well (US EPA, 2011). We postulate that the combination of coal-fired plants at SRS, wind circulation, together with wet and dry deposition for over five decades, may have also lead to increased airborne levels of these natural radionuclides.

Anthropogenic Radionuclides

All manmade radionuclides measured in this study can be attributed either to SRS activities or found in worldwide nuclear fallout. Plutonium and strontium are subsequently discussed as they were the only anthropogenic radionuclides with significant activity concentrations and can be traced to stack emissions from SRS (SRS Environmental Report, 2007). ^{137}Cs, an important radionuclide at SRS, is discussed and chosen as a representative of the other radionuclides with no significant airborne activity concentrations.

^{238}Pu median concentration during burns at SRS is five-fold higher compared to offsite and nearly six-fold higher on nonburn days. This indicates the presence of the radionuclide at SRS, with fire activity increasing its availability in air. Although the continuous surface map for ^{238}Pu revealed high levels towards the northeast of SRS, there did not appear to be a direct link between those areas with higher activity concentrations and SRS facilities (Fig. 3.4 c) nor was there significant spatial correlation (p=0.9852). The elevated levels are consistent with the true presence of plutonium and consistent with historical results from stack releases (SRS Environmental Report, 2008). For $^{239,\ 240}$Pu, median values for both offsite and onsite burn samples were measurable while nonburn days had no measurable median activity. Plutonium is ubiquitous in the global atmosphere due to nuclear fallout. (Hölgye, 2008). Fire activity may have also increased the availability of $^{239,\ 240}$Pu due to fallout events in these forest areas.

$^{89,\ 90}$Sr is released in small amounts from SRS stacks (SRS Environmental Report, 2008). The 2007 environmental monitoring data had 2 out of 11 $^{89,\ 90}$Sr samples above the MDC (SRS Environmental Report, 2007). Since 2007, a few environmental monitoring results have shown detectable levels of $^{89,\ 90}$Sr, attributed to the implementation of a more sensitive analytical protocol (SRS Environmental Report, 2008,). In the current study strontium was detected at a

greater frequency yet with a similar wide range in variability as the SRS environmental surveillance data. Differences in air sampling locations at SRS may have led to the differences in detection frequency. Additionally, ^{90}Sr is found globally due to nuclear fallout (UNSCEAR, 2000; Peterson et al., 2007; Masson et al., 2010). Measured concentrations of $^{89, 90}$Sr at SRS possibly stem from the above cited sources.

^{137}Cs exists in soil, grassy vegetation and water samples at SRS (SRS Environmental Report, 2007). Levels of ^{137}Cs in air can be mainly attributed to nuclear fallout since results from this study are consistent with SRS historical air surveillance data (SRS Environmental Report, 2007). In a study of the severity of prescribed burns on longleaf pine stands, Sullivan et al. (2003) measured peak soil surface temperatures between 28.9 – 60.3 °C during SRS prescribed burns. Low activity concentrations of airborne ^{137}Cs across SRS burn areas (Fig. 3.4 b) may be attributed to prescribed fires which are typically surface fires and may not have high enough temperatures to volatilize ^{137}Cs with a boiling point of 678.4 °C. This will be different during forest fires which have expected temperatures between 100 – 1000 °C (Volkerding, 2004).

Gross Alpha and Gross Beta Activities

Gross alpha activity concentrations in air appear to be similar at SRS and offsite locations (Fig.3.3 and Table 3.2). Potential sources of alpha radiation may stem from the uranium, radium and thorium natural decay series possibly occurring at the sampling locations (Peterson et al., 2007). Transformation of parent radionuclide into daughter products during decay involves the emission of alpha (and beta) particles, yielding products which include radium, radon, lead and bismuth (Peterson et al., 2007). In a study which described the ambient environmental profile at SRS, gross alpha activities were comparable to measurements at the Aiken Municipal Airport located approximately 48 km from the F- and H-Areas of SRS (Rollins, 2008).

Gross beta activity on the other hand had statistically significant differences in median values when both burn and nonburn days are compared with offsite burns (Table 3.2). Of the beta emitters measured, ^{40}K has the highest activity concentration; hence the corresponding increase in gross beta activity may be attributed to this radionuclide (Table 3.1), as well as beta emitters in the natural decay series alluded to earlier. Gross alpha and beta measurements are usually performed to determine atypical radionuclide levels (Hernández et al., 2005). All gross activity measurements in this study can be used as baseline data (Ackay et al., 2007) to evaluate changes in airborne radioactivity levels at SRS in the future.

Literature Comparison of Radionuclide Concentrations Measured in Other Forest Fires

We compare airborne levels of radionuclides from forest fires which occurred at Los Alamos. Since activity concentrations are expressed in pCi/m^3 in our study, results from both studies are converted to pCi/m^3 using 1 Bq = 27 pCi.

Volkerding (2004) presents data from the Cerro Grande and Viveash fires which occurred May 2000 in New Mexico both within and around the Los Alomos National Laboratory. Air sampling was performed for multiple locations and sampling durations, and a range of average values from the two fires are compared with measurements from burn days at SRS. The only overlapping radionuclide with detectable activity concentrations in all three fires was ^{234}U. During prescribed burns at SRS, ^{234}U levels ranged from non-detect to 5.4×10^{-3} pCi/m^3 and nonburn days values ranged from non-detect to 3.8×10^{-3} pCi/m^3. In comparison, ^{234}U levels at both fires in New Mexico differed by less than 0.08%, with levels from the Cerro Grande fire between 1.4×10^{2} and 2.5×10^{2} pCi/m^3; background levels ranged from non-detect to 5.1×10^{1} pCi/m^3 (Volkerding, 2004). These values are higher than the 4 year average concentration measured during prescribed burns at SRS.

During the Cerro Grande fires, mean gross alpha activities were within the same order of magnitude as SRS concentrations. In contrast, mean gross beta at SRS were an order of magnitude higher (Fig. 3) and may be attributed to high ^{40}K activity. Gross alpha and beta measurements from the current study are also within an order of magnitude of surveillance data (SCDHEC 1999; SRS Environmental Report, 2003, 2004, 2006, 2007) thereby confirming consistency with historical data and showing long term variability in sampling results.

It is assumed in calculations of the contribution of biomass to airborne radionuclide concentrations that about 20% of most elements are emitted to the atmosphere (Amiro and Davis, 1991). Atmospheric transport models demonstrate that about 1% ^{90}Sr, 1% ^{137}Cs and 7% ^{238}Pu are emitted into the atmosphere during grassland fires in the Chernobyl exclusion zone (Yoschenko et al., 2006b). How the measured airborne radionuclide concentrations at SRS then translate into estimated personal doses is crucial and needs to be addressed.

Public Health Implications

At SRS, estimated doses from all atmospheric releases of radionuclides during the 4 years in which this study occurred were 0.4%, 1.1%, 0.6% and 0.7% of the DOE air pathway standard of 0.10 mSv/year (SRS Environmental Reports for 2003, 2004, 2006 and 2007 respectively). The lower 2003 dose compared to that in 2004 can be attributed to a reduction in operations and releases from H-Area in 2003 (SRS Environmental Report, 2004). The rise in dose in 2004 was due to increases in the estimated diffuse and fugitive releases of unspecified alpha- and beta emitters, mostly from the General Separations Area Consolidated Unit (GSACU) remediation project (SRS Environmental Report, 2006). This project was terminated in 2006 and was no longer a factor, as can be seen with the subsequent decrease in dose in 2006 and 2007 compared to 2004.

To further understand potential exposures that airborne radionuclides from smoke could pose to either firefighters working on these burns or to the general public, an SRS exposure-to-dose report (Lee and Hunter, 2006) is considered. The report calculates the highest downwind dose the general public and nearby workers receives at the SRS boundary and 100 meters from the fire line during a controlled burn adjacent to an old reactor cooling pond with elevated levels of ^{137}Cs in sediments. Assuming all of the ^{137}Cs in the available fuel was volitalized, the highest modeled dose (3.2×10^{-7} mSv), was from ^{137}Cs at the SRS boundary line during an 8 hour controlled burn. Doses were estimated from ^{137}Cs and gross beta activity concentrations.

Comparison of the Lee and Hunter report to results from the current study needs to exclude the contribution of ^{137}Cs to exposure dose since no significant airborne levels were detected in our study. Gross beta in our study had a lower mean activity/mass (3.1×10^{-3} pCi/g; data in Supplementary Table 3.1 converted from pCi/mg) compared to the Lee and Hunter report with 5.3 pCi/g. Hence the dose after exposure to smoke from the sampled SRS prescribed burns is expected to be lower than the estimated dose in the above report.

Undoubtedly, if radionuclides are present in fuel, they can be released into the environment upon a fire event and increase the overall dose to the public (Volkerding, 2004). However radionuclides released from SRS prescribed burn areas appear to be a small percentage of the total radiation dose received from numerous other radiation sources (McBride et al., 1978; Volkerding, 2004). Although radiation exposure from measureable levels of airborne radionuclides during SRS prescribed burns appears insignificant, there are potential health impacts associated with exposure to forest biomass combustion products (Naeher et al., 2007). Further exploration of this risk is needed; however, it is beyond the scope of this manuscript.

Limitations

Data presented in this study are not without limitation and must be interpreted with caution. Offsite locations and nonburn day samples used as controls had smaller sample sizes compared to the SRS samples and this may have reduced the statistical power needed to detect differences in radionuclide concentrations. Future studies will need to include larger sample sizes for control samples. Analysis of fly and bottom ashes will help elucidate how the combustion of wood and coal lead to the bioavailability of radionuclides (Etiégni and Campbell, 1991; Amiro et al., 1996). Another study limitation is that there were no analyses of soil and vegetation samples from air sampling locations to aid in emissions characterization.

Given the limited sample size and sampling locations (confined to prescribed burn areas only) measured radionuclide concentrations during prescribed burns at SRS revealed no physical patterns in relation to SRS facilities upon visual examination of continuous surface maps. Our spatial analysis was intended to provide a broad overview of radionuclide concentrations from prescribed burn areas at SRS. Therefore, our results are limited to being able to only examine spatial trends across sampled prescribed burn areas and not patterns that may exist at small scale (i.e. local) around individual SRS facilities. Nevertheless, our data can be used to make inferences in this regard; more specialized spatial analysis is needed to determine if significant localized spatial trends exist around individual facilities. Additionally extensive spatial analysis can be performed with a larger sample size to increase understanding on the airborne behavior of radionuclides.

CONCLUSION

Prescribed burns at SRS lead to measurable levels of airborne ^{40}K, gross beta, plutonium, thorium and uranium isotopes compared to offsite prescribed burns and during nonburn days at SRS. ^{40}K demonstrated significant spatial correlation across samples taken at prescribed burn areas yet none of the continuous surface maps generated from kriging parameters appear to directly link higher activity concentrations of any radionuclide with SRS facilities.

REFERENCES

Anttila, P., Makkonen, U., Hellén, H., Kyllönen, K., Leppänen, S., Saari, H., Hakola, H. 2008. Impact of the open biomass fires in spring and summer of 2006 on the chemical composition of background air in south-eastern Finland. *Atmospheric Environment*, 42**,** 6472-6486.

Agency for Toxic Substances and Disease Registry (ATSDR), 2007. The ATSDR Public Health Assessment 2007: Evaluation of Off-Site Groundwater and Surface Water Contamination at the Savannah River Site (USDOE). EPA Facility ID: SC1890008989.

Akcay, S., Tulumen, S., Oymak, S., Kaya, H.I., 2007. Determination of gross α and β activities in Ankara airborne particulate samples in 2003–2004. Journal of Radioanalytical and Nuclear Chemistry 273, 603-608.

American National Standards Institute (ANSI), 1999. American national standard for calibration and use of germanium spectrometers for the measurement of gamma-ray emission rates of radionuclides, (N42.14). New York, NY.

Amiro, B.D., Davis, P.A., 1991. A pathways model to assess transport of radionuclides from terrestrial and aquatic surfaces to the atmosphere. Waste Management 11, 41-57.

Amiro, B.D., Sheppard, S.C., Johnston, F.L., Evenden, W.G., Harris, D.R., 1996. Burning radionuclide question: what happens to iodine, cesium, and chlorine in biomass fires? Science of Total Environment 187, 93-103.

Bush, P.B., Neary, D.G., McMahon, C.K., 2000. Fire and pesticides: a review of air quality considerations, in: W. K. Moser and C. E. Moser (Eds.), Fire and forest ecology: innovative silviculture and vegetation management, Tallahassee, FL, Tall Timbers Research Station, pp. 132-136.

Carlton, W. H., 1999. Assessment of Radionuclides in the Savannah River Site Environment Summary. Chapter 2. Origin and Disposition of Radionuclides at SRS. WSRC-TR-98-O0162. Retrieved from http://www.osti.gov/bridge/servlets/purl/4786-oQrZb3/webviewable/4786.pdf on September 25, 2011.

Corey, J.C., Pinder III, J.E., Watts, J.R., Adriano, D.C., Boni, A.L., and McLeod, K.W., 1982. Stack-released plutonium in the terrestrial environment of a chemical separations facility. Nuclear Safety 23, 310-319.

Currie, L. A., 1968. Limits for qualitative detection and quantitative determination, application to radiochemistry. Analytical Chemistry 40, 586–593.

El-Arabi, A.M., 2005. Natural radioactivity in sand used in thermal therapy at the Red Sea Coast. Journal of Environmental Radioactivity 81, 11-19.

Ellickson, K.M., Schopfer, C.J., Lioy, P.J., 2002. The bioaccessibility of low level radionuclides from two Savannah River Site soils. Health Physics 83, 476-484.

Etiégni, L., Campbell, A.G., 1991. Physical and chemical characteristics of wood ash. Bioresource Technology 37, 173-178.

Garten, C.T., Hamby, D.M., Schreckhise, R.G., 2000. Radiocesium discharges and subsequent environmental transport at the major US weapons production facilities. Science of the Total Environment 255, 55-73.

Ghiassi-Nejad, M., Beitollahi, M.M., Fallahian, N., Amidi, J., Ramezani, H., 2001. Concentrations of natural radionuclides in imported mineral substances. Environment International 26, 557-560.

Ghuman, G.S., Sajwan, K.S., Paramasivam, S., 2006. Potential of fly ash and organic wastes for uses as amendments to agricultural soils: a review, in: K.S. Sajwan (Ed.), Coal combustion byproducts and environmental issues, Springer, New York, pp. 216-224.

Hardy, E.P., Jrey, P.W., Volchok, H.L., 1973. Global inventory and distribution of fallout plutonium. Nature 241, 444-446.

Hedvall, R., Erlandsson, B., 1992. Radioactivity in peat fuel and ash from a peat-fired power plant. Journal of Environmental Radioactivity 16, 205-228.

Hernández, F., Hernández-Armas, J., Catalán, A., Fernández-Aldecoa, J.C., Karlsson, L., 2005. Gross alpha, gross beta activities and gamma emitting radionuclides composition of airborne particulate samples in an oceanic island. Atmospheric Environment 39, 4057-4066.

Hölgye, Z., 2008. Plutonium isotopes in surface air of Prague in 1986-2006. Journal of Environmental Radioactivity 99, 1653-1655.

Hu, Q.H., Weng, J.Q., Wang, J.S., 2010. Sources of anthropogenic radionuclides in the environment: a review. Journal of Environmental Radioactivity 101, 426-437.

Jannik, G.T., 1999. Critical radionuclide/critical pathways analysis for the U.S. Department of Energy's Savannah River Site. Risk Analysis 19, 417-425.

Kilgo, J.C., Blake, J.I., 2005. Ecology and management of a forested landscape: fifty years on the Savannah River Site. Island Press, Covelo, CA, pp 78-81.

Lee, P.L., Hunter, C.L., 2006 (SRNL-EST-2006-00058). Maximum dose from exposure to a controlled burn near Par Pond. Savannah River Site, Aiken, SC: Washington Savannah River Company.

Littell, R.C., Milliken, G.A., Stroup, W.W., Wolfinger, R.D., 1996. SAS System for mixed models. SAS Institute Inc, Cary, NC.

Masson, O., Piga, D., Gurriaran, R., D'Amico, D., 2010. Impact of an exceptional Saharan dust outbreak in France: PM10 and artificial radionuclides concentrations in air and in dust deposit. Atmospheric Environment 44, 2478-2486.

Maxwell III, S.L., Culligan, B.K., Noyes, G.W., 2010. Rapid separation method for actinides in emergency air filter samples. Applied Radiation and Isotopes 68, 2125-2131.

McBride, J.P., Moore R.E., Witherspoon, J.P., Blanco, R.E., 1978. Radiological impact of airborne effluents of coal and nuclear plants. Science 202, 1045-1050.

McCroan, K. D., Keller, J.M., 2007. Data calculation analysis and reporting, in: B. Kahn (Ed.) Radioanalytical Chemistry, Springer, New York, NY, pp 189-219.

Peterson, J., MacDonell, M., Haroun, L., Monnete, F., Hilderbrand, R. D., Taboas, A., 2007. Radiological and Chemical Fact Sheets to Support Health Risk Analyses for Contaminated Areas. Argonne National Laboratory Environmental Science Division and U.S. Department of Energy. Retrieved from http://www.evs.anl.gov/pub/doc/ANL_ContaminantFactSheets_All_070418.pdf on June 19 2010.

Reinhardt, T., Wrobel, C., Eberhart, C., 2004 (LA-14113). Radionuclide emission factors from prescribed burns in Northern New Mexico. Los Alamos, NM: Los Alamos National Laboratory publication. Retrieved from http://www.lanl.gov/environment/air/docs/reports/LA-14113.pdf on June 28, 2010.

Representative minimum detectable concentrations for radiological analyses. Savannah River Site Environmental Reports for 2003, 2004, 2006 and 2007. Savannah River Site, Aiken, SC. Retrieved from http://www.srs.gov/general/pubs/ERsum/index.html on January 20th 2011.

Rollins, E.M., 2008. Ambient environmental profile for the Savannah River Site. Health Physics 95, 55– 68.

South Carolina Department of Health and Environmental Control (SCDHEC) 1999. Environmental Surveillance and Oversight Program (ESOP) Data Report. Columbia, SC, Retrieved from http://www.scdhec.gov/environment/envserv/docs/ESOPDat99.pdf on January 20th 2011.

SRS Environmental Report, 2003 (WSRC-TR-2004-00015). SRS Environmental Data for 2003: Sampling. Table 2: Representative Minimum Detectable Concentrations for Radiological Analyses. Retrieved from http://www.srs.gov/general/pubs/ERsum/ersum04/sampling/mdcs.pdf on January 20, 2011.

SRS Environmental Report, 2003 (WSRC-TR-2004-00015). Environmental Surveillance,

Chapter 4. Pete Fledderman, Donald Padgett and Monte Steedley: Environmental Services Section, Timothy Jannik: Savannah River Technology Center, Robert Turner: Site Utilities Department. Retrieved from http://www.srs.gov/general/pubs/ERsum/ersum04/03erpdfs/chap4.pdf on January 20, 2011.

SRS Environmental Report, 2004 (WSRC-TR-2005-00005). SRS Environmental Data for 2004: Sampling. Table 2: Representative Minimum Detectable Concentrations for Radiological Analyses. Retrieved from http://www.srs.gov/general/pubs/ERsum/er05/sampling/rep-min-rad.pdf on January 20, 2011.

SRS Environmental Report, 2004 (WSRC-TR-2005-00005). Environmental Surveillance, Chapter 4. Pete Fledderman, Donald Padgett and Monte Steedley: Environmental Services Section, Timothy Jannik: Savannah River River National Laboratory, Robert Turner: Site Utilities Department. Retrieved from http://www.srs.gov/general/pubs/ERsum/er05/04erpdfs/chap4.pdf on January 20, 2011

SRS Environmental Report, 2006 (WSRC-TR-2007-00008). SRS Environmental Data for 2006: Sampling. Table 2: Representative Minimum Detectable Concentrations for Radiological Analyses. Retrieved from http://www.srs.gov/general/pubs/ERsum/er07/sampling/mdcs06.pdf on January 20, 2011.

SRS Environmental Report, 2006 (WSRC-TR-2007-00008). Environmental Surveillance, Chapter 5. Pete Fledderman, Donald Padgett and Monte Steedley: Environmental Services Section, Timothy Jannik: Savannah River National Laboratory. Retrieved from http://www.srs.gov/general/pubs/ERsum/er07/06erpdfs/chap5.pdf on January 20, 2011.

SRS Environmental Report, 2007 (WSRC-TR-2008-00057). SRS Environmental Data for 2007: Sampling. Table 2: Representative Minimum Detectable Concentrations for Radiological Analyses (MDCs 2007 Excel sheet). Retrieved from http://www.srs.gov/general/pubs/ERsum/er08/sample.htm on January 20, 2011.

SRS Environmental Report, 2007 (WSRC-TR-2008-00057). Environmental Surveillance, Chapter 5. Pete Fledderman, Donald Padgett and Monte Steedley: Environmental Services Section, Timothy Jannik: Savannah River National Laboratory. Retrieved from http://www.srs.gov/general/pubs/ERsum/er08/07erpdfs/chap5.pdf on January 20, 2011.

SRS Environmental Report, 2008 (SRNS-STI-2009-00190). Environmental Surveillance, Chapter 5. Donald Padgett, Monte Steedley, Pete Fledderman, and Teresa Eddy: Regulatory Integration & Environmental Services and Timothy Jannik: Savannah River National Laboratory. Retrieved from http://www.srs.gov/general/pubs/ERsum/er09/08erpdfs/chap5.pdf on January 20, 2011.

Sullivan, B. T., Fettig, C. J., Otrosina, W. J., Dalusky, M. J., Berisford, C. W., 2003. Association between severity of prescribed burns and subsequent activity of conifer-infesting beetles in stands of longleaf pine. *Forest Ecology and Management,* 185**,** 327-340.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), 2000. Sources and effects of ionizing radiation; Annex J. Exposure and effects of Chernobyl accident, report to General Assembly, United Nations, New York, NY.

United States Department of Agriculture Forest Service-Savannah River (USFS-SR), 2005. Natural Resources Management Plan for the Savannah River Site. Prepared for United States Department of Energy (DOE) by (USFS-SR). New Ellenton, SC. Retrieved from http://www.fs.usda.gov/Internet/FSE_DOCUMENTS/stelprdb5208304.pdf on September 11 2010.

United States Environmental Protection Agency (US EPA), 2010. Coal-Fired Power Plant Emissions. Retrieved from http://www.epa.gov/radtown/coal-plant.html on August 11, 2011.

United States Environmental Protection Agency (US EPA) 2011. Region 4: Superfund. Savannah River Site (US DOE). Retrieved from http://www.epa.gov/region4/waste/sf/sites/fedfacs/savrivscareas.html#ou94 on August 11, 2011.

Volkerding, J. M., 2004. Comparison of the radiological dose from the Cerro Grande fire to a natural wildfire. Environment International 29, 987-93.

Webster, R., Oliver, M.A., 2001. Geostatistics for environmental scientists. John Wiley & Sons, Ltd, West Sussex, England.

Yoschenko, V.I., Kashparov, V.A., Levchuk, S.E., Glukhovskiy, A.S., Khomutinin, Y.V., Protsak, V.P., Lundin, S.M., Tschiersch, J., 2006b. Resuspension and redistribution of radionuclides during grassland and forest fires in the Chernobyl exclusion zone: part II. Modeling the transport process. Journal of Environmental Radioactivity 87, 260-278.

Table 3.1 TSP and radionuclide isotope concentrations during prescribed burns at SRS and offsite locations, and during non-prescribed burns at SRS

Table 3.1 (part 1) Mass concentrations of TSP samples collected during prescribed burns at SRS and 5 offsite locations, and during nonburns at SRS

Burn Type	**Date**	**Location**	**Burn** area (Ha)	**Run time** (min)	**Volume** (m^3)	**Mass** (mg)	**Conc**[b] (mg/m^3)
SRS Onsite Burn	2/20/03	H-Area [c]	-	50	87.80	27.60	0.31
	2/21/03	SRS 30 [d]	-	61	108.81	12.95	0.12
	3/10/03	SRS 39 [e]	-	40	54.90	35.05	0.64
	3/10/03	SRS 39	-	38	61.90	50.35	0.81
	3/11/03	SRS 19	-	17	27.90	53.15	1.90
	3/11/03	SRS 19	-	7	10.80	18.90	1.76
	3/12/03	SRS 70	-	17	27.40	<LOD	<LOD
	3/12/03	SRS 70	-	28	42.80	<LOD	<LOD
SRS Onsite Burn	2/8/04	SRS 24	172	180	274.00	285.10	1.00
	2/11/04	SRS 24	77	180	280.30	26.60	0.10
	2/19/04	SRS 17	81	150	225.10	244.70	1.10
	2/20/04	SRS 82	670	121	184.20	107.60	0.60
	2/23/04	SRS 56	97	120	183.50	86.80	0.50
	3/1/04	SRS 81/82	1111	166	251.50	243.00	1.00
	3/2/04	SRS 83	11	120	180.10	207.20	1.20
	3/4/04	SRS 17	279	150	225.11	136.30	0.60
SRS Onsite Burn	2/1/06	SRS 81	126	90	127.40	19.90	0.20
	2/2/06	SRS 51	221	90	129.30	4.40	0.03
	2/10/06	SRS 82	688	120	163.10	119.50	0.70
	2/15/06	SRS 51	473	90	122.33	376.00	3.07
	3/8/06	SRS 80	-	60	82.83	14.50	0.18

	3/28/06	SRS 81	445	60	82.83	1.80	0.02
	3/29/06	SRS 1	121	31	42.79	0.60	0.01
	3/30/06	SRS 55	-	60	80.70	20.60	0.26
SRS Onsite Burn	1/27/07	SRS 52	486	90	135.07	190.30	1.41
	1/30/07	SRS 53	486	90	141.44	120.90	0.85
	1/31/07	SRS 2	61	90	136.35	185.65	1.36
	2/8/07	SRS 25	194	90	141.44	64.30	0.45
	2/9/07	SRS 62	693	90	143.99	57.20	0.40
	2/20/07	SRS 31	425	90	139.53	107.10	0.77
	3/5/07	SRS 53	206	90	137.62	79.80	0.58
	3/7/07	SRS 19/20	346	90	136.98	456.40	3.33
	3/20/07	SRS 54	93	90	142.72	95.40	0.67
	3/21/07	SRS 28	403	90	135.07	511.30	3.79
SRS Onsite Burns (n=34)[a]		**Mean**					**0.93**
		95% (LCL, UCL)					**(0.60, 1.26)**
Offsite Burn	2/12/2003	FMNF 1[f]	2	37	58.67	134.40	2.29
	2/12/2003	FMNF 6	2	37	58.67	-	-
	4/28/2004	Fort B. 4a[g]	83	180	258.67	509.90	2.00
	4/29/2004	Fort B. 4b	130	65	88.81	533.70	6.00
	4/16/2004	Fort G. 24[h]	140	89	137.35	30.50	0.20
	4/15/2004	Fort G. 25	134	172	252.05	320.40	1.30
	1/22/2004	ONF 101/5[i]	336	120	181.79	152.90	0.80
	1/23/2004	ONF 140	135	150	224.06	156.60	0.70
	3/24/2004	PNWR 1[j]	230	124	186.10	223.00	1.20
	3/24/2004	PNWR 2	230	75	112.56	93.00	0.80

Offsite Burns (n=10)[a]		**Mean**					**1.70**
		95% (LCL, UCL)					**(0.56, 2.84)**
CSRS Onsite Nonburn	2/28/03	USFS-SR F[k]	0	1260	1944.53	11.30	0.01
	2/28/03	USFS-SR F	0	1260	1980.21	22.65	0.01
	3/3/03	USFS-SR F	0	1320	2055.81	46.40	0.02
	3/3/03	USFS-SR F	0	1320	2139.92	50.05	0.02
	3/4/03	USFS-SR F	0	1320	2141.78	33.35	0.02
	3/4/03	USFS-SR F	0	1320	2055.81	30.20	0.02
	3/8/03	USFS-SR E[l]	0	1260	1937.39	29.95	0.02
	3/8/03	USFS-SR E	0	1260	1908.85	12.85	0.01
	3/9/03	USFS-SR E	0	1320	2167.95	76.65	0.04
	3/9/03	USFS-SR E	0	1320	1973.58	74.00	0.04
	3/9/03	USFS-SR E	0	467	757.74	42.50	0.06
	3/9/03	USFS-SR E	0	467	747.16	44.45	0.06
Onsite Nonburns (n=12)[a]		**Mean**					**0.03**
		95% (LCL, UCL)					**(0.015, 0.036)**

Table 3.1 (part 2) Radionuclide isotope concentrations during prescribed burns at SRS and 5 offsite locations, and during nonburns at SRS

Burn Type	Date	Location	^{241}Am (pCi/m^3)	^{242}Cm (pCi/m^3)	^{244}Cm (pCi/m^3)	^{60}Co (pCi/m^3)	^{137}Cs (pCi/m^3)
SRS Onsite Burn	2/20/03	H-Area [c]	-1.88×10^{-5}	0.00×10^{0}	-1.02×10^{-4}	-2.57×10^{-2}	1.32×10^{-2}
	2/21/03	SRS 30 [d]	-1.96×10^{-4}	-6.60×10^{-3}	-8.21×10^{-5}	-1.84×10^{-2}	2.45×10^{-2}
	3/10/03	SRS 39 [e]	-7.57×10^{-4}	8.52×10^{-3}	4.93×10^{-6}	-3.58×10^{-2}	7.32×10^{-3}
	3/10/03	SRS 39	-9.92×10^{-4}	7.10×10^{-3}	-1.66×10^{-4}	-4.01×10^{-2}	-2.67×10^{-2}
	3/11/03	SRS 19	-1.25×10^{-3}	-4.75×10^{-2}	9.16×10^{-5}	1.06×10^{-1}	-4.57×10^{-2}
	3/11/03	SRS 19	-3.16×10^{-3}	-1.21×10^{-1}	-3.25×10^{-4}	2.36×10^{-2}	-6.03×10^{-2}
	3/12/03	SRS 70	-6.18×10^{-4}	-3.31×10^{-3}	-2.12×10^{-4}	-3.86×10^{-2}	1.28×10^{-2}
	3/12/03	SRS 70	-7.20×10^{-4}	-4.66×10^{-3}	-4.60×10^{-4}	-1.62×10^{-1}	-2.06×10^{-2}
SRS Onsite Burn	2/8/04	SRS 24	-2.86×10^{-4}	-	-2.31×10^{-4}	-1.21×10^{-2}	1.13×10^{-2}
	2/11/04	SRS 24	-1.74×10^{-4}	-	-1.05×10^{-4}	3.12×10^{-2}	-1.08×10^{-1}
	2/19/04	SRS 17	-2.21×10^{-4}	-	-3.25×10^{-4}	2.67×10^{-3}	1.22×10^{-2}
	2/20/04	SRS 82	-3.16×10^{-4}	-	3.95×10^{-5}	2.99×10^{-2}	-3.33×10^{-2}
	2/23/04	SRS 56	4.41×10^{-4}	-	7.44×10^{-4}	5.64×10^{-2}	-3.74×10^{-1}
	3/1/04	SRS 81/82	-1.07×10^{-4}	-	-2.44×10^{-4}	-8.24×10^{-3}	-1.05×10^{-2}
	3/2/04	SRS 83	-1.94×10^{-4}	-	-3.21×10^{-4}	3.61×10^{-2}	1.46×10^{-3}
	3/4/04	SRS 17	-3.42×10^{-4}	-	2.70×10^{-4}	-4.16×10^{-3}	-5.58×10^{-3}
SRS Onsite Burn	2/1/06	SRS 81	-3.94×10^{-5}	-	6.14×10^{-5}	2.29×10^{-2}	2.07×10^{-2}
	2/2/06	SRS 51	6.83×10^{-5}	-	-4.71×10^{-5}	2.00×10^{-2}	-3.49×10^{-2}
	2/10/06	SRS 82	5.05×10^{-5}	-	0.00×10^{0}	-9.88×10^{-3}	-2.57×10^{-2}
	2/15/06	SRS 51	7.24×10^{-5}	-	7.48×10^{-5}	-1.87×10^{-2}	-4.01×10^{-3}

	3/8/06	SRS 80	0.00×10^{0}	-	0.00×10^{0}	-1.44×10^{-2}	-2.98×10^{-2}
	3/28/06	SRS 81	-6.92×10^{-5}	-	1.07×10^{-4}	-4.74×10^{-2}	-3.57×10^{-3}
	3/29/06	SRS 1	3.83×10^{-4}	-	1.97×10^{-4}	1.38×10^{-2}	-4.41×10^{-3}
	3/30/06	SRS 55	-1.42×10^{-4}	-	1.10×10^{-4}	-2.23×10^{-2}	5.58×10^{-2}
SRS Onsite Burn	1/27/07	SRS 52	0.00×10^{-0}	-	-2.24×10^{-5}	4.84×10^{-2}	-1.82×10^{-2}
	1/30/07	SRS 53	0.00×10^{-0}	-	-2.14×10^{-5}	-1.46×10^{-2}	3.82×10^{-2}
	1/31/07	SRS 2	-3.93×10^{-5}	-	-6.23×10^{-5}	3.21×10^{-2}	2.47×10^{-2}
	2/8/07	SRS 25	5.81×10^{-5}	-	1.46×10^{-4}	-2.04×10^{-2}	-1.85×10^{-2}
	2/9/07	SRS 62	6.10×10^{-5}	-	-2.11×10^{-5}	-4.73×10^{-2}	1.88×10^{-2}
	2/20/07	SRS 31	6.17×10^{-5}	-	1.67×10^{-4}	-3.70×10^{-2}	2.65×10^{-2}
	3/5/07	SRS 53	6.29×10^{-5}	-	-2.20×10^{-5}	1.20×10^{-2}	-4.27×10^{-3}
	3/7/07	SRS 19/20	0.00×10^{-0}	-	-6.03×10^{-7}	1.09×10^{-2}	3.90×10^{-2}
	3/20/07	SRS 54	0.00×10^{-0}	-	-6.25×10^{-5}	2.85×10^{-2}	-4.77×10^{-3}
	3/21/07	SRS 28	6.30×10^{-5}	-	-2.24×10^{-5}	-6.32×10^{-2}	-1.18×10^{-2}
SRS Onsite Burns (n=34)[a]		**Mean**	**-2.45×10^{-4}**	**-2.09×10^{-2}**	**-2.48×10^{-5}**	**-4.88×10^{-3}**	**-1.58×10^{-2}**
		95% (LCL, UCL)	**(-4.54×10^{-4}, -3.51×10^{-5})**	**(-5.15×10^{-2}, 9.60×10^{-3})**	**(-9.62×10^{-5}, 4.66×10^{-5})**	**(-2.00×10^{-2}, 1.02×10^{-2})**	**(-3.95×10^{-2}, 7.89×10^{-3})**
Offsite Burn	2/12/2003	FMNF 1[f]	3.93×10^{-3}	0.00×10^{0}	1.15×10^{-1}	-1.76×10^{-4}	-8.36×10^{-3}
	2/12/2003	FMNF 6	-7.32×10^{-5}	0.00×10^{0}	-2.64×10^{-4}	4.08×10^{-2}	-6.56×10^{-2}
	4/28/2004	Fort B. 4a[g]	-3.78×10^{-5}	-	-3.07×10^{-4}	-1.69×10^{-2}	3.45×10^{-3}
	4/29/2004	Fort B. 4b	-6.67×10^{-4}	-	-7.22×10^{-4}	-2.26×10^{-2}	-2.51×10^{-2}
	4/16/2004	Fort G. 24[h]	-4.30×10^{-4}	-	-4.67×10^{-4}	4.85×10^{-2}	-2.82×10^{-2}
	4/15/2004	Fort G. 25	-1.84×10^{-4}	-	-2.51×10^{-4}	1.10×10^{-2}	3.66×10^{-3}

	1/22/2004	ONF 101/5 [i]	-1.81×10^{-4}	-	-3.49×10^{-4}	2.01×10^{-2}	-1.48×10^{-2}
	1/23/2004	ONF 140	7.04×10^{-4}	-	6.51×10^{-4}	1.05×10^{-2}	2.25×10^{-2}
	3/24/2004	PNWR 1 [j]	-1.90×10^{-4}	-	-3.44×10^{-4}	1.12×10^{-2}	1.88×10^{-3}
	3/24/2004	PNWR 2	-6.91×10^{-4}	-	-6.46×10^{-4}	2.05×10^{-2}	-2.24×10^{-2}
Offsite Burns (n=10)[a]		**Mean**	**2.18×10^{-4}**	**0.00×10^{0}**	**1.12×10^{-2}**	**1.23×10^{-2}**	**-1.33×10^{-2}**
		95% (LCL, UCL)	**(-6.26×10^{-4}, 1.06×10^{-3})**	**-**	**(-1.14×10^{-2}, 3.38×10^{-2})**	**(-1.54×10^{-3}, 2.61×10^{-2})**	**(-2.83×10^{-2}, 1.72×10^{-3})**
CSRS Onsite Nonburn	2/28/03	USFS-SR F [k]	-2.32×10^{-6}	-1.31×10^{-3}	-4.59×10^{-6}	4.02×10^{-4}	-6.30×10^{-4}
	2/28/03	USFS-SR F	-3.99×10^{-4}	0.00×10^{0}	-1.60×10^{-4}	1.48×10^{-2}	-5.59×10^{-2}
	3/3/03	USFS-SR F	7.07×10^{-6}	0.00×10^{0}	4.66×10^{-7}	-4.41×10^{-4}	-1.60×10^{-4}
	3/3/03	USFS-SR F	-1.39×10^{-5}	-7.07×10^{-6}	-4.18×10^{-6}	1.70×10^{-3}	-3.88×10^{-4}
	3/4/03	USFS-SR F	-	-	-	-3.21×10^{-2}	3.87×10^{-2}
	3/4/03	USFS-SR F	1.39×10^{-6}	0.00×10^{0}	-6.82×10^{-6}	1.54×10^{-3}	-2.00×10^{-3}
	3/8/03	USFS-SR E [l]	2.80×10^{-5}	0.00×10^{0}	1.66×10^{-6}	5.49×10^{-4}	-5.56×10^{-4}
	3/8/03	USFS-SR E	-1.15×10^{-5}	0.00×10^{0}	0.00×10^{0}	2.20×10^{-3}	-3.06×10^{-3}
	3/9/03	USFS-SR E	5.73×10^{-6}	-8.16×10^{-6}	4.35×10^{-6}	-1.71×10^{-3}	-1.46×10^{-3}
	3/9/03	USFS-SR E	-2.31×10^{-5}	1.76×10^{-3}	-7.94×10^{-6}	-3.52×10^{-4}	-7.78×10^{-4}
	3/9/03	USFS-SR E	-7.69×10^{-5}	2.15×10^{-3}	-1.20×10^{-5}	5.09×10^{-3}	4.88×10^{-4}
	3/9/03	USFS-SR E	-6.10×10^{-5}	2.22×10^{-3}	-1.22×10^{-5}	-1.25×10^{-3}	-7.00×10^{-4}
Onsite Nonburns (n=12)[a]		**Mean**	**-4.96×10^{-5}**	**4.37×10^{-4}**	**-1.83×10^{-5}**	**-7.98×10^{-4}**	**-2.20×10^{-3}**
		95% (LCL, UCL)	**(-1.20×10^{-4}, 2.12×10^{-4})**	**(-2.18×10^{-4}, 1.09×10^{-3})**	**(-3.33×10^{-5}, 8.45×10^{-6})**	**(-6.91×10^{-3}, 5.31×10^{-3})**	**(-1.38×10^{-2}, 9.34×10^{-3})**

Table 3.1 (part 3) Radionuclide isotope concentrations during prescribed burns at SRS and 5 offsite locations, and during nonburns at SRS

Burn Type	Date	Location	^{228}Th (pCi/m^3)	^{230}Th (pCi/m^3)	^{232}Th (pCi/m^3)	^{234}U (pCi/m^3)	^{235}U (pCi/m^3)	^{238}U (pCi/m^3)
SRS Onsite Burn	2/20/03	H-Area [c]	-3.82×10^{-3}	-8.88×10^{-3}	-1.47×10^{-3}	8.56×10^{-4}	-1.67×10^{-4}	1.13×10^{-4}
	2/21/03	SRS 30 [d]	4.78×10^{-4}	-5.22×10^{-3}	-6.87×10^{-4}	2.49×10^{-4}	-2.42×10^{-4}	6.44×10^{-4}
	3/10/03	SRS 39 [e]	-4.30×10^{-4}	-1.65×10^{-3}	5.61×10^{-4}	-4.08×10^{-4}	-2.20×10^{-4}	1.08×10^{-3}
	3/10/03	SRS 39	-1.15×10^{-3}	-4.10×10^{-3}	-5.10×10^{-4}	-1.08×10^{-3}	-2.60×10^{-4}	1.75×10^{-4}
	3/11/03	SRS 19	1.03×10^{-2}	4.15×10^{-3}	1.51×10^{-3}	5.43×10^{-3}	5.79×10^{-4}	3.97×10^{-3}
	3/11/03	SRS 19	6.80×10^{-3}	-9.53×10^{-3}	9.33×10^{-4}	2.81×10^{-3}	-2.82×10^{-3}	4.12×10^{-4}
	3/12/03	SRS 70	8.80×10^{-3}	-7.47×10^{-3}	1.43×10^{-3}	1.44×10^{-3}	3.43×10^{-4}	-5.63×10^{-5}
	3/12/03	SRS 70	1.76×10^{-3}	-8.42×10^{-3}	-5.23×10^{-4}	-1.16×10^{-3}	-1.15×10^{-3}	-8.74×10^{-4}
SRS Onsite Burn	2/8/04	SRS 24	-9.97×10^{-5}	4.81×10^{-5}	-1.20×10^{-4}	3.90×10^{-4}	-1.12×10^{-4}	-2.45×10^{-4}
	2/11/04	SRS 24	-3.01×10^{-4}	-3.89×10^{-4}	-1.64×10^{-4}	8.11×10^{-4}	-8.09×10^{-5}	-1.44×10^{-4}
	2/19/04	SRS 17	4.31×10^{-4}	8.55×10^{-4}	-2.92×10^{-4}	1.03×10^{-4}	-3.64×10^{-5}	-3.49×10^{-4}
	2/20/04	SRS 82	1.18×10^{-3}	1.04×10^{-3}	7.75×10^{-5}	1.47×10^{-3}	-3.61×10^{-5}	8.67×10^{-5}
	2/23/04	SRS 56	2.31×10^{-5}	1.85×10^{-3}	3.74×10^{-4}	9.50×10^{-4}	1.23×10^{-5}	-2.66×10^{-4}
	3/1/04	SRS 81/82	-7.40×10^{-4}	-1.11×10^{-3}	-5.68×10^{-5}	6.16×10^{-4}	2.58×10^{-5}	-2.98×10^{-4}
	3/2/04	SRS 83	1.00×10^{-3}	5.95×10^{-4}	1.55×10^{-4}	7.19×10^{-4}	5.76×10^{-6}	-2.94×10^{-4}
	3/4/04	SRS 17	3.03×10^{-6}	1.14×10^{-3}	1.56×10^{-5}	4.31×10^{-4}	1.16×10^{-4}	-1.03×10^{-4}
SRS Onsite Burn	2/1/06	SRS 81	5.49×10^{-3}	1.80×10^{-3}	5.27×10^{-4}	2.64×10^{-3}	7.65×10^{-5}	2.36×10^{-3}
	2/2/06	SRS 51	4.40×10^{-3}	8.53×10^{-4}	2.33×10^{-4}	1.67×10^{-3}	1.51×10^{-4}	1.88×10^{-3}
	2/10/06	SRS 82	2.93×10^{-3}	8.76×10^{-4}	6.11×10^{-4}	1.63×10^{-3}	2.08×10^{-4}	1.96×10^{-3}

	2/15/06	SRS 51	7.33×10^{-3}	3.69×10^{-3}	1.42×10^{-3}	4.38×10^{-3}	2.60×10^{-4}	3.14×10^{-3}
	3/8/06	SRS 80	6.75×10^{-3}	2.83×10^{-3}	4.95×10^{-4}	3.12×10^{-3}	2.44×10^{-4}	3.98×10^{-3}
	3/28/06	SRS 81	8.18×10^{-3}	2.45×10^{-3}	5.85×10^{-4}	1.70×10^{-3}	3.07×10^{-5}	1.45×10^{-3}
	3/29/06	SRS 1	1.77×10^{-2}	3.28×10^{-3}	4.50×10^{-4}	4.91×10^{-3}	5.42×10^{-4}	3.78×10^{-3}
	3/30/06	SRS 55	8.32×10^{-3}	2.37×10^{-3}	4.24×10^{-5}	2.79×10^{-3}	3.84×10^{-4}	2.50×10^{-3}
SRS Onsite Burn	1/27/07	SRS 52	9.04×10^{-4}	9.90×10^{-4}	1.82×10^{-4}	9.61×10^{-4}	2.79×10^{-5}	1.15×10^{-3}
	1/30/07	SRS 53	1.66×10^{-4}	8.68×10^{-3}	0.00×10^{0}	1.48×10^{-3}	1.94×10^{-4}	1.32×10^{-3}
	1/31/07	SRS 2	2.64×10^{-4}	9.21×10^{-4}	2.46×10^{-4}	1.17×10^{-3}	2.02×10^{-5}	1.10×10^{-3}
	2/8/07	SRS 25	3.01×10^{-3}	2.11×10^{-3}	1.77×10^{-5}	1.25×10^{-3}	2.43×10^{-4}	2.04×10^{-4}
	2/9/07	SRS 62	3.74×10^{-4}	1.48×10^{-3}	1.22×10^{-4}	9.53×10^{-4}	2.17×10^{-4}	1.54×10^{-3}
	2/20/07	SRS 31	5.87×10^{-4}	1.72×10^{-3}	8.35×10^{-5}	1.10×10^{-3}	9.76×10^{-5}	6.46×10^{-4}
	3/5/07	SRS 53	1.38×10^{-3}	1.38×10^{-3}	3.18×10^{-4}	1.49×10^{-3}	-1.01×10^{-5}	1.24×10^{-3}
	3/7/07	SRS 19/20	5.83×10^{-4}	3.06×10^{-3}	3.51×10^{-4}	1.95×10^{-4}	2.89×10^{-5}	4.88×10^{-4}
	3/20/07	SRS 54	6.94×10^{-4}	8.76×10^{-4}	1.27×10^{-4}	1.24×10^{-3}	-1.06×10^{-4}	9.78×10^{-4}
	3/21/07	SRS 28	4.67×10^{-4}	4.63×10^{-3}	1.99×10^{-4}	1.58×10^{-3}	9.09×10^{-5}	2.37×10^{-3}
SRS Onsite Burns (n=34)[a]		**Mean**	**2.76×10^{-3}**	**2.03×10^{-4}**	**2.13×10^{-4}**	**1.41×10^{-3}**	**-3.95×10^{-5}**	**1.06×10^{-3}**
		95% (LCL, UCL)	**(1.31×10^{-3}, 4.21×10^{-3})**	**(-1.15×10^{-3}, 1.56×10^{-3})**	**(1.59×10^{-5}, 4.10×10^{-4})**	**(9.15×10^{-4}, 1.90×10^{-3})**	**(-2.31×10^{-4}, 1.52×10^{-4})**	**(6.13×10^{-4}, 1.50×10^{-3})**
Offsite Burn	2/12/2003	FMNF 1[f]	3.29×10^{-3}	-1.09×10^{-2}	-2.09×10^{-3}	1.12×10^{-4}	3.38×10^{-5}	2.29×10^{-4}
	2/12/2003	FMNF 6	-1.99×10^{-3}	-1.07×10^{-2}	-1.57×10^{-3}	2.46×10^{-3}	-5.91×10^{-4}	2.19×10^{-3}
	4/28/2004	Fort B. 4a[g]	8.59×10^{-4}	4.96×10^{-4}	1.08×10^{-4}	2.16×10^{-3}	-8.04×10^{-5}	1.60×10^{-3}
	4/29/2004	Fort B. 4b	1.73×10^{-3}	2.61×10^{-3}	1.48×10^{-3}	2.21×10^{-3}	-9.84×10^{-5}	5.21×10^{-4}
	4/16/2004	Fort G. 24[h]	1.41×10^{-3}	5.20×10^{-4}	-7.98×10^{-5}	9.31×10^{-4}	-1.51×10^{-4}	-5.04×10^{-4}
	4/15/2004	Fort G. 25	2.61×10^{-4}	-2.36×10^{-4}	2.13×10^{-4}	5.18×10^{-4}	-1.76×10^{-4}	-3.21×10^{-4}

	1/22/2004	ONF 101/5 [i]	1.23×10^{-3}	1.91×10^{-3}	-4.91×10^{-5}	1.17×10^{-3}	-2.96×10^{-4}	-3.75×10^{-4}
	1/23/2004	ONF 140	-6.34×10^{-5}	6.20×10^{-4}	3.05×10^{-5}	1.10×10^{-3}	-3.82×10^{-5}	9.35×10^{-5}
	3/24/2004	PNWR 1 [j]	-7.06×10^{-5}	-1.97×10^{-5}	-1.76×10^{-4}	2.09×10^{-3}	-1.63×10^{-4}	1.18×10^{-5}
	3/24/2004	PNWR 2	-7.88×10^{-5}	1.43×10^{-3}	8.94×10^{-4}	1.80×10^{-3}	-1.93×10^{-4}	-3.36×10^{-5}
Offsite Burns (n=10)[a]		**Mean**	**6.58×10^{-4}**	**-1.43×10^{-3}**	**-1.24×10^{-4}**	**1.46×10^{-3}**	**-1.75×10^{-4}**	**3.41×10^{-4}**
		95% (LCL, UCL)	**(-2.13×10^{-4}, 1.53×10^{-3})**	**(-4.54×10^{-3}, 1.68×10^{-3})**	**(-7.70×10^{-4}, 5.22×10^{-4})**	**(9.59×10^{-4}, 1.95×10^{-3})**	**(-2.82×10^{-4}, -6.88×10^{-5})**	**(-2.06×10^{-4}, 8.89×10^{-4})**
CSRS Onsite Nonburn	2/28/03	USFS-SR F [k]	9.30×10^{-5}	-2.98×10^{-4}	-5.05×10^{-5}	9.49×10^{-6}	-1.30×10^{-6}	3.69×10^{-5}
	2/28/03	USFS-SR F	-5.38×10^{-4}	-9.25×10^{-3}	-2.27×10^{-3}	3.80×10^{-3}	-1.87×10^{-4}	8.51×10^{-4}
	3/3/03	USFS-SR F	1.41×10^{-4}	-3.74×10^{-4}	-7.10×10^{-5}	4.27×10^{-5}	-1.33×10^{-5}	1.48×10^{-5}
	3/3/03	USFS-SR F	-2.21×10^{-5}	-2.97×10^{-4}	-4.71×10^{-5}	3.77×10^{-7}	-1.57×10^{-5}	7.51×10^{-6}
	3/4/03	USFS-SR F	9.58×10^{-4}	-1.56×10^{-3}	8.94×10^{-4}	-8.80×10^{-4}	-6.47×10^{-4}	-1.96×10^{-3}
	3/4/03	USFS-SR F	-9.00×10^{-5}	-2.05×10^{-4}	-8.93×10^{-5}	-2.64×10^{-5}	-3.95×10^{-9}	3.28×10^{-5}
	3/8/03	USFS-SR E [l]	4.78×10^{-5}	-3.20×10^{-4}	-1.05×10^{-4}	3.08×10^{-5}	-1.23×10^{-6}	2.77×10^{-5}
	3/8/03	USFS-SR E	-3.74×10^{-5}	5.41×10^{-5}	-1.91×10^{-5}	4.59×10^{-5}	-1.38×10^{-5}	-1.09×10^{-5}
	3/9/03	USFS-SR E	-3.18×10^{-5}	1.72×10^{-4}	1.65×10^{-5}	2.86×10^{-5}	-1.57×10^{-5}	3.42×10^{-5}
	3/9/03	USFS-SR E	3.48×10^{-8}	-3.31×10^{-4}	-7.25×10^{-5}	2.34×10^{-5}	-4.69×10^{-7}	1.54×10^{-5}
	3/9/03	USFS-SR E	2.98×10^{-4}	-4.17×10^{-4}	7.05×10^{-5}	-4.21×10^{-5}	-1.75×10^{-5}	-2.56×10^{-5}
	3/9/03	USFS-SR E	-3.84×10^{-4}	-2.48×10^{-4}	8.83×10^{-5}	1.20×10^{-5}	-5.24×10^{-5}	-2.27×10^{-5}
Onsite Nonburns (n=12)[a]		**Mean**	**3.62×10^{-5}**	**-1.09×10^{-3}**	**-1.38×10^{-4}**	**2.54×10^{-4}**	**-8.05×10^{-5}**	**-8.32×10^{-5}**
		95% (LCL, UCL)	**(-1.28×10^{-4}, 2.77×10^{-4})**	**(-2.56×10^{-3}, 3.84×10^{-4})**	**(-5.48×10^{-4}, 2.72×10^{-4})**	**(-3.95×10^{-4}, 9.02×10^{-4})**	**(-1.86×10^{-4}, 2.47×10^{-5})**	**(-4.45×10^{-4}, 2.78×10^{-4})**

Table 3.1 (part 4) Radionuclide isotope concentrations during prescribed burns at SRS and 5 offsite locations, and during nonburns at SRS

Burn Type	Date	Location	^{40}K (pCi/m^3)	^{238}Pu (pCi/m^3)	$^{239, 240}$Pu (pCi/m^3)	$^{89, 90}$Sr (pCi/m^3)	Gr. Alpha (pCi/m^3)	Gr. Beta (pCi/m^3)
SRS Onsite Burn	2/20/03	H-Area [c]	-3.41×10^{-1}	0.00×10^{0}	-1.32×10^{-6}	-	-5.09×10^{-2}	2.12×10^{-1}
	2/21/03	SRS 30 [d]	-2.21×10^{-1}	1.72×10^{-3}	2.87×10^{-4}	-	3.11×10^{-2}	3.60×10^{-1}
	3/10/03	SRS 39 [e]	2.23×10^{0}	2.80×10^{-3}	1.49×10^{-4}	-	-1.22×10^{-1}	7.90×10^{-1}
	3/10/03	SRS 39	1.22×10^{0}	1.93×10^{-3}	0.00×10^{0}	-	-1.31×10^{-1}	5.83×10^{-1}
	3/11/03	SRS 19	4.45×10^{0}	2.08×10^{-2}	2.35×10^{-3}	-	4.59×10^{-1}	4.58×10^{0}
	3/11/03	SRS 19	3.41×10^{0}	1.74×10^{-3}	2.21×10^{-4}	-	-2.09×10^{-2}	1.08×10^{0}
	3/12/03	SRS 70	5.44×10^{0}	4.18×10^{-3}	7.76×10^{-4}	-	2.85×10^{-1}	1.45×10^{0}
	3/12/03	SRS 70	3.95×10^{0}	-3.59×10^{-4}	1.55×10^{-4}	-	-7.16×10^{-3}	1.51×10^{-1}
SRS Onsite Burn	2/8/04	SRS 24	-2.01×10^{-1}	3.31×10^{-5}	-4.51×10^{-5}	-	1.66×10^{-2}	1.42×10^{-1}
	2/11/04	SRS 24	3.28×10^{-2}	3.47×10^{-2}	1.56×10^{-4}	-	6.72×10^{-2}	2.28×10^{-1}
	2/19/04	SRS 17	-1.35×10^{-1}	9.16×10^{-4}	-8.14×10^{-5}	-	1.15×10^{-1}	3.09×10^{-1}
	2/20/04	SRS 82	-5.84×10^{-1}	2.31×10^{-4}	-6.91×10^{-5}	-	6.37×10^{-2}	3.28×10^{-1}
	2/23/04	SRS 56	-1.48×10^{-1}	9.27×10^{-5}	-8.00×10^{-6}	-	2.46×10^{-2}	2.96×10^{-1}
	3/1/04	SRS 81/82	7.91×10^{-1}	6.31×10^{-5}	-3.18×10^{-5}	-	4.65×10^{-2}	2.13×10^{-1}
	3/2/04	SRS 83	-1.28×10^{-1}	4.03×10^{-5}	1.15×10^{-5}	-	1.13×10^{-2}	2.16×10^{-1}
	3/4/04	SRS 17	8.10×10^{-2}	2.80×10^{-3}	-4.31×10^{-5}	-	-1.18×10^{-2}	5.80×10^{-1}
SRS Onsite Burn	2/1/06	SRS 81	-8.52×10^{-1}	7.80×10^{-4}	1.50×10^{-4}	-4.59×10^{-3}	4.03×10^{-2}	3.44×10^{-1}
	2/2/06	SRS 51	-3.36×10^{-1}	8.93×10^{-4}	1.35×10^{-4}	1.58×10^{-3}	4.91×10^{-2}	2.34×10^{-1}
	2/10/06	SRS 82	-1.77×10^{-1}	5.91×10^{-4}	-2.60×10^{-6}	-4.46×10^{-4}	5.23×10^{-2}	1.76×10^{-1}

	2/15/06	SRS 51	5.41×10^{-1}	7.66×10^{-4}	1.96×10^{-4}	-7.61×10^{-4}	7.51×10^{-2}	2.93×10^{-1}
	3/8/06	SRS 80	7.12×10^{-2}	1.28×10^{-3}	3.27×10^{-4}	1.03×10^{-2}	1.43×10^{-2}	4.71×10^{-1}
	3/28/06	SRS 81	1.57×10^{0}	1.40×10^{-3}	-5.12×10^{-6}	1.75×10^{-3}	-1.58×10^{-2}	3.40×10^{-1}
	3/29/06	SRS 1	-4.36×10^{-1}	2.99×10^{-3}	1.22×10^{-3}	2.26×10^{-2}	1.47×10^{-1}	7.32×10^{-1}
	3/30/06	SRS 55	-1.21×10^{0}	1.53×10^{-3}	1.96×10^{-4}	1.01×10^{-2}	-1.25×10^{-3}	5.30×10^{-1}
	1/27/07	SRS 52	5.08×10^{-1}	1.03×10^{-4}	-2.08×10^{-5}	9.14×10^{-3}	4.86×10^{-2}	1.77×10^{-1}
	1/30/07	SRS 53	-9.07×10^{-1}	6.41×10^{-4}	0.00×10^{0}	5.68×10^{-3}	5.07×10^{-2}	2.51×10^{-1}
	1/31/07	SRS 2	-4.62×10^{-1}	5.86×10^{-4}	4.83×10^{-5}	4.86×10^{-3}	-3.31×10^{-2}	1.82×10^{-1}
	2/8/07	SRS 25	-7.54×10^{-1}	1.05×10^{-3}	1.98×10^{-5}	1.19×10^{-4}	-1.88×10^{-2}	2.33×10^{-1}
SRS Onsite Burn	2/9/07	SRS 62	-4.82×10^{-1}	8.62×10^{-4}	-4.08×10^{-5}	5.66×10^{-3}	3.28×10^{-2}	1.85×10^{-1}
	2/20/07	SRS 31	-1.05×10^{0}	4.73×10^{-4}	2.01×10^{-4}	1.76×10^{-2}	3.35×10^{-2}	1.54×10^{-1}
	3/5/07	SRS 53	-1.81×10^{-1}	8.38×10^{-4}	1.42×10^{-4}	7.30×10^{-3}	-1.77×10^{-2}	2.60×10^{-1}
	3/7/07	SRS 19/20	-4.17×10^{-1}	8.43×10^{-4}	8.60×10^{-5}	3.19×10^{-3}	1.48×10^{-2}	1.58×10^{-1}
	3/20/07	SRS 54	-4.82×10^{-1}	4.22×10^{-4}	2.41×10^{-4}	-7.98×10^{-4}	-1.68×10^{-2}	1.79×10^{-1}
	3/21/07	SRS 28	-9.61×10^{-1}	3.08×10^{-4}	4.39×10^{-5}	-5.22×10^{-4}	1.64×10^{-2}	2.14×10^{-1}
SRS Onsite Burns (n=34)[a]		**Mean**	**4.07×10^{-1}**	**2.59×10^{-3}**	**1.99×10^{-4}**	**5.15×10^{-3}**	**3.67×10^{-2}**	**4.89×10^{-1}**
		95% (LCL, UCL)	**(-1.44×10^{-1}, 9.57×10^{-1})**	**(3.43×10^{-4}, 4.84×10^{-3})**	**(4.58×10^{-5}, 3.52×10^{-4})**	**(1.96×10^{-3}, 8.35×10^{-3})**	**(2.01×10^{-3}, 7.14×10^{-2})**	**(2.28×10^{-1}, 7.50×10^{-1})**
	2/12/2003	FMNF 1[f]	-3.66×10^{-1}	6.08×10^{-3}	1.62×10^{-6}	-	1.37×10^{-1}	1.01×10^{0}
	2/12/2003	FMNF 6	2.17×10^{0}	4.80×10^{-4}	4.14×10^{-5}	-	3.99×10^{-2}	8.85×10^{-1}
Offsite Burn	4/28/2004	Fort B. 4a[g]	-3.04×10^{-1}	7.59×10^{-5}	-1.74×10^{-5}	-	1.14×10^{-1}	9.67×10^{-2}
	4/29/2004	Fort B. 4b	-7.49×10^{-1}	6.68×10^{-4}	-2.95×10^{-5}	-	1.08×10^{-2}	6.93×10^{-1}
	4/16/2004	Fort G. 24[h]	-4.41×10^{-1}	2.83×10^{-4}	-8.46×10^{-5}	-	6.90×10^{-3}	5.46×10^{-1}
	4/15/2004	Fort G. 25	-4.53×10^{-1}	1.75×10^{-4}	-3.78×10^{-5}	-	3.83×10^{-3}	2.18×10^{-1}

	1/22/2004	ONF 101/5 [i]	7.08×10^{-2}	0.00×10^{0}	-1.48×10^{-4}	-	2.49×10^{-2}	2.87×10^{-1}
	1/23/2004	ONF 140	-4.06×10^{-1}	5.06×10^{-5}	-6.97×10^{-5}	-	-1.17×10^{-2}	2.18×10^{-1}
	3/24/2004	PNWR 1 [j]	-1.24×10^{-1}	9.96×10^{-5}	-2.99×10^{-5}	-	8.20×10^{-2}	2.62×10^{-1}
	3/24/2004	PNWR 2	-4.63×10^{-1}	3.04×10^{-4}	-6.71×10^{-6}	-	-2.31×10^{-2}	5.29×10^{-1}
Offsite Burns (n=10)[a]		**Mean**	**-1.07×10^{-1}**	**8.22×10^{-4}**	**-3.81×10^{-5}**	**-**	**3.85×10^{-2}**	**4.74×10^{-1}**
		95% (LCL, UCL)	**(-6.20×10^{-1}, 4.07×10^{-1})**	**(-3.31×10^{-4}, 1.97×10^{-3})**	**(-7.05×10^{-5}, -5.59×10^{-6})**	**-**	**(4.65×10^{-3}, 7.23×10^{-2})**	**(2.82×10^{-1}, 6.67×10^{-1})**
CSRS Onsite Nonburn	2/28/03	USFS-SR F [k]	-3.31×10^{-2}	1.34×10^{-4}	-7.43×10^{-6}	-	2.65×10^{-3}	1.96×10^{-2}
	2/28/03	USFS-SR F	7.28×10^{-1}	-1.05×10^{-4}	2.08×10^{-4}	-	3.63×10^{-1}	5.88×10^{-1}
	3/3/03	USFS-SR F	5.38×10^{-2}	4.81×10^{-5}	-2.76×10^{-6}	-	1.57×10^{-2}	3.38×10^{-2}
	3/3/03	USFS-SR F	-1.21×10^{-2}	3.75×10^{-6}	2.22×10^{-6}	-	3.46×10^{-3}	2.83×10^{-2}
	3/4/03	USFS-SR F	1.65×10^{0}	2.12×10^{-3}	0.00×10^{0}	-	4.77×10^{-1}	1.17×10^{0}
	3/4/03	USFS-SR F	-1.14×10^{-2}	2.39×10^{-4}	1.61×10^{-5}	-	2.02×10^{-2}	2.66×10^{-2}
	3/8/03	USFS-SR E [l]	-3.40×10^{-2}	3.88×10^{-5}	4.41×10^{-6}	-	5.94×10^{-3}	2.59×10^{-2}
	3/8/03	USFS-SR E	-1.12×10^{-2}	1.62×10^{-4}	1.58×10^{-6}	-	5.38×10^{-4}	1.51×10^{-2}
	3/9/03	USFS-SR E	4.58×10^{-2}	5.37×10^{-5}	-1.20×10^{-6}	-	2.09×10^{-2}	1.86×10^{-2}
	3/9/03	USFS-SR E	4.53×10^{-2}	3.14×10^{-4}	0.00×10^{0}	-	1.65×10^{-2}	2.81×10^{-2}
	3/9/03	USFS-SR E	-2.42×10^{-2}	4.63×10^{-4}	3.97×10^{-6}	-	4.01×10^{-2}	1.20×10^{-2}
	3/9/03	USFS-SR E	1.51×10^{-1}	4.03×10^{-4}	3.68×10^{-5}	-	1.03×10^{-2}	6.55×10^{-2}
CSRS Onsite Nonburns (n=12)[a]		**Mean**	**2.12×10^{-1}**	**3.23×10^{-4}**	**2.18×10^{-5}**	**-**	**8.14×10^{-2}**	**1.69×10^{-1}**
		95% (LCL, UCL)	**(-7.03×10^{-2}, 4.95×10^{-1})**	**(-1.13×10^{-5}, 6.57×10^{-4})**	**(-1.05×10^{-5}, 5.41×10^{-5})**	**-**	**(-9.39×10^{-3}, 1.72×10^{-1})**	**(-3.11×10^{-2}, 3.70×10^{-1})**

- denotes missing data, 95% (LCL, UCL) refers to 95% confidence intervals of mean radionuclide activity concentration, LOD refers to limit of detection. Gr alpha and beta refer to gross alpha and beta respectively. Please note that first 8 columns are repeated in the three parts of table.

[a] ^{242}Cm measurements were only in 2003 at SRS (n=8); $^{89, 90}$Sr measured in 2006 and 2007 during SRS onsite burns only (n=18); no radionuclide analysis results for ^{241}Am, ^{242}Cm and ^{244}Cm on 2nd filter collected on 03/04/2003 (n=11 for respective radionuclides for SRS onsite nonburns)

[b] Conc (concentration) are field-blank adjusted

[c] Prescribed burn was around H-Area seepage basin cap at SRS

[d] Prescribed burn was for SRS compartment 30 piles

[e] Number denotes Savannah River Site compartments – for example SRS 39 refers to SRS compartment 39

[f] Francis Marion National Forest (sampled from plots 1 and 6)

[g] Fort Benning (sampled from burn units F4a and F4b)

[h] Fort Gordon (sampled from burn units 24 and 25)

[i] Oconee National Forest (sampled from burn units 101 /105 and 140)

[j] Piedmont National Wildlife Refuge; (sampled from visitor centers (v) 1 and 2)

[k] Sample taken behind USDA Forest Service-Savannah River (USFS-SR) facility complex

[l] Sample taken behind USFS-SR education complex

Table 3.2 Wilcoxon mean scores and medians per site by radionuclide using a one way non parametric model

Radionuclide	SRS		CSRS		Offsite		X^2	*p*-value
	Mean Score	Median pCi/m^3	Mean Score	Median pCi/m^3	Mean Score	Median pCi/m^3		
^{241}Am	27.32	-3.94×10^{-5}	24.45	-1.15×10^{-5}	34.20	2.59×10^{-6}	2.10	0.3502
^{242}Cm	9.38	-3.99×10^{-3}	12.40	0.00×10^{0}	13.90	0.00×10^{0}	2.06	0.3575
^{244}Cm	28.62	-2.17×10^{-5}	19.73	-4.59×10^{-6}	35.00	0.00×10^{0}	4.90	0.0864
^{60}Co	26.56	-9.06×10^{-3}	35.08	4.76×10^{-4}	27.20	-2.20×10^{-4}	2.50	0.2865
^{137}Cs	27.32	-4.34×10^{-3}	29.33	-6.65×10^{-4}	31.50	-7.39×10^{-4}	0.55	0.7609
^{40}K	26.53	-1.79×10^{-1}	28.67	1.71×10^{-2}	35.00	2.27×10^{-2}	2.09	0.3524
^{238}Pu	35.16	8.41×10^{-4}	17.54	1.48×10^{-4}	19.00	1.46×10^{-4}	14.48	**0.0007**
$^{239,\,240}$Pu	32.74	6.72×10^{-5}	17.29	1.90×10^{-6}	27.55	2.78×10^{-6}	8.00	**0.0183**
^{228}Th	34.15	7.99×10^{-4}	22.08	3.48×10^{-8}	17.00	-1.67×10^{-5}	10.91	**0.0043**
^{230}Th	33.09	1.02×10^{-3}	22.83	-3.09×10^{-4}	19.70	-2.71×10^{-4}	7.05	**0.0294**
^{232}Th	32.71	1.69×10^{-4}	22.50	-4.88×10^{-5}	21.40	-2.80×10^{-6}	5.78	0.0556
^{234}U	34.18	1.21×10^{-3}	26.25	1.77×10^{-5}	11.90	2.60×10^{-5}	14.71	**0.0006**
^{235}U	33.71	2.84×10^{-5}	16.08	-1.48×10^{-5}	25.70	-1.36×10^{-5}	10.71	**0.0047**
^{238}U	34.35	8.12×10^{-4}	17.92	1.51×10^{-5}	21.30	3.87×10^{-6}	11.38	**0.0034**
Gr. Beta	34.32	2.56×10^{-1}	30.08	2.74×10^{-2}	6.80	2.24×10^{-2}	22.15	**<0.0001[a]**
Gr. Alpha	29.29	2.06×10^{-2}	29.75	1.61×10^{-2}	24.30	5.14×10^{-3}	0.81	0.6656

Wilcoxon scores (rank sums) and medians are reported for each radionuclide by site. SRS, Offsite and CSRS refer to burns at SRS, offsite locations and nonburn days at SRS respectively.

Sample sizes for SRS, CSRS and Offsite locations are 34, 12 and 10 respectively, except for ^{241}Am and ^{244}Cm where n=11 (for ^{242}Cm n=8, 5 and 10 respectively for the three burn sample populations).

All Chi square tests degree of freedom=2. LOD refers to limit of detection.

Gr Alpha and Beta refer to gross alpha and beta respectively. [a] Highly statistically significant.

Table 3.3 Parameter estimates of the variogram model used in the ordinary kriging

Parameter	**Radionuclide**		
	^{40}K	**^{137}Cs**	**^{238}Pu**
Range (km)	7.34×10^{0}	2.59×10^{-1}	2.03×10^{0}
Nugget (pCi/m^3)^2	5.50×10^{-1}	3.28×10^{-3}	4.50×10^{-5}
Partial Sill (pCi/m^3)^2	2.68×10^{0}	1.43×10^{-4}	4.11×10^{-6}
Mean (pCi/m^3)	5.10×10^{-1}	-1.21×10^{-2}	2.88×10^{-3}
SE	7.80×10^{-1}	1.23×10^{-2}	1.30×10^{-3}
df	2	2	2
***X*^{2}**	15.48	2.15	0.03
***p*-value**	**0.0004**	0.3419	0.9852

Range represents the distance (km) beyond which there is no spatial correlation; Nugget gives the microscale variation (pCi/m^3)^2 and is a measurement of effect; and Partial sill (pCi/m^3)^2 is the difference between the nugget and the sill (the overall variability of the data). SE is the standard error, X^2 is the chi square test statistic, df is degree of freedom for the X^2 value.
P-value with bold face implies statistical significance.

Supplementary Table 3.1. Descriptive statistics on radiological activities during burn days at SRS, Offsite locations and during nonburn days at SRS

SITE: SRS **Radionuclide**	**Number of samples[a] (% above background)**	**Mean Concentration ± SD (pCi/mg)**	**95% Confidence Interval (pCi/mg)**	**Range (pCi/mg)**
Gr. Alpha	32 (66%)	$3.99 \times 10^{-1} \pm 4.03 \times 10^{-3}$	$(3.97 \times 10^{-1}, 4.00 \times 10^{-1})$	$(-9.53 \times 10^{-3}, 8.68 \times 10^{-3})$
Gr. Beta	32 (100%)	$3.09 \times 10^{0} \pm 9.43 \times 10^{0}$	$(-8.47 \times 10^{-2}, 6.25 \times 10^{0})$	$(4.74 \times 10^{-2}, 5.22 \times 10^{1})$
^{241}Am	32 (47%)	$5.15 \times 10^{-4} \pm 4.97 \times 10^{-3}$	$(-1.16 \times 10^{-3}, 2.19 \times 10^{-3})$	$(-3.18 \times 10^{-3}, 2.73 \times 10^{-2})$
^{242}Cm	6 (50%)	$-2.12 \times 10^{-2} \pm 3.46 \times 10^{-2}$	$(-4.52 \times 10^{-2}, 2.80 \times 10^{-3})$	$(-6.89 \times 10^{-1}, 1.34 \times 10^{-2})$
^{244}Cm	32 (44%)	$5.39 \times 10^{-4} \pm 2.66 \times 10^{-3}$	$(-3.56 \times 10^{-4}, 1.43 \times 10^{-3})$	$(-1.38 \times 10^{-3}, 1.41 \times 10^{-2})$
^{60}Co	32 (47%)	$-1.69 \times 10^{-2} \pm 4.50 \times 10^{-1}$	$(-1.68 \times 10^{-1}, 1.35 \times 10^{-1})$	$(-2.18 \times 10^{0}, 9.84 \times 10^{-1})$
^{137}Cs	32 (41%)	$-5.33 \times 10^{-2} \pm 2.03 \times 10^{-1}$	$(-1.22 \times 10^{-1}, 1.50 \times 10^{-2})$	$(-1.03\text{E} \times 10^{0}, 2.19 \times 10^{-1})$
^{40}K	32 (34%)	$6.46 \times 10^{-1} \pm 1.43 \times 10^{-1}$	$(-4.17 \times 10^{0}, 5.46 \times 10^{0})$	$(-3.11 \times 10^{1}, 7.22 \times 10^{1})$
^{238}Pu	32(100%)	$2.31 \times 10^{-2} \pm 7.35 \times 10^{-2}$	$(-1.66 \times 10^{-3}, 4.78 \times 10^{-2})$	$(0.00 \times 10^{0}, 3.66 \times 10^{-1})$
$^{239, 240}$Pu	32 (66%)	$3.14 \times 10^{-3} \pm 1.53 \times 10^{-2}$	$(-2.01 \times 10^{-3}, 8.30 \times 10^{-3})$	$(-2.36 \times 10^{-4}, 8.70 \times 10^{-2})$
$^{89, 90}$Sr	16 (72%)	$1.04 \times 10^{-1} \pm 3.77 \times 10^{-1}$	$(-7.02 \times 10^{-2}, 2.78 \times 10^{-1})$	$(-2.94 \times 10^{-2}, 1.61 \times 10^{0})$
^{228}Th	32 (81%)	$5.91 \times 10^{-2} \pm 2.30 \times 10^{-1}$	$(-1.83 \times 10^{-2}, 1.37 \times 10^{-1})$	$(-1.21 \times 10^{-2}, 1.26 \times 10^{0})$
^{230}Th	32 (78%)	$1.13 \times 10^{-2} \pm 4.67 \times 10^{-2}$	$(-4.42 \times 10^{-3}, 2.69 \times 10^{-2})$	$(-4.39 \times 10^{-2}, 2.34 \times 10^{-1})$
^{232}Th	32 (78%)	$2.03 \times 10^{-3} \pm 2.89 \times 10^{-3}$	$(1.06 \times 10^{-3}, 3.00 \times 10^{-3})$	$(-1.09 \times 10^{-2}, 8.68 \times 10^{-3})$
^{234}U	32 (94%)	$1.76 \times 10^{-2} \pm 6.28 \times 10^{-2}$	$(-3.46 \times 10^{-3}, 3.87 \times 10^{-2})$	$(-1.69 \times 10^{-3}, 3.50\text{E} \times 10^{-1})$
^{235}U	32 (69%)	$1.38 \times 10^{-3} \pm 6.88 \times 10^{-3}$	$(-9.31 \times 10^{-4}, 3.70 \times 10^{-3})$	$(-2.03\text{E} \times 10^{-3}, 3.87 \times 10^{-2})$
^{238}U	32 (78%)	$1.45 \times 10^{-2} \pm 4.09 \times 10^{-2}$	$(-2.02 \times 10^{-3}, 3.09 \times 10^{-2})$	$(-1.52 \times 10\text{-}^{3}, 2.70 \times 10^{-1})$

SITE: OFFSITE **Radionuclide**	**Number of samples[b]** **(% above background)**	**Mean Concentration ± SD (pCi/mg)**	**95% Confidence Interval (pCi/mg)**	**Range (pCi/mg)**
Gr. Alpha	9 (78%)	$2.30 \times 10^{-2} \pm 3.49 \times 10^{-2}$	$(1.59 \times 10^{-4}, 4.58 \times 10^{-2})$	$(-2.80 \times 10^{-2}, 6.84E \times 10^{-2})$
Gr. Beta	9 (100%)	$5.28 \times 10^{-1} \pm 7.46 \times 10^{-1}$	$(4.01 \times 10^{-2}, 1.01 \times 10^{0})$	$(4.91 \times 10^{-2}, 2.46 \times 10^{0})$
^{241}Am	9 (22%)	$-8.14 \times 10^{-5} \pm 1.27 \times 10^{-4}$	$(-1.64 \times 10^{-4}, 1.29 \times 10^{-6})$	$(-3.81 \times 10^{-4}, 7.07 \times 10^{-3})$
^{242}Cm	-	-	-	-
^{244}Cm	9 (22%)	$5.23 \times 10^{-3} \pm 1.69 \times 10^{-2}$	$(-5.80 \times 10^{-3}, 1.63 \times 10^{-2})$	$(-2.10 \times 10^{-4}, 5.02 \times 10^{-2})$
^{60}Co	9 (67%)	$3.20 \times 10^{-2} \pm 7.09 \times 10^{-2}$	$(-1.43 \times 10^{-2}, 7.83 \times 10^{-2})$	$(-8.57 \times 10^{-3}, 2.18 \times 10^{-1})$
^{137}Cs	9 (44%)	$-1.57 \times 10^{-2} \pm 4.48 \times 10^{-2}$	$(-4.49 \times 10^{-2}, 1.36 \times 10^{-2})$	$(-1.27 \times 10^{-1}, 3.22 \times 10^{-2})$
^{40}K	9 (11%)	$-4.38 \times 10^{-1} \pm 6.20 \times 10^{-1}$	$(-8.43 \times 10^{-1}, -3.26 \times 10^{-1})$	$(-1.99 \times 10^{0}, 8.42 \times 10^{-2})$
^{238}Pu	9 (100%)	$5.27 \times 10^{-4} \pm 8.91 \times 10^{-4}$	$(-5.57 \times 10^{-5}, 1.11 \times 10^{-3})$	$(0.00 \times 10^{0}, 2.66 \times 10^{-3})$
$^{239, 240}$Pu	9 (11%)	$-8.14 \times 10^{-5} \pm 1.27 \times 10^{-4}$	$(-1.64 \times 10^{-4}, 1.29 \times 10^{-6})$	$(-3.81 \times 10^{-4}, 7.07 \times 10^{-7})$
^{228}Th	9 (67%)	$1.10 \times 10^{-3} \pm 2.06 \times 10^{-3}$	$(-2.41 \times 10^{-4}, 2.45 \times 10^{-3})$	$(-9.54 \times 10^{-5}, 6.35 \times 10^{-3})$
^{230}Th	9 (67%)	$3.28 \times 10^{-4} \pm 2.13 \times 10^{-3}$	$(-1.07 \times 10^{-3}, 1.72 \times 10^{-3})$	$(-4.76 \times 10^{-3}, 2.34 \times 10^{-3})$
^{232}Th	9 (56%)	$1.30 \times 10^{-5} \pm 5.30 \times 10^{-4}$	$(-3.33 \times 10^{-4}, 3.59 \times 10^{-4})$	$(-9.13 \times 10^{-4}, 1.08 \times 10^{-3})$
^{234}U	9 (100%)	$1.44 \times 10^{-3} \pm 1.25 \times 10^{-3}$	$(6.28 \times 10^{-4}, 2.26 \times 10^{-3})$	$(4.89 \times 10^{-5}, 4.19 \times 10^{-3})$
^{235}U	9 (11%)	$-1.82 \times 10^{-4} \pm 2.20 \times 10^{-4}$	$(-3.25 \times 10^{-4}, -3.84 \times 10^{-5})$	$(-6.80 \times 10^{-4}, 1.48 \times 10^{-5})$
^{238}U	9 (56%))	$-2.07 \times 10^{-4} \pm 8.46 \times 10^{-4}$	$(-7.60 \times 10^{-4}, 3.45 \times 10^{-4})$	$(-2.27 \times 10^{-3}, 8.12 \times 10^{-4})$

SITE: CSRS **Radionuclide**	**Number of samples** **(% above background)**	**Mean Concentration ± SD (pCi/mg)**	**95% Confidence Interval (pCi/mg)**	**Range (pCi/mg)**
Gr. Alpha	12 (100%)	$5.62 \times 10^{0} \pm 1.19 \times 10^{1}$	$(-1.14 \times 10^{0}, 1.24 \times 10^{1})$	$(7.99 \times 10^{-2}, 3.17 \times 10^{1})$
Gr. Beta	12 (100%)	$1.17 \times 10^{1} \pm 2.46 \times 10^{1}$	$(-2.18 \times 10^{0}, 2.57 \times 10^{1})$	$(2.14 \times 10^{-1}, 7.51 \times 10^{1})$
^{241}Am	11 (55%)	$-3.47 \times 10^{-3} \pm 1.05 \times 10^{-2}$	$(-9.66 \times 10^{-3}, 2.71 \times 10^{-3})$	$(-3.49 \times 10^{-2}, 1.81 \times 10^{-3})$
^{242}Cm	11 (55%)	$-9.40 \times 10^{-3} \pm 7.41 \times 10^{-2}$	$(-5.32 \times 10^{-2}, 3.44 \times 10^{-2})$	$(-2.25 \times 10^{-1}, 4.69 \times 10^{-2})$
^{244}Cm	11 (55%)	$-1.44 \times 10^{-3} \pm 4.17 \times 10^{-3}$	$(-3.90 \times 10^{-3}, 1.03 \times 10^{-3})$	$(-1.40 \times 10^{-2}, 1.23 \times 10^{-4})$
^{60}Co	12 (50%)	$-1.38 \times 10^{-2} \pm 7.42 \times 10^{-1}$	$(-4.34 \times 10^{-1}, 4.06 \times 10^{-1})$	$(-2.06 \times 10^{0}, 1.29 \times 10^{0})$
^{137}Cs	12 (33%)	$-2.69 \times 10^{-1} \pm 1.64 \times 10^{0}$	$(-1.19 \times 10^{0}, 6.57 \times 10^{-1})$	$(-4.89 \times 10^{0}, 2.49 \times 10^{0})$
^{40}K	12 (50%)	$1.38 \times 10^{1} \pm 3.44 \times 10^{1}$	$(-5.67 \times 10^{0}, 3.33 \times 10^{1})$	$(-5.70 \times 10^{0}, 1.06 \times 10^{2})$
^{238}Pu	12 (92%)	$1.83 \times 10^{-2} \pm 3.83 \times 10^{-2}$	$(-3.34 \times 10^{-3}, 4.00 \times 10^{-2})$	$(-9.18 \times 10^{-3}, 1.36 \times 10^{-1})$
$^{239, 240}$Pu	12 (75%)	$1.60 \times 10^{-3} \pm 5.25 \times 10^{-3}$	$(-1.38 \times 10^{-3}, 4.57 \times 10^{-3})$	$(-1.28 \times 10^{-3}, 1.82 \times 10^{-2})$
^{228}Th	12 (55%)	$2.10 \times 10^{-3} \pm 2.41 \times 10^{-2}$	$(-1.15 \times 10^{-2}, 1.57 \times 10^{-2})$	$(-4.70 \times 10^{-2}, 6.15 \times 10^{-2})$
^{230}Th	12 (25%)	$-8.60 \times 10^{-2} \pm 2.29 \times 10^{-1}$	$(-2.16 \times 10^{-1}, 4.39 \times 10^{-2})$	$(-8.09 \times 10^{-1}, 8.04 \times 10^{-3})$
^{232}Th	12 (50%)	$-1.41 \times 10^{-2} \pm 6.07 \times 10^{-2}$	$(-4.84 \times 10^{-2}, 2.02 \times 10^{-2})$	$(-1.98 \times 10^{-1}, 5.74 \times 10^{-2})$
^{234}U	12 (58%)	$2.39 \times 10^{-2} \pm 9.85 \times 10^{-2}$	$(-3.18 \times 10^{-2}, 7.97 \times 10^{-2})$	$(-5.65 \times 10^{-2}, 3.32 \times 10^{-1})$
^{235}U	12 (0%)	$-5.26 \times 10^{-3} \pm 1.23 \times 10^{-2}$	$(-1.22 \times 10^{-2}, 1.70 \times 10^{-3})$	$(-4.16 \times 10^{-2}, -2.69 \times 10^{-7})$
^{238}U	12 (50%)	$-3.43 \times 10^{-3} \pm 4.40 \times 10^{-2}$	$(-2.83 \times 10^{-2}, 2.15 \times 10^{-2})$	$(-1.26 \times 10^{-1}, 1.73 \times 10^{-1})$

SRS, Offsite and CSRS refer to burns at SRS, offsite locations and nonburn days at SRS respectively. Gr. Alpha and Beta refer to gross alpha and beta respectively.

[a] Two samples in SRS burn days data had masses that were <LOD (limit of detection)

[b] Sample from Francis Marion National Forest mass not obtained, just estimated. ^{242}Cm was not analyzed.

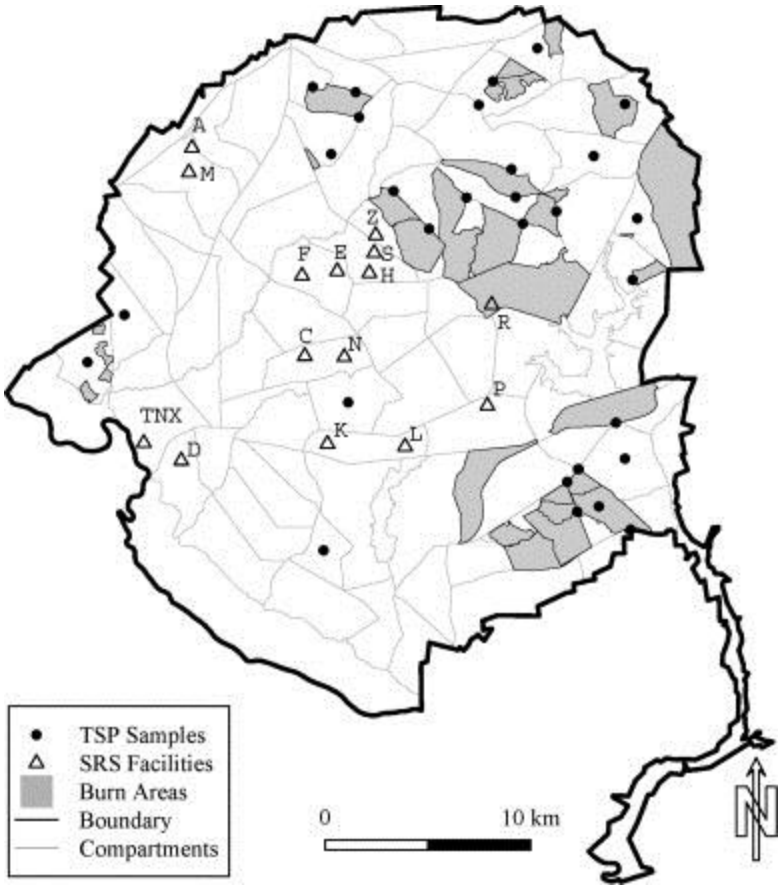

Fig. 3.1. Air sampling locations within SRS compartments over the four-year study period. TSP samples refer to locations where total suspended particulates were collected. SRS facilities refer to areas that have released and/or had the potential to release radionuclides (ATSDR, 2007; Carlton, 1999) and thus are identified as major nuclear production facilities and reactors at SRS (ATSDR, 2007), hence:▪ C, K, L, P and R are identified as nuclear reactors, all of which are permanently shut down (ATSDR, 2007);▪ P-area also houses a coal-fired power plant along with coal storage and disposal piles (US EPA, 2011).▪ F and H as nuclear processing and separation areas;▪ A and M as fuel fabrication areas;▪ A-area also consists of coal storage and disposal piles (US EPA, 2011)▪ E, S and Z as general separation and waste management areas;▪ N as a storage of construction materials area;▪ D as a heavy water reprocessing area, also houses a coal-fired power plant since its construction in 1952 (ATSDR, 2007; US EPA, 2011), and;▪ TNX as a testing equipment and plutonium and uranium recovery area.Burn areas are locations where prescribed burn occurred during this four-year study. Boundary refers to the SRS boundary line. Compartments refer forest management partitions across SRS.

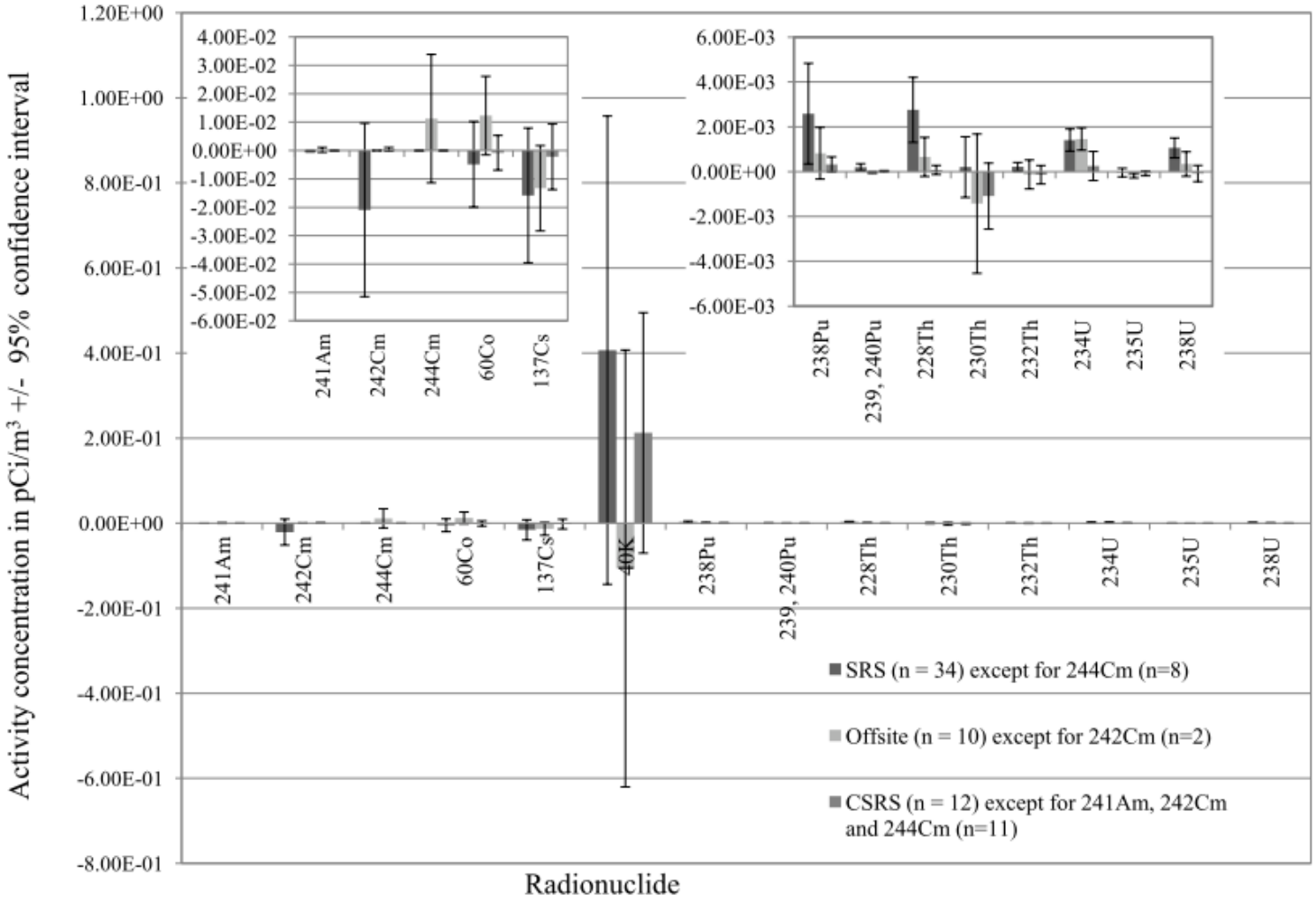

Fig. 3.2. Mean radionuclide concentrations of the three burn sample populations. SRS, offsite and CSRS refer to burns at SRS, offsite locations and nonburn days at SRS respectively. ^{40}K has the highest activity concentration at SRS during prescribed burns compared to all other measured radionuclides. The figure also shows the high background levels at SRS when there are no burns.

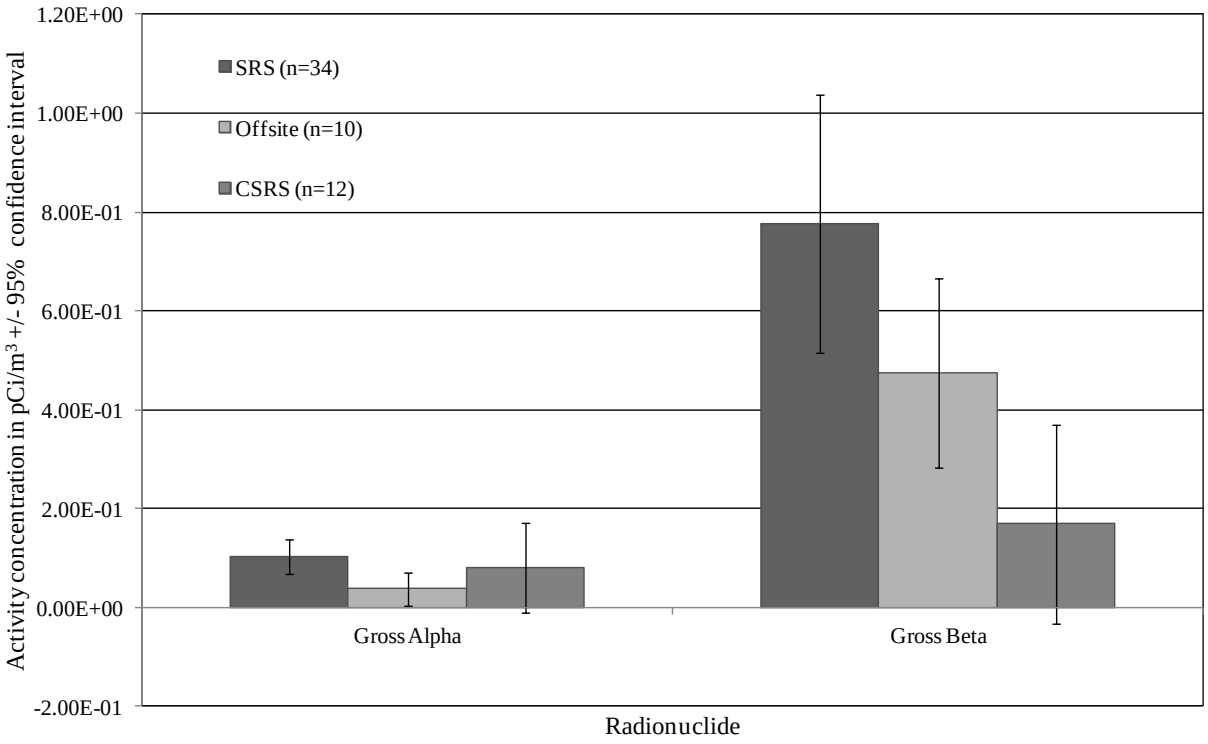

Fig. 3.3 Mean gross alpha and beta concentrations of the three burn sample populations. SRS, offsite and CSRS refer to burns at SRS, offsite locations and nonburn days at SRS respectively. Mean gross alpha activities do not differ when there is a prescribed burn at SRS (SRS vs. CSRS) or when offsite measurements are compared to SRS levels (SRS vs. Offsite). Gross beta activities with high airborne concentrations during nonburns are enhanced during burns at SRS.

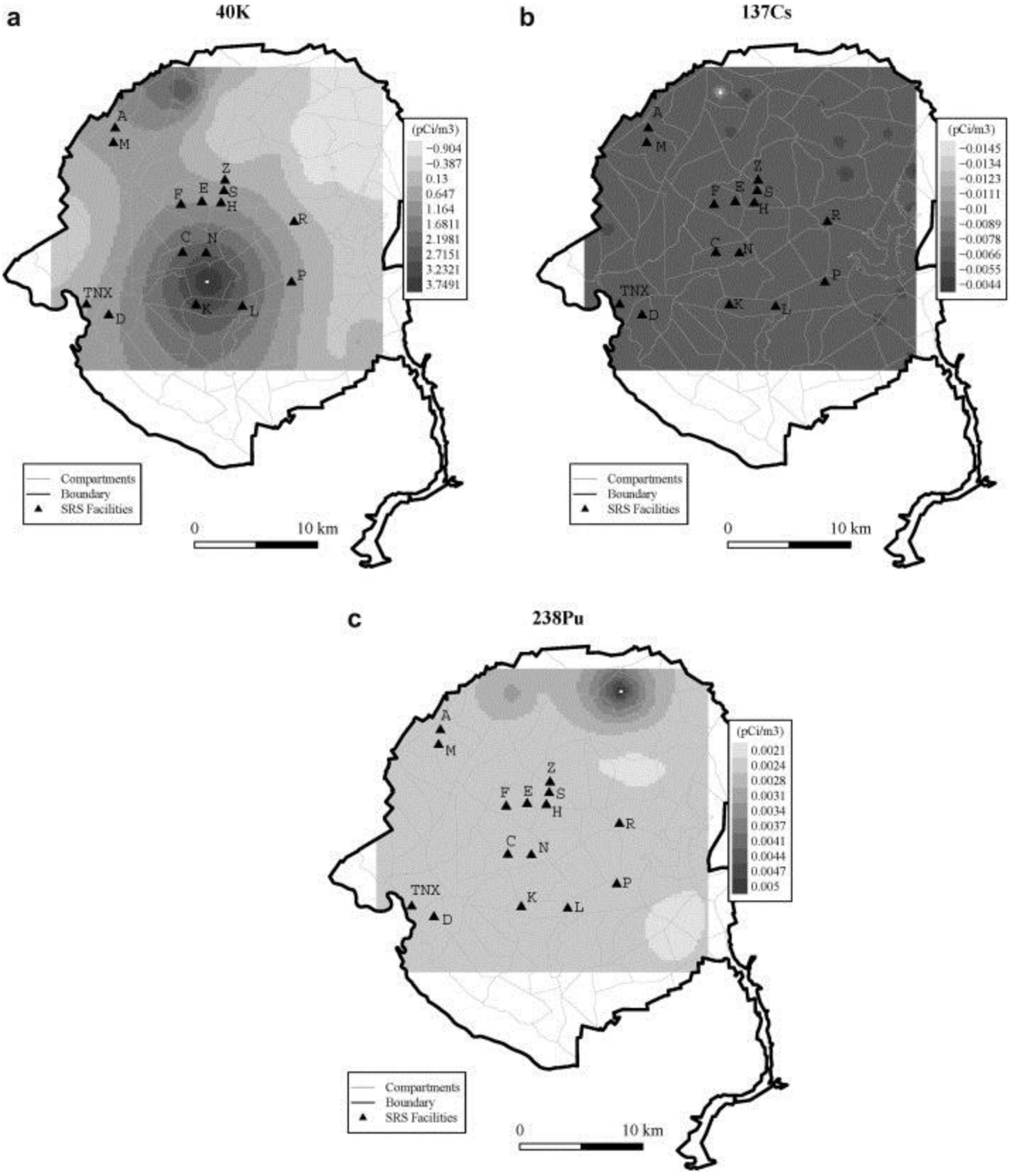
a
40K
(pCi/m3)
−0.904
−0.387
0.13
0.647
1.164
1.6811
2.1981
2.7151
3.2321
3.7491
A
M
Z
F
E
S
H
R
C
N
P
TNX
D
K
L
Compartments
Boundary
SRS Facilities
0
10 km
b
137Cs
(pCi/m3)
−0.0145
−0.0134
−0.0123
−0.0111
−0.01
−0.0089
−0.0078
−0.0066
−0.0055
−0.0044
c
238Pu
(pCi/m3)
0.0021
0.0024
0.0028
0.0031
0.0034
0.0037
0.0041
0.0044
0.0047
0.005

Fig. 3.4. a. Continuous surface map for ^{40}K during SRS prescribed burns over the four sampling years. A strong spatial correlation exists ($p = 0.0004$, $n = 34$) for ^{40}K airborne levels across burn sample areas. There did not appear to be a direct link between areas with high activity concentrations and SRS facilities. Compartments refer forest management partitions across SRS. SRS border refers to the SRS boundary line. SRS facilities refer to the 15 plotted SRS facilities (A, C, D, E, F, H, K, L, M, N, P, R, S, TNX, and Z) are defined in Fig. 3.1 b. Continuous surface map for ^{137}Cs during SRS prescribed burns over the four sampling years. No significant spatial correlation existed for measured airborne ^{137}Cs across burn sample areas ($p = 0.3419$, $n = 34$). There were low activity concentrations of ^{137}Cs in air samples across SRS burn areas and there did not appear to be a direct link between areas with high activity concentrations and SRS facilities. Compartments refer forest management partitions across SRS. SRS border refers to the SRS boundary line. SRS facilities refer to the 15 plotted SRS facilities (A, C, D, E, F, H, K, L, M, N, P, R, S, TNX, and Z) are defined in Fig. 3.1. c. Continuous surface map for ^{238}Pu during SRS prescribed burns over the four sampling years. No significant spatial correlation existed for measured airborne ^{238}Pu across burn sample areas ($p = 0.9852$, $n = 34$). Akin to ^{137}Cs, there did not appear to be a direct link between those areas with higher activity concentrations (toward the northeast) and SRS facilities. Compartments refer forest management partitions across SRS. SRS border refers to the SRS boundary line. SRS facilities refer to the 15 plotted SRS facilities (A, C, D, E, F, H, K, L, M, N, P, R, S, TNX, and Z) are defined in Fig. 3.1.

CHAPTER 4

OVERVIEW ON HOUSEHOLD AIR POLLUTION

HOUSEHOLD AIR POLLUTION: A MAJOR PUBLIC HEALTH HAZARD

Nearly half of the world's population burns biomass, mostly as fuel for cooking (Martin et al. 2011; Naeher et al. 2007; Smith 1987) resulting in household air pollution (HAP). Women and young children bear the brunt of high HAP exposures due to long hours spent in close proximity to cooking fires (Ezzati and Kammen 2002). Household stoves typically used for cooking and heating in the developing world do not burn fuel cleanly leading to incomplete combustion in the domestic environment (Perez-Padilla et al. 2010; Smith 1987). Smoke from incomplete biomass combustion contains health-damaging pollutants (Bølling et al. 2009; Jalava et al. 2010; Naeher et al. 2007), of which carbon monoxide (CO) and particulate matter with an aerodynamic diameter of ≤2.5 microns ($PM_{2.5}$) are major constituents and are considered chief inhalation hazards (Naeher et al. 2007). The efficiency of the three-stone open fire used in many developing countries is only about 10-15% however, thus most of the energy content of the fuel is wasted (Ballard-Tremeer and Jawurek 1996; Y von Schirnding 2002). Studies from around the world have demonstrated that HAP levels from combustion of biofuels are extremely high – often many times the standards in industrialized countries (Y von Schirnding 2002).

HAP levels measured in kitchens in the developing world often exceed WHO air quality guidelines (24hr mean: 25 $\mu g/m^3$) by 10 to 20 times (Bruce 2010; Y von Schirnding 2002) with measurements of $PM_{2.5}$ from studies in developing countries in the range of 97 to 3500 $\mu g/m^3$ (Naeher et al. 2007). Results from the first randomized control trial on the impacts of chimney stoves on health (Randomized Exposure Study of Pollution Indoors and Respiratory Effects:

RESPIRE) indicate that in Guatemalan households using traditional open fire stoves, 48hr kitchen $PM_{2.5}$ measurements ranged from 500 – 1000 $\mu g/m^3$; post intervention stove installation achieved a mean of 125 $\mu g/m^3$ in those same households (Bruce 2010). HAP measurements using passive diffusion CO tubes from RESPIRE showed 90%, 61% and 52% reduction in CO levels in the kitchen (n=65), for mothers (n=529) and children (n=270) respectively after chimney stove installation (Smith et al. 2010).

Recent studies have focused on estimating $PM_{2.5}$ exposures given that $PM_{2.5}$ has been identified as the best single indicator of the health effects of combustion of biomass such as wood (Naeher et al. 2007; Perez-Padilla et al. 2010). In India a range of 500 to 2000 $\mu g/m^3$ has been recorded for $PM_{2.5}$ during cooking (Balakrishnan, Ramaswamy, and Sankar 2004). HAP levels may vary depending on factors such as the time spent cooking, fuel type and cooking environment (Armendáriz-Arnez et al. 2010; Dutta et al. 2007; Romieu et al. 2009) Concentrations of CO and PM also vary over short (less than a day) time periods (McCracken and Smith 1998). Two of these factors which can exacerbate HAP exposures are discussed below in an effort to highlight the complexity of these exposures.

Undoubtedly, the quality of stove employed plays a significant role in the extent of HAP exposure. In Honduras, a four-level subjective scale revealed 8hr (geometric mean) personal $PM_{2.5}$ exposures of 60 ug/m^3, 71 ug/m^3, 108 ug/m^3 and 202 ug/m^3 for high quality, high mid quality, low-mid quality and low quality stoves respectively (Clark et al. 2010). In San Marcos, Peru on the other hand, chimney stoves that had been in use for about 7 month resulted in mean $PM_{2.5}$ personal exposures and kitchen concentrations of 104 and 148$\mu g/m^3$ respectively (Hartinger in review). Approximately 35% of these improved stoves in use in San Marcos were in need of repair at the time of air quality monitoring.

The duration of stove use is also essential in this discussion. In Santiago de Chuco, Peru subjects using two types of chimney stoves installed three weeks prior to air quality monitoring, had 48hr $PM_{2.5}$ exposures of 68.4 and 58.3 ug/m^3 (Fitzgerald et al. 2012a). In Mexico, the use of an improved chimney stove together with a traditional open fire resulted in median real time personal 24hr $PM_{2.5}$ exposures of 190 µg/m^3 and 48hr kitchen median concentration of 300 µg/m^3 (Armendáriz-Arnez et al. 2010). These stoves had been in use for approximately 10 months.

In many instances, the $PM_{2.5}$ exposures are higher than 24hr air quality limits for $PM_{2.5}$ set or recommended by regulatory and international agencies. These standards for PM may also be applied to the indoor environment, specifically in the developing world, where large populations are exposed to high levels of combustion particles derived from indoor cookstoves (Martin et al. 2011). When you consider the potential for cumulative exposures and decrease in recovery time, it becomes evident that meeting these limits for a 24hr period can alleviate substantial excess morbidity and mortality resulting from HAP exposures (Y von Schirnding 2002).

GLOBAL STOVE INTERVENTION STUDIES

A variety of cost effective interventions can be implemented in order to address the problem of HAP. First and foremost the type of stove employed can be changed by improving the technology and or design. Then the fuel type can be changed to cleaner burning fuels. The cooking environment can also be improved to better circulate the pollutants emitted during cooking activities. Families can be advised to build more windows for increased ventilation, eaves can be added to the construction of kitchens and doors can be opened during cooking. Finally the primary household cooks can modify their behaviors to avoid exposure during peak

cooking times and lastly there can also be a rotation of the primary household cooks, if possible, to reduce cumulative exposures. Of all the above mentioned interventions, improved cookstoves have become a widespread intervention. Highlights of intervention stove programs around the world are briefly outlined below.

By far, the Chinese National Improved Stove Project (CNISP) is the pioneer example of cookstove distribution at a large scale. The program introduced 129 million cookstoves to rural areas between 1982 and 1992 (Smith et al. 2000). This program has not only been largest, but also has been the most cost effective (15% of total stove program cost) (Smith et al. 2000). However, the program was not without its faults. Even with a reduction of particulate matter and CO by 43 and 62% respectively (Edwards et al. 2007), the reductions were still high compared to Chinese health standards. With current increasing economic growth in China and the prevalence of coal use, air pollution, particularly HAP still needs to be addressed (Zhang and Smith 2007).

In India, there have been many improved cookstove (ICS) units with many designs installed, however, there have also been limited air quality monitoring to correct and improve stove designs (Venkataraman et al. 2010). For example the evaluation of an ICS revealed that 75% were functional when installed by trained persons while 25% were in various states of disrepair (Aggarwal and Chandel 2004). Lessons learned from the India ICS programs include creating awareness on the harmful effects of smoke from cookstoves, the need for fuel conservation the including beneficiaries in the design of new stoves and constant training to ensure proper stove use techniques. An important factor undergirding all these lessons is the impact of geography and climate, where even in the same province in India, different types of stoves have to be designed to meet cooking, space heating and lighting needs (Aggarwal and Chandel 2004).

In Bangladesh, improved earthen stoves were provided to beneficiaries in an effort to decrease the use of biomass energy. Questionnaires administered to the households involved revealed that a third of the households saved 1.5 kg/day of fuel, 20% of the households cooked 45 min/day less than when they used traditional stoves (Alam et al. 2006; Nazmul Alam and Chowdhury 2010). Subjects also reported an alleviation of health symptoms such as eye irritation and eye itching during ICS use (Alam et al. 2006). With the improved earthen stove an upgraded version of the traditional stoves in Bangladesh, the ICS proved to be cost saving and designed to meet the needs of the users.

The Honduras Stove Project is another important program when considering global ICS. The project aimed to document the health effects and the economic benefits after installing *Justa* stoves (Henkle et al. 2010). The use of improved stoves was associated with 63% and 73% lower personal and indoor $PM_{2.5}$ exposures respectively (Clark et al. 2009). This project was able to demonstrate the practicability of a low-cost stove capable of being constructed using local materials, a model which can be producible in other developing countries (Henkle et al. 2010). It also points to the need for improved stove technologies.

During the 1980s and 1990s, ICS were introduced in Sudan to increase the efficiency of using energy from biomass sources. A study based on primary data collected through personal interviews in 300 randomly selected households was conducted in Khartoum State (El Tayeb Muneer and Mukhtar Mohamed 2003). Results indicated low ICS adoption (17.6%). When the primary household cook was exposed to information regarding improved stoves, and either the primary cook or members of her household were educated, there was a tendency to use the improved cookstove. This lesson from the African continent highlights that fact that failure to

fully understand the social setting in the developing world context can impede both the development and adopting of improved stoves (El Tayeb Muneer and Mukhtar Mohamed 2003).

In Kenya, smoke hoods introduced in rural households reduced particulate levels from 4383 ug/m^3 to a mean of 1075 ug/m^3. Users of *Upesi* stoves also experienced little improvement in HAP but benefited from reduced fuel use, shorter cooking time, increased safety and ease of use (Bates et al. 2002). These projects also point to the importance of stove installment and stove use training, as well as women empowerment in the local communities.

In Ghana, after wood-burning rocket stoves, the Gyapa, were evaluated in Accra, average 24-hr $PM_{2.5}$ concentrations decreased from 650 μg/m^3 to 320 μg/m^3 and average 24-hr kitchen CO concentrations decreased from 12.3 ppm to 7.4 ppm (Pennise et al. 2009). An important point from this project is the need to evaluate changes in stove performance over time. This will help determine whether the achieved HAP reductions are sustained over time.

In Mexico, the use of Patsari stoves have resulted in over 60% reductions in particulate matter and CO concentrations (Masera et al. 2007) as well as over 30% reduction in personal exposures (Cynthia et al. 2008). With a 50% adoption rate after 10 months of use (Romieu et al. 2009), lessons from the introduction of the Patsari stoves in Mexico include the importance of air quality monitoring and the potential impact of biomass smoke on climate change (Masera et al. 2007).

In Pakistan women who used the ICS, called Smoke Free Stoves, reported a reduction in the incidence of adverse respiratory and health symptoms during cooking compared to women who used traditional stoves (Khushk et al. 2005). The importance of long term monitoring and evaluation, in order to assess the impact of long term ICS use is evident from this program. On the other hand, in Thailand, a pattern of energy consumption in households was investigated in

order to develop strategies to implement the use of ICS and biogas digesters (Limmeechokchai and Chawana 2007). Results revealed three important barriers in the adoption of the ICS that need to be considered in future ICS programs: (1) high investment cost (2) lack of information, and (3) lack of financial sources (Limmeechokchai and Chawana 2007).

In Peru, three communities in the Santiago de Chuco province received improved chimney cookstoves from the Juntos National Program (stove 1) and the Barrick Gold Corporation's Community Relations Department (stove 2). There were exposure reductions of over 40% in personal and kitchen $PM_{2.5}$ after the installation of stove 1. Likewise, households which received stove 2 had over 50% reductions (Fitzgerald et al. 2012a). Also, after the installation of both improved stoves, urine samples from subjects, analyzed for urinary hydroxylate PAH metabolites (OH-PAHs) had significantly reduced OH-PAHs (Li et al. 2011). Although these stoves improved household air quality, the extent to which associated adverse health outcomes in the long term were impacted could not be determined.

To date, there has been only one completed randomized controlled trial using improved stoves with chimneys to study the impact of reduced HAP on child pneumonia (Martin et al. 2011). The RESPIRE (Randomized Exposure Study of Pollution Indoors and Respiratory Effects) study involved the dissemination of locally produced improved chimney stoves called Planchas (Smith et al. 2010). On average, the Planchas reduced CO exposure by 50% while in an exposure-response analysis, a 50% exposure reduction was significantly associated with physician-diagnosed pneumonia among children (Smith et al. 2011). Implications from these results are grave: although great strides had been made in global ICS programs, other interventions, which can result in lower exposures, may be needed among biomass using populations.

HEALTH EFFECTS OF WOODSMOKE EXPOSURE

HAP is responsible for almost 50% of the burden of diseases resulting from poor household environments (Rehfuess et al. 2006). The use of biomass fuel in the household environment is linked to respiratory and nonrespiratory illnesses and is considered a major public health threat globally (Kim, Jahan, and Kabir 2011a). Biomass smoke exposure is associated with the risk of acute lower respiratory infections (ALRI) (WHO 2011). ALRI is the chief cause of death in children in developing countries (Martin et al. 2011). In particular, the most fatal cases are in the form of pneumonia among children less than five years old (Bryce et al. 2005). There is the need for more randomized trials to better quantify the risk of ALRI. This will ensure that the degree of exposure reduction needed to significantly improve health is adequately characterized (Balakrishnan et al. 2011; Martin et al. 2011; Smith et al. 2011).

HAP has also been identified as a risk factor for chronic obstructive pulmonary disease (COPD) and chronic bronchitis (Kurmi et al. 2010). The risk, particularly for COPD, is well established among women (Dennis et al. 1996; Ekici et al. 2005; Liu et al. 2007; Orozco-Levi et al. 2006; Regalado et al. 2006). Exposure to smoke from coal fires is consistently associated with the risk of lung cancer (Hosgood et al. 2011). Results from studies on the association between household coal use and lung cancer risk present evidence that there is an elevated lung cancer risk (Hosgood et al. 2011). There is increasing evidence which suggest that HAP is also associated with factors such as low birth weight, asthma, tuberculosis, cataract, and cardiovascular disease (McCracken et al. 2011; McCracken et al. 2007; Naeher et al. 2007; Saha et al. 2005; Smith and Mehta 2003; Thompson et al. 2011).

There needs to be further investigation on these associated adverse health effects. Below is a brief discussion on emerging evidence which suggests that HAP may increase the risk of (1) an altered physiological state which is a precursor to numerous health effects, (2) adverse respiratory health and (3) cardiovascular disease.

Altered Physiological State

Evidence from population based and experimental studies point to the fact that $PM_{2.5}$ including that derived from woodsmoke has been shown to induce oxidative stress, as well as pulmonary and systemic inflammation (Ghio et al. 2011; Li, Xia, and Nel 2008; Ling and Van Eeden 2009; Naeher et al. 2007). In particular, exposure to particulate matter (PM) in woodsmoke is linked with oxidative stress in both *in-vitro* and *in-vivo* studies (Barregard et al. 2008; Barregard et al. 2006; Danielsen et al. 2008; Jalava et al. 2010; Kocbach et al. 2008a). Oxidative stress and lipid peroxidation have been associated with the development and progression of COPD (Drost et al. 2005; Kinnula et al. 2007; Ling and Van Eeden 2009). The injurious effect of PM, whether geologic or anthropogenic can occur both in the lungs and systemically (Ling and Van Eeden 2009). Reactive oxygen species and free radicals associated with PM can react with DNA and lipids to generate products including 8-OHdG and 8-isoprostane, both of which are measureable in biological fluids as markers of physiological responses due to PM exposure (Kipen et al. 2010). There is scantly available information on epidemiological studies of the effect of exposure to elevated levels of woodsmoke on oxidative stress in the natural environment in the scientific literature.

Respiratory Health

The most studied health effects for biomass smoke exposure are respiratory symptoms such as cough, phlegm production, wheeze, sore throat, and eye and nose irritation (Romieu et al. 2009). Decline in lung function is also reported in the literature upon exposure to woodsmoke (Adetona, Hall, and Naeher 2011). Women using improved cooking stoves have been reported to have less declines in lung function compared to women using over fire stoves after a one year follow up period (Romieu et al. 2009). Additionally, women who cook with biomass fuels have been reported to have higher respiratory health effects compared to women with clean burning fuels (Orozco-Levi et al. 2006).

Cardiovascular Health

Epidemiological studies have reported that exposure to fine particles promotes inflammation in the lungs (Ghio et al. 2012; Van Eeden et al. 2012), which ultimately leads to cardiovascular ill health. Circulating cytokines such as interleukin-6, interleukin-8, interleukin-1β, and Tumor Necrosis Factor-α are used as biomarkers of inflammation after exposure to woodsmoke and ambient particulates (Swiston et al. 2008; van Eeden and Sin 2008). The persistence of these markers in the body are risk factors for cardiovascular disease (Van Eeden et al. 2012).

In a recent study conducted in India (Banerjee et al. 2011), premenopausal, never-smoking women (median age, 34 years) who cook exclusively with biomass had significantly higher neutrophil count in blood and sputum compared with age-matched control women who cooked with cleaner fuel liquefied petroleum gas. Flow cytometric analysis also revealed significant increase in the surface expression of CD35 (complement receptor-1), CD16 (F C γ receptor III), and β 2 Mac-1 integrin (CD11b/CD18) on circulating neutrophils among biomass

users (Banerjee et al. 2011). Additionally enzyme-linked immunosorbent assay showed that they had 72%, 67%, and 54% higher plasma levels of the proinflammatory cytokines TNF-α, IL-6, and IL-12, respectively (Banerjee et al. 2011). This is an indication that oxidative stress plays a role in mediating neutrophilic inflammatory response following chronic inhalation of biomass smoke (Banerjee et al. 2011).

Additionally, in an air filter intervention study in a woodsmoke impacted community, air filtration was associated with improved endothelial function and decreased concentrations of inflammatory biomarkers (Allen et al. 2011). This intervention was not however associated with markers of oxidative stress. There is an implication then that a reduction in indoor particle concentrations can reduce systemic inflammation and impaired endothelial function (Allen et al. 2011).

Subclinical symptoms of cardiovascular risk can be used as measurements of cardiovascular related health risks in a population of Guatemalan women (McCracken et al. 2011; McCracken et al. 2007). Heart rate variability (HRV) and ST- segments (McCracken et al. 2011) as well as blood pressure (McCracken et al. 2007) were measured. Mean ST segment levels were approximately 0.10 mm lower among women using improved intervention stoves and nonspecific ST-segment depression was 74% lower compared to the control group. After the control group was given a stove at the end of the trial, a 72% lower rate of ST-segment depression events was observed. Similarly, the improved stove intervention was associated with 3.7 mm Hg lower SBP (95% CI: −8.1 to 0.6) and 3.0 mm Hg lower DBP (95% CI: −5.7 to −0.4) compared with controls (McCracken et al. 2007). Statistically significant differences in HRV were not observed between the intervention and control groups.

KNOWLEDGE GAPS AND RESEARCH NEEDS

There is a dearth of knowledge on HAP exposures in the developing world human woodsmoke exposures and the physiological response due to these exposures. In the advent of national cookstove programs in Peru and other countries, it is essential that stove programs be thoroughly evaluated and solutions with lasting benefits be invested in and developed. Below is a brief discussion on some key knowledge gaps and challenges in the effort to implement improved stove programs worldwide.

Current HAP exposure assessment tools such as biomarkers, personal and stationary monitors as well as questionnaires need to be improved upon to better quantify HAP. Future technologies for exposure assessment tools need to be more rugged and capable of sampling for longer durations of time to reduce variability in the data. The tools also need to be affordable, acceptable to study subjects and able to measure over a wide range of HAP pollutants and concentrations. There is also the need to develop and validate biomarkers of HAP exposure and resulting health effects.

Secondly there is the need to determine which interventions, whether stove or fuel type, are capable of reducing HAP in a given geographic location. Currently, there is only one randomized controlled trial for pneumonia which suggests exposure reductions of 90% are needed to achieve noticeable reductions in pneumonia risk. More studies are needed to support this finding, particularly in other regions of the world with different stove and fuel type combinations. Simultaneously, the association of HAP exposure and other adverse health outcomes needs investigation, as these outcomes may require lower or perhaps even higher reductions in HAP exposures.

Thirdly, stove designs and models need to be improved upon. This global solution for cooking needs to be in partnership with engineers, researchers and of course the users themselves. This will ensure stoves that are acceptable to the community, installed properly and well designed to reduce HAP exposures and reduce adverse health outcomes. Along with stove design is the need for educating the entire household to whom the stove is provided. All family members need to be trained on proper stove use, repair and maintenance to ensure sustained benefits.

Finally, since there are current stove programs underway worldwide, there needs to be evaluations to understand the health benefits or the lack thereof before global stove dissemination occurs for any particular stove type. Given that nearly half of the world's population is exposed to HAP, these studies will be useful in determining the health effects associated with biomass exposure as well as the mechanisms by which biomass constituents exert toxicity.

The success of an indoor air pollution mitigation program will depend on the number of stoves that are well designed, properly installed, adopted, continually used, and functionally maintained over time rather than on the number of disseminated stoves (Armendáriz-Arnez et al. 2010; Dutta et al. 2007; Romieu et al. 2009). Given the numerous gaps in knowledge outlined above, the following objectives were investigated in an effort to contribute to the current scientific literature on stove intervention programs:

1. To assess personal, and kitchen exposure to CO in study households in San Marcos, Cajarmarca region, Peru. With nearly half of the world's population exposed to HAP, the ability to use an inexpensive tool for measuring CO to characterize HAP exposure can be a low cost means to evaluate stove programs.

2. To assess concentrations of urinary 8-OHdG and 8-isoprostane of study subjects in order to understand the mechanism by which biomass smoke induces oxidative stress. Urinary 8-OHdG and 8-isoprostane were used as measures of systemic oxidative stress. In this study, we investigated whether HAP exposure dose-dependently increased urinary levels of 8-OHdG and 8-isoprostane and also examined the factors that are associated with oxidative stress among study subjects.
3. To investigate the relationship between CO and $PM_{2.5}$ in real time in a small sample of households. In a smaller sub sample of these households, the relationship between the two pollutants were also investigated in real time, since this real time data is rarely available for biomass smoke exposure.
4. To assess personal and kitchen exposure to CO and $PM_{2.5}$ in a sub sample of households. Another aim was to investigate whether the use of a chimney stove would result in lower urinary hydroxylated polycyclic aromatic hydrocarbons metabolites among intervention stove users when compared to control stove users in San Marcos, Peru. In this subset of homes, detailed exposure measurements was performed in order to characterize HAP exposure among this population. The relationships between these exposure measurements among this population in San Marcos were investigated in an effort to determine whether the use of study promoted chimney stoves would result in lower HAP exposures compared to the use of control stoves.

REFERENCES

Adetona, O., D. B. Hall, and L. P. Naeher. 2011. "Lung function changes in wildland firefighters working at prescribed burns." *Inhalation Toxicology* 23(13): 835-41.

Aggarwal, R. K. and S. S. Chandel. 2004. "Review of Improved Cookstoves Programme in Western Himalayan State of India." *Biomass and Bioenergy* 27(2): 131-44.

Alam, S., S. J. Chowdhury, A. Begum, and M. Rahman. 2006. "Effect of improved earthen stoves: improving health for rural communities in Bangladesh." *Energy for Sustainable Development* 10(3): 46-53.

Allen, R. W., C. Carlsten, B. Karlen, S. Leckie, S. van Eeden, S. Vedal, I. Wong, and M. Brauer. 2011. "An air filter intervention study of endothelial function among healthy adults in a woodsmoke-impacted community." *American journal of respiratory and critical care medicine* 183(9): 1222-30.

Armendáriz-Arnez, C., R. D. Edwards, M. Johnson, I. A. Rosas, F. Espinosa, and O. R. Masera. 2010. "Indoor particle size distributions in homes with open fires and improved Patsari cook stoves." *Atmospheric Environment* 44(24): 2881-86.

Balakrishnan, K., P. Ramaswamy, S. Sambandam, G. Thangavel, S. Ghosh, P. Johnson, K. Mukhopadhyay, V. Venugopal, and V. Thanasekaraan. 2011. "Air pollution from household solid fuel combustion in India: an overview of exposure and health related information to inform health research priorities." *Global health action* 4.

Balakrishnan, K., P. Ramaswamy, and S. Sankar. 2004. "Biomass Smoke and Health Risks–The Situation in Developing Countries." *Air Pollution*: 219-39.

Ballard-Tremeer, G. and H. Jawurek. 1996. "Comparison of five rural, wood-burning cooking devices: efficiencies and emissions." *Biomass and Bioenergy* 11(5): 419-30.

Banerjee, A., N. K. Mondal, D. Das, and M. R. Ray. 2011. "Neutrophilic inflammatory response and oxidative stress in premenopausal women chronically exposed to indoor air pollution from biomass burning." *Inflammation*: 1-13.

Barregard, L., G. Sällsten, L. Andersson, A. C. Almstrand, P. Gustafson, M. Andersson, and A. C. Olin. 2008. "Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress." *Occupational and Environmental Medicine* 65(5): 319.

Barregard, L., G. Sällsten, P. Gustafson, L. Andersson, L. Johansson, S. Basu, and L. Stigendal. 2006. "Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation." *Inhalation Toxicology* 18(11): 845-53.

Bates, E., N. Bruce, A. Doig, and S. Gitonga. 2002. "Participatory approaches for alleviating indoor air pollution in rural Kenyan kitchens." *Boiling point*: 12-15.

Bølling, A. K., J. Pagels, K. E. Yttri, L. Barregard, G. Sallsten, P. E. Schwarze, and C. Boman. 2009. "Health effects of residential wood smoke particles: the importance of combustion conditions and physicochemical particle properties." *Particle and fibre toxicology* 6(1): 29.

Bruce, N. G. 2010. "Symposium: The RESPIRE randomized control trial: preventing child ALRI by reducing household air pollution and the implications for pneumonia prevention globally. Presented at the Health Effects Institute 2010 annual conference, April 27th 2010."

Bryce, J., C. Boschi-Pinto, K. Shibuya, and R. E. Black. 2005. "WHO estimates of the causes of death in children." *The Lancet* 365(9465): 1147-52.

Clark, M. L., J. L. Peel, J. B. Burch, T. L. Nelson, M. M. Robinson, S. Conway, A. M. Bachand, and S. J. Reynolds. 2009. "Impact of improved cookstoves on indoor air pollution and adverse health effects among Honduran women." *International Journal of Environmental Health Research* 19(5): 357-68.

Clark, M. L., S. J. Reynolds, J. B. Burch, S. Conway, A. M. Bachand, and J. L. Peel. 2010. "Indoor air pollution, cookstove quality, and housing characteristics in two Honduran communities." *Environmental research* 110(1): 12-18.

Cynthia, A. A., R. D. Edwards, M. Johnson, M. Zuk, L. Rojas, R. D. Jiménez, H. Riojas-Rodriguez, and O. Masera. 2008. "Reduction in personal exposures to particulate matter and carbon monoxide as a result of the installation of a Patsari improved cook stove in Michoacan Mexico." *Indoor air* 18(2): 93-105.

Danielsen, P. H., E. V. Bräuner, L. Barregard, G. Sällsten, M. Wallin, R. Olinski, R. Rozalski, P. Møller, and S. Loft. 2008. "Oxidatively damaged DNA and its repair after experimental exposure to wood smoke in healthy humans." *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis* 642(1-2): 37-42.

Danielsen, P. H., S. Loft, N. R. Jacobsen, K. A. Jensen, H. Autrup, J. L. Ravanat, H. Wallin, and P. Møller. 2010. "Oxidative stress, inflammation, and DNA damage in rats after intratracheal instillation or oral exposure to ambient air and wood smoke particulate matter." *Toxicological Sciences* 118(2): 574-85.

Danielsen, P. H., S. Loft, A. Kocbach, P. E. Schwarze, and P. Møller. 2009. "Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines." *Mutation Research/Genetic Toxicology and Environmental Mutagenesis* 674(1-2): 116-22.

Dennis, R. J., D. Maldonado, S. Norman, E. Baena, and G. Martinez. 1996. "Woodsmoke exposure and risk for obstructive airways disease among women." *Chest* 109(1): 115-19.

Drost, E., K. Skwarski, J. Sauleda, N. Soler, J. Roca, A. Agusti, and W. MacNee. 2005. "Oxidative stress and airway inflammation in severe exacerbations of COPD." *Thorax* 60(4): 293.

Dutta, K., K. N. Shields, R. Edwards, and K. R. Smith. 2007. "Impact of improved biomass cookstoves on indoor air quality near Pune, India." *Energy for Sustainable Development* 11(2): 19-32.

Edwards, R. D., Y. Liu, G. He, Z. Yin, J. Sinton, J. Peabody, and K. Smith. 2007. "Household CO and PM measured as part of a review of China's National Improved Stove Program." *Indoor air* 17(3): 189-203.

Ekici, A., M. Ekici, E. Kurtipek, A. Akin, M. Arslan, T. Kara, Z. Apaydin, and S. Demir. 2005. "Obstructive airway diseases in women exposed to biomass smoke." *Environmental research* 99(1): 93-98.

El Tayeb Muneer, S. and E. W. Mukhtar Mohamed. 2003. "Adoption of biomass improved cookstoves in a patriarchal society: an example from Sudan." *Science of the Total Environment* 307(1): 259-66.

Ezzati, M. and D. M. Kammen. 2002. "The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps, and data needs." *Environmental health perspectives* 110(11): 1057.

Fitzgerald, C., M. Aguilar-Villalobos, A. R. Eppler, S. C. Dorner, S. L. Rathbun, and L. P. Naeher. 2012. "Testing the effectiveness of two improved cookstove interventions in the Santiago de Chuco Province of Peru." *Science of the Total Environment* 420(0): 54-64.

Fullerton, D. G., N. Bruce, and S. B. Gordon. 2008. "Indoor air pollution from biomass fuel smoke is a major health concern in the developing world." *Transactions of the Royal Society of Tropical Medicine and Hygiene* 102(9): 843-51.

GACC. 2011. "The Global Alliance for Clean Cookstoves: Overview [Document on the Internet] [updated 2011; cited 2011 Oct 23]. Available from http://cleancookstoves.org/."

Ghio, A. J., J. M. Soukup, M. Case, L. A. Dailey, J. Richards, J. Berntsen, R. B. Devlin, S. Stone, and A. Rappold. 2011. "Exposure to wood smoke particles produces inflammation in healthy volunteers." *Occupational and Environmental Medicine*.

Ghio, A. J., J. M. Soukup, M. Case, L. A. Dailey, J. Richards, J. Berntsen, R. B. Devlin, S. Stone, and A. Rappold. 2012. "Exposure to wood smoke particles produces inflammation in healthy volunteers." *Occupational and Environmental Medicine* 69(3): 170-75.

Hartinger, S., Commodore, AA, Hattendorf, J, Lanata, CF, Gil, AI, Verastegui, H, Aguilar-Villalobos, M, Mäusezahl, D, Naeher, LP. in review. "Chimney stoves in an intervention study do not improve household air pollution when compared with traditional open fire stoves in rural Peru." *Indoor air*.

Hartinger, S., C. Lanata, J. Hattendorf, A. Gil, H. Verastegui, T. Ochoa, and D. Mäusezahl. 2011. "A community randomised controlled trial evaluating a home-based environmental intervention package of improved stoves, solar water disinfection and kitchen sinks in rural Peru: Rationale, trial design and baseline findings." *Contemporary Clinical Trials* 32(6): 864-73.

Henkle, J., C. Mandzuk, E. Emery, L. Schrowe, and J. Sevilla-Martir. 2010. "Global Health and International Medicine: Honduras Stove Project." *Hispanic Health Care International* 8(1): 36-46.

Hosgood, H. D., H. Wei, A. Sapkota, I. Choudhury, N. Bruce, K. R. Smith, N. Rothman, and Q. Lan. 2011. "Household coal use and lung cancer: systematic review and meta-analysis of case–control studies, with an emphasis on geographic variation." *International Journal of Epidemiology* 40(3): 719-28.

INEI. 2007. "Instituto Nacional de Estadística e Informática[Document on the Internet] [updated 2007; cited 2012 May 10]. Available from http://www.inei.gob.pe."

Jalava, P. I., R. O. Salonen, K. Nuutinen, A. S. Pennanen, M. S. Happo, J. Tissari, A. Frey, R. Hillamo, J. Jokiniemi, and M. R. Hirvonen. 2010. "Effect of combustion condition on cytotoxic and inflammatory activity of residential wood combustion particles." *Atmospheric Environment* 44(13): 1691-98.

Khushk, W., Z. Fatmi, F. White, and M. Kadir. 2005. "Health and social impacts of improved stoves on rural women: a pilot intervention in Sindh, Pakistan." *Indoor air* 15(5): 311-16.

Kim, K. H., S. A. Jahan, and E. Kabir. 2011. "A review of diseases associated with household air pollution due to the use of biomass fuels." *Journal of hazardous materials*.

Kinnula, V. L., H. Ilumets, M. Myllärniemi, A. Sovijärvi, and P. Rytilä. 2007. "8-Isoprostane as a marker of oxidative stress in nonsymptomatic cigarette smokers and COPD." *European Respiratory Journal* 29(1): 51-55.

Kipen, H., D. Rich, W. Huang, T. Zhu, G. Wang, M. Hu, S. Lu, P. Ohman-Strickland, P. Zhu, and Y. Wang. 2010. "Measurement of inflammation and oxidative stress following drastic changes in air pollution during the Beijing Olympics: a panel study approach." *Annals of the New York Academy of Sciences* 1203(1): 160-67.

Kocbach, A., J. I. Herseth, M. Låg, M. Refsnes, and P. E. Schwarze. 2008. "Particles from wood smoke and traffic induce differential pro-inflammatory response patterns in co-cultures." *Toxicology and applied pharmacology* 232(2): 317-26.

Kurmi, O. P., S. Semple, P. Simkhada, W. C. S. Smith, and J. G. Ayres. 2010. "COPD and chronic bronchitis risk of indoor air pollution from solid fuel: a systematic review and meta-analysis." *Thorax* 65(3): 221-28.

Li, C., S. Kang, P. Chen, Q. Zhang, J. Guo, J. Mi, P. Basang, Q. Luosang, and K. R. Smith. 2012a. "Personal PM< sub> 2.5</sub> and indoor CO in nomadic tents using open and chimney biomass stoves on the Tibetan Plateau." *Atmospheric Environment*.

Li, N., T. Xia, and A. E. Nel. 2008. "The role of oxidative stress in ambient particulate matter-induced lung diseases and its implications in the toxicity of engineered nanoparticles." *Free Radical Biology and Medicine* 44(9): 1689-99.

Li, Z., A. Commodore, S. Hartinger, E. Pittman, D. Trinidad, R. Ostovar, K. Hubbard, A. Sjodin, C. Lanata, A. Gil, D. Mäusezahl, and L. Naeher. 2012b. "O-046: Assessment of Exposure to PAHs in Cook Stove Intervention Projects in Peru by Urinary Biomonitoring." *Epidemiology* 23(5S): 10.1097/01.ede.0000416704.62764.3d.

Li, Z., A. Sjödin, L. C. Romanoff, K. Horton, C. L. Fitzgerald, A. Eppler, M. Aguilar-Villalobos, and L. P. Naeher. 2011. "Evaluation of exposure reduction to indoor air pollution in stove intervention projects in Peru by urinary biomonitoring of polycyclic aromatic hydrocarbon metabolites." *Environment international* 37(7): 1157-63.

Limmeechokchai, B. and S. Chawana. 2007. "Sustainable energy development strategies in the rural Thailand: The case of the improved cooking stove and the small biogas digester." *Renewable and Sustainable Energy Reviews* 11(5): 818-37.

Ling, S. H. and S. F. Van Eeden. 2009. "Particulate matter air pollution exposure: role in the development and exacerbation of chronic obstructive pulmonary disease." *International journal of chronic obstructive pulmonary disease* 4: 233.

Liu, S., Y. Zhou, X. Wang, D. Wang, J. Lu, J. Zheng, N. Zhong, and P. Ran. 2007. "Biomass fuels are the probable risk factor for chronic obstructive pulmonary disease in rural South China." *Thorax* 62(10): 889-97.

Martin, W. J., R. I. Glass, J. M. Balbus, and F. S. Collins. 2011. "A Major Environmental Cause of Death." *Science* 334(6053): 180-81.

Masera, O., R. Edwards, C. A. Arnez, V. Berrueta, M. Johnson, L. R. Bracho, H. Riojas-Rodríguez, and K. R. Smith. 2007. "Impact of Patsari improved cookstoves on indoor air quality in Michoacán, Mexico." *Energy for Sustainable Development* 11(2): 45-56.

McCracken, J., K. R. Smith, P. Stone, A. Díaz, B. Arana, and J. Schwartz. 2011. "Intervention to Lower Household Wood Smoke Exposure in Guatemala Reduces ST-Segment Depression on Electrocardiograms." *Environmental health perspectives* 119(11): 1562.

McCracken, J. P. and K. R. Smith. 1998. "Emissions and efficiency of improved woodburning cookstoves in Highland Gatemala." *Environment international* 24(7): 739-47.

McCracken, J. P., K. R. Smith, A. Díaz, M. A. Mittleman, and J. Schwartz. 2007. "Chimney stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women." *Environmental health perspectives* 115(7): 996.

Naeher, L. P., M. Brauer, M. Lipsett, J. T. Zelikoff, C. D. Simpson, J. Q. Koenig, and K. R. Smith. 2007. "Woodsmoke Health Effects: A Review." *Inhalation Toxicology* 19(1): 67-106.

Nazmul Alam, S. and S. J. Chowdhury. 2010. "Improved earthen stoves in coastal areas in Bangladesh: Economic, ecological and socio-cultural evaluation." *Biomass and Bioenergy* 34(12): 1954-60.

Orozco-Levi, M., J. Garcia-Aymerich, J. Villar, A. Ramirez-Sarmiento, J. Anto, and J. Gea. 2006. "Wood smoke exposure and risk of chronic obstructive pulmonary disease." *European Respiratory Journal* 27(3): 542-46.

Pennise, D., S. Brant, S. M. Agbeve, W. Quaye, F. Mengesha, W. Tadele, and T. Wofchuck. 2009. "Indoor air quality impacts of an improved wood stove in Ghana and an ethanol stove in Ethiopia." *Energy for Sustainable Development* 13(2): 71-76.

Perez-Padilla, R., A. Schilmann, and H. Riojas-Rodriguez. 2010. "Respiratory health effects of indoor air pollution [Review article]." *The International Journal of Tuberculosis and Lung Disease* 14(9): 1079-86.

Peterson, J., M. MacDonell, L. Haroun, F. Monette, R. D. Hildebrand, and A. Taboas. 2007. "Radiological and chemical fact sheets to support health risk analyses for contaminated areas." *Human Health Fact Sheet, Argonne*: 38-39.

Regalado, J., R. Pérez-Padilla, R. Sansores, J. I. P. Ramirez, M. Brauer, P. Paré, and S. Vedal. 2006. "The effect of biomass burning on respiratory symptoms and lung function in rural Mexican women." *American journal of respiratory and critical care medicine* 174(8): 901-05.

Rehfuess, E., S. Mehta, and A. Prüss-Üstün. 2006. "Assessing household solid fuel use: multiple implications for the Millennium Development Goals." *Environmental health perspectives* 114(3): 373.

Romieu, I., H. Riojas-Rodriguez, A. T. Marron-Mares, A. Schilmann, R. Perez-Padilla, and O. Masera. 2009. "Improved biomass stove intervention in rural Mexico: impact on the respiratory health of women." *American journal of respiratory and critical care medicine*: 200810-1556OCv1.

Saha, A., P. Kulkarni, A. Shah, M. Patel, and H. Saiyed. 2005. "Ocular morbidity and fuel use: an experience from India." *Occupational and Environmental Medicine* 62(1): 66.

Smith, K. R. 1987. *Biofuels, air pollution, and health: a global review*: Plenum press.

Smith, K. R., J. P. McCracken, L. Thompson, R. Edwards, K. N. Shields, E. Canuz, and N. Bruce. 2010. "Personal child and mother carbon monoxide exposures and kitchen levels: Methods and results from a randomized trial of woodfired chimney cookstoves in Guatemala (RESPIRE)." *J Expos Sci Environ Epidemiol* 20(5): 406-16.

Smith, K. R., J. P. McCracken, M. W. Weber, A. Hubbard, A. Jenny, L. M. Thompson, J. Balmes, A. Diaz, B. Arana, and N. Bruce. 2011. "Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial." *The Lancet* 378(9804): 1717-26.

Smith, K. R. and S. Mehta. 2003. "The burden of disease from indoor air pollution in developing countries: comparison of estimates." *International Journal of Hygiene and Environmental Health* 206(4-5): 279-89.

Smith, K. R., J. M. Samet, I. Romieu, and N. Bruce. 2000. "Indoor air pollution in developing countries and acute lower respiratory infections in children." *Thorax* 55(6): 518-32.

Swiston, J. R., W. Davidson, S. Attridge, G. T. Li, M. Brauer, and S. F. van Eeden. 2008. "Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters." *European Respiratory Journal* 32(1): 129-38.

Thompson, L. M., N. Bruce, B. Eskenazi, A. Diaz, D. Pope, and K. R. Smith. 2011. "Impact of Reduced Maternal Exposures to Wood Smoke from an Introduced Chimney Stove on Newborn Birth Weight in Rural Guatemala." *Environmental health perspectives* 119(10): 1489.

Van Eeden, S., J. Leipsic, S. F. P. Man, and D. D. Sin. 2012. "The relationship between lung inflammation and cardiovascular disease." *American journal of respiratory and critical care medicine* 186(1): 11-16.

van Eeden, S. F. and D. D. Sin. 2008. "Chronic obstructive pulmonary disease: a chronic systemic inflammatory disease." *Respiration* 75(2): 224-38.

Venkataraman, C., A. Sagar, G. Habib, N. Lam, and K. Smith. 2010. "The Indian national initiative for advanced biomass cookstoves: the benefits of clean combustion." *Energy for Sustainable Development* 14(2): 63-72.

WHO. 2011. "Indoor air pollution and health; fact sheet 292.".

Y von Schirnding, N. B., K Smith, G Ballard-Tremeer, M Ezzati, K Lvovsky. 2002. *Addressing the Impact of Household Energy and Indoor Air Pollution on the Health of Poor: Implications for Policy Action and Intervention Measures—paper prepared for the Commission on Macroeconomics and HealthWHO, Geneva (2002)*: World Health Organization.

Zhang, J. J. and K. R. Smith. 2007. "Household air pollution from coal and biomass fuels in China: measurements, health impacts, and interventions." *Environmental health perspectives* 115(6): 848.

CHAPTER 5

CARBON MONOXIDE EXPOSURES AND KITCHEN CONCENTRATIONS FROM COOKSTOVE RELATED WOODSMOKE IN SAN MARCOS, PERU[1]

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ABSTRACT

Background: Nearly half of the world's population is exposed to household air pollution (HAP) due to long hours spent in close proximity to biomass-fueled fires.

Objective: We compare CO exposures and concentrations among study promoted intervention stove users and control stove users in San Marcos Province, Cajamarca region, Peru.

Methods: Passive CO diffusion tubes were deployed over a 48-hour sampling period to measure kitchen CO concentrations and personal mother and child CO exposures in 197 control and 182 intervention households.

Results: Geometric means (95%CI) for child, mother and kitchen measurements were 1.1 (0.9, 1.2) ppm, 1.4 (1.3, 1.6) ppm and 7.3 (6.4, 8.3) ppm in control households, and 1.0 (0.9, 1.1) ppm, 1.4 (1.3, 1.6) ppm and 7.3 (6.4, 8.2) ppm among intervention households respectively.

Conclusion: With no significant differences between control and intervention CO measurements, results suggest intervention stove maintenance may be necessary for long-term reductions in CO exposures.

INTRODUCTION

It is estimated that nearly half of the world's population burns biomass, mostly as fuel for cooking (Smith 1987; WHO 2011) resulting in household air pollution (HAP). Women and young children bear the brunt of high HAP exposures due to the long hours spent in close proximity to cooking fires (Ezzati and Kammen 2002; Martin et al. 2011). Household stoves typically used for cooking and heating in the developing world do not burn fuel cleanly leading to incomplete combustion in the domestic environment (Martin et al. 2011; Naeher et al. 2007; Smith 1987). Smoke from incomplete biomass combustion contains health-damaging pollutants, (Bølling et al. 2009; Jalava et al. 2010) of which carbon monoxide (CO) and particulate matter with an aerodynamic diameter of $\leq$2.5 microns ($PM_{2.5}$) are major constituents (Naeher et al. 2007; Northcross et al. 2010).

HAP is responsible for almost 50% of the burden of diseases resulting from poor household environments (Kim, Jahan, and Kabir 2011b; Rehfuess et al. 2006; WHO 2011), while other problems such as inadequate waste management and sanitation, overcrowding, malnutrition and lack of access to clean water also contribute to this burden (Gopalan et al. 1999). HAP levels may vary depending on factors such as the time spent cooking, fuel type, cooking environment and household ventilation (Armendáriz-Arnez et al. 2010; Dutta et al. 2007; Romieu et al. 2009). Concentrations of CO and PM also vary over short (less than one day) time periods (McCracken and Smith 1998).

As such it is essential to capture high intensity exposures and emissions over an extended period of time. Adequate characterization of exposure to residential biomass combustion is crucial in vulnerable populations such as in rural communities in Peru where biomass fuels are used on a daily basis for cooking and heating (INEI 2007).

Personal exposures and kitchen concentrations of HAP can be estimated using questionnaires and exposure modeling, measured directly with air pollution monitors and to a limited extent, biomarkers can also be used to estimate internal dose from HAP exposures (Allen et al. 2011; Banerjee et al. 2011; Li et al. 2011; McCracken et al. 2011; Naeher et al. 2007; Oanh, Reutergårdh, and Dung 1999). CO can be used as a proxy for $PM_{2.5}$ when both pollutants are from the same source and air pollution levels are high as observed in indoor cooking conditions (Naeher et al. 2001). A few studies in the past decade have successfully demonstrated the use of inexpensive passive diffusion tubes in quantifying exposure to HAP as well as ambient concentrations (Dionisio et al. 2008; Dionisio et al. 2012; Naeher et al. 2001; Northcross et al. 2010; Smith et al. 2010).

We report a cross sectional study conducted within the framework of a community-randomized controlled trial (c-RCT, parent study) by the Instituto de Investigación Nutricional (IIN) and the Swiss Tropical and Public Health Institute (Hartinger et al. 2011). Our primary objective was to compare CO exposures and concentrations among study promoted intervention stove users and control stove users in San Marcos Province, Cajamarca region, Peru.

We also investigated factors that are associated with CO exposures and kitchen concentrations among study subjects. Finally, we examined correlations in CO measurements between personal mothers' and children's exposures and between personal exposures and kitchen concentrations in this population.

METHODS

Study Design and Study Homes

Measurements presented in this paper occurred between June and August 2009. The May–August period in the study region is characterized by dry conditions and cold nights. All measurements were taken during this season, no follow up measurements occurred during the rainy season. The altitude in the region ranges between 2200 and 3900 meters above sea level. Mean altitudes ± SD for intervention and control households are 2684 ± 284 and 2727 ± 438 meters above sea level respectively.

For this cross sectional study, control and intervention households were from participating households in the parent c-RCT (n=250 and 253 for intervention and control homes respectively). The c-RCT involved 51 community clusters who used solid fuels in the Province of San Marcos, Cajamarca region, Peru (Hartinger et al. 2011). The intervention was randomized at the community level, with the 51 community clusters allocated into the intervention arms by using covariate-based constrained randomization (Hartinger et al. 2011). Field workers for the c-RCT visited all study homes during this 3 month period; however subjects' availability, willingness to participate, as well as time and budget constraints limited the total sample size of the present study.

The aim of the parent study was to evaluate an integrated home-based environmental intervention package (IHIP) against childhood diarrhea and respiratory infections. A pilot study

was conducted in seven communities outside the study area, where several potential stove designs were tested, and subjects were consulted on cooking habits and preferences to provide a user-friendly stove design which met their household and cooking needs (Hartinger 2012). The final stove model, for the c-RCT was called the OPTIMA-improved stove (hereafter OPTIMA stove). Kitchen performance tests of the OPTIMA stoves revealed a 15% reduction in daily fuel and energy use and a 16% reduction in fuel and energy use per capita compared with the traditional open fire stoves, although there was wide variability (Hartinger 2012; Hartinger et al. 2011). The OPTIMA stove was built with red burnt bricks plastered with a mixture of mud, straw and donkey manure (Hartinger 2012). It has three pot holes for cooking, a closed combustion chamber, metal chimney with a regulatory valve, a hood, and metal rods for support.

OPTIMA stoves were installed between October 2008 and January 2009 in 250 households (hereafter intervention households). There were no emissions tests or HAP exposure assessment prior to installation of the intervention stoves. The current study reports the only exposure assessment conducted for these stoves 6 to 8 months after installation (median 7.4 IQR = 6.6-8.1 month) (Hartinger in review). OPTIMA stoves were later stratified (after exposure assessment had occurred) into two categories based on their levels of functionality (FL). FL-I stoves were in good running conditions at the time of the assessment (plastered stove and no visible leaks when in use) and FL-II stoves were in need of repairs (re-plastering, filling small cracks, cleaning the chimney, chimney valve replacement). Field workers, during monthly visits, instructed OPTIMA stove users in the correct use of the stoves including cleaning and removing ashes and wood residues. Although surveillance occurred in all study homes, stove repair and maintenance were not addressed during home visits until after air quality monitoring had occurred. Households with OPTIMA-improved stoves were re-visited 9 months (median 9.3

IQR= 9.0-9.7 month) after installation and repaired as needed by the original stove builders (Hartinger in review).

The control arm of the c-RCT included households with a diversity of stove types. (Hartinger SM) As such control households in this study had a wide range of stove types including (1) chimney stoves whose raw materials were provided by nongovernmental organizations (hereafter referred to as NGO; n=30), (2) chimney stoves built by the households themselves (hereafter referred to as self-improved by household; n=34), (3) gas stoves (n=4), and (4) non-vented stoves with pot holes for cooking including the common three stone open fire stove (hereafter referred to as traditional; n=129). At the time of sampling, control households had stoves which had been in use between 4 months to over 10 years. Lastly, households in each arm of the intervention were classified according to the primary stove in use and it is possible that some chimney stoves were used together with traditional stoves in some households, particularly for cooking animal feed or other meals which required substantial cooking times.

Sample size

Mothers/primary caregivers (hereafter referred to as mothers) were sampled from 182 intervention households (final n=161) and 197 control households (final n=154) (Table 5.1). Some households were sampled two or three times during the study period (13 intervention households and 12 control households) and in eight control households two tubes were used on the same day. In each case, the multiple measurements (pseudo-replicates) were averaged to get a single value for data analysis per subject. Losses in sample size were similar except for the number of broken tubes (n=5/182 (3%) among intervention and n=18/197 (9%) among control households). We are unsure as to why there was a higher breakage rate among control

households, but we do not expect this to influence our study findings. Measurements were not reattempted in households with lost or broken tubes.

During the first month of sampling, kitchen tubes were taped directly above stove openings in study kitchens at ~1.5m. These tubes, representing 29% of the data have been excluded from all analysis to avoid inflating the values of the kitchen measurements (Table 5.1). There were a total of forty tubes (n=11/182 (6%) from intervention households and n=29/197 (14%) from control households) that had yellow and/or white stains. Like Smith et al in 2010, these tubes were excluded from the final data set as the stains may be due to other gases that entered the tube along with CO during sampling. Duplicate same day measurements in a small subset of households were collected to check for reliability in tube measurement. All collocated tubes had stain length measurements within 1.5 mm of each other (10 ppm-hr). Due to field workers monitoring previously sampled community clusters, certain households were sampled more than once during the three month exposure assessment.

Exposure Assessment

Time integrated CO measurements were taken using Dräger Diffusion Tube for Carbon Monoxide, with a range of 6-600 ppm-hr (parts per million-hour). All tubes were from the same manufacturing lot. The sampler uses principles of diffusion and colorimetry where CO passively diffuses into the tube and causes the reduction of sodium palladosulfite to palladium metal.(Dräger-Tubes & Chip-Measurement-System Handbook (PDF). 16th edition Soil) The result is a grayish stain inside the tube, which corresponds to a cumulative dose of CO.

Three CO passive diffusion samplers were set up and left in place for 48 hours in each household to measure exposures to CO. Two tubes were for personal sampling: one worn in the breathing zone of the mother and one worn by a child under the age of 5 years who was enrolled in the

parent c-RCT. The third tube was set up in the kitchen, at the breathing height (approximately 1.5m) of the mother and close to where she stands during cooking. The times of tube breakage and capping, which marked the beginning and termination of sampling respectively, were recorded on data sheets.

For all but 93 study subjects, tubes were placed in cloth coverings with an attached string for hanging around the neck, and pinned in the subject's breathing zones. The cloth covering was for comfort, protection of tubes from direct sunlight (Smith et al. 2010), and has been shown to not affect CO measurements (Bruce et al. 2004). For 93 of the mothers (50 control and 43 intervention stove users) in this study, CO tubes were placed in vests worn in the breathing zones of subjects. These vests held real time CO monitors and 48hr time integrated $PM_{2.5}$ samplers for personal air sampling and the data for these measurements are presented elsewhere (Hartinger in review). CO tubes from these 93 mothers are included in the final data set of this study. Subjects were instructed to keep the tubes on at all times and to place them by their bedside at night.

Upon return to the field station, tubes were stored in a +4°C refrigerator before and after reading. Tubes were read by two of the authors (AAC and SMH) and an arithmetic mean was taken. Reading took place in a white, bright fluorescent tube lit laboratory room at a table with a white surface. The least squares regression technique developed by Smith et al (2010) for RESPIRE (Randomized Exposure Study of Pollution Indoors and Respiratory Effects) was employed. In brief, the length of stain was measured for each tube and converted to a cumulative exposure in ppm-hr. Ppm-hr was subsequently divided by the total sampling time to obtain CO personal exposures and kitchen levels. Questionnaires were administered on the second day of air sampling to obtain data on household air pollution, respiratory health-related symptoms, demographics, daily activities and commuting habits (Hartinger in review).

Human Subjects and Ethical Issues Statement

This study was approved by the Internal Review Boards at University of Georgia, the Centers for Disease Control and Prevention of the United States, and by the ethical committee of the IIN and the ethical review board at the Cayetano Heredia University in Peru. The demographic and socio-economic data had previously been collected in the parent study (ClinicalTrials.gov Identifier: NCT00731497) which had received clearance from the independent ethics committees of IIN and the ethical review board of University of Basel, Switzerland (Ethikkommission Beider Basel, EKBB). Signed consent forms were obtained from all participating households. During May 2010, workshops were held to present study results and hold discussions with the communities.

Statistical Analysis

SAS version 9.1 (SAS Institute, NC, USA) was used for all data analysis. Sampling duration ranged from 2399 (40hr) to 3442 (57hr) minutes, with a mean of 2860 minutes (48hr). All CO data were natural log transformed for regression analyses. SAS PROC GLM was used to fit general linear models which assess the impact of select variables on personal mother and child CO exposures as seen in equation (1):

$$y_{ij} = \mu_j + \beta_1 X_{1ij} + \ldots + \beta_p X_{pij} + \varepsilon_{ij} \quad (1)$$

Here y_{ij} is the log CO exposure/concentration measured on the i^{th} subject/kitchen with the j^{th} stove type; μ_j is the population mean log CO for the jth stove type at the average value of the covariate; β_p is the effect of X_p, the covariate under consideration and ε_{ij} is a mean zero, constant variance error term assumed to follow a normal distribution.

The passive CO tubes placed in kitchens were found to have reached the 600 ppm-hr upper limit after 48-hours for 46/113 intervention households and 59/120 control households.

Hence for kitchen concentrations only, due to right censoring of approximately 47% of the data, PROC LIFEREG was used to fit linear models to the kitchen data. These models explain the linear relationship between kitchen CO concentrations and select variables in the form given in equation (1). The LIFEREG procedure implements maximum likelihood estimation and inference in the presence of censored data. Ignoring the censoring (e.g., by fitting the model via least squares as in PROC GLM) would result in biased parameter estimates, incorrect standard errors and invalid statistical inference.

Information on covariates was obtained from the administered questionnaires on the second day of HAP sampling. Covariates were included in GLM models individually to test for significant associations with personal or kitchen CO. The final group of covariates considered for inclusion in the full models include mother's age, time spent playing with child, cooking time, number of people in household, presence of smokers in household, age of stove, wood type used in cooking, kitchen environment, mother's frequency of cleaning ashes from stove, distance of household to road and stove type (Table 5.4).

Backward elimination was the process used for model selection. Starting with all candidate variables, we removed nonsignificant variables other than stove type using a chosen model comparison criterion (P=0.2). Variables were deleted one at a time if the P values for their corresponding regression coefficients were higher than 0.2. This process was repeated until only variables that were statistically significant remained in the model. The effect of stove type was retained in all models to allow comparison of CO exposures and concentrations across stove type.

Kitchen environment refers to the nature of the cooking area which was categorized as enclosed (four full walls and a roof) or open (less than four walls, or open to the outside). Wood

type refers to the most common wood type cooked with by the various wood stove users in this study. Cooking time refers to the estimated amount of time mothers spent cooking a meal on a typical day and it is a way to estimate proximity to the cooking fire. The time mothers spent in playing with their children during the day was also assessed to determine whether this affected their respective exposures. This variable was chosen as a proxy for how often the mother and child are together on any given day. This variable was considered to be potentially important because if playing time did not overlap with cooking time, it could impact personal exposures.

Stove type was retained in all models in order to compare personal exposures and kitchen log CO concentrations across stove types (first by control and intervention stoves and then by specific stove types). Comparisons were done with an F test for equal means across all stove types for all models. Then for personal mother and child exposures, Dunnett's test for pairwise comparisons of each stove type with OPTIMA FL-I as the reference stove was performed. Cooking time and time mothers spent playing with their children were centered at their respective means across households in all regression models. Finally, to examine how well the tubes predict personal and kitchen area measurements, Spearman correlation coefficients (r) between personal mother and child CO exposures and between personal (mother and child) and kitchen CO measurements were calculated separately by stove type.

RESULTS

Household characteristics

Demographic and household information for households using various stove types are presented in Table 5.2. Except for differences in television ownership and the number of smokers present in households, the study population was comparable with respect to their socio-demographic and kitchen characteristics (Table 5.2). Households with NGO stoves and self-

improved by household stoves owned fewer television sets: 5% and 6% respectively compared to 17% , 29% and 30% for the OPTIMA FL-II, FL-I and traditional stoves respectively (Table 5.2). Households with self-improved stoves had 19% of family members who smoked, compared to 7% - 8% of smokers in households with other stove types (Table 5.2). All variables mentioned above are comparable among control and intervention households in the entire c-RCT population (Hartinger et al. 2011) and were not statistically significant during subsequent regression analysis. *Eucalypto* (eucalyptus) was the most common wood type used for cooking by 34% (n=32 OPTIMA FL-I stoves) to 65% (n=13 self-improved by household stoves) of the women in this study (Table 5.2).

CO exposures and concentrations

Summary statistics of unadjusted CO exposures and kitchen concentrations in intervention and control households and across stove type are presented in Table 3. It must be noted that our study population included mothers who used gas stoves (n=4), these have been excluded from subsequent analysis.

Regression Analysis: control and intervention households

There were no statistically significant differences between intervention and control households for any of personal CO exposures: mother (F=0.02; df=1, 288; P=0.89), child (F=0.49; df=1, 287; P=0.48). Likewise for kitchen concentrations, the model revealed no differences in kitchen CO concentrations in intervention and control households (X^2=0.28; df=1; P=0.59). Due to the lack of statistical significant differences between control and intervention measurements, for the remainder of the results, we analyze data by stove type: OPTIMA FL-I, OPTIMA FL-II, NGO, traditional and stoves which were self-improved by the households.

Regression Analysis: specific stove types

Personal CO exposures: Mothers

Summary statistics for covariates included in our models are presented according to stove type (Table 5.4). The regression model for mothers in this study revealed that personal CO exposures did not differ significantly across stove types (overall F test statistic=0.24, P=0.92, Table 5.5A).

All other variables were found to be statistically insignificant using backward elimination. Dunnett's test revealed no significant difference between mean mother personal log CO exposures using the OPTIMA FL-I stove (n=92) and any other stove type (P=1.00, 1.00, 0.85 and 1.00 for OPTIMA FL-II (n=59), NGO (n=23), self-improved by household (n=20), traditional (n=96) stoves respectively, Table 5.5A).

Personal CO exposures: children

Although not found to be statistically significant, children's CO exposures were lower in households with self-improved stoves and higher for all other stove types (overall F test statistic=1.67, P=0.16, Table 5.5B). Dunnett's test revealed no significant differences in mean child personal CO exposures between OPTIMA FL-I (n=92) and other stove types (P=1.00, 0.79, 0.34, 0.56 for OPTIMA FL-II (n=59), NGO (n=22), self-improved by household (n=19), traditional (n=95) stoves respectively, Table 5.5B).

For children in this study, the regression model showed that time mothers spent cooking during the sampling period was marginally associated (P=0.0504) with their CO exposures (Table 5.5B). The model estimated a decrease of 0.11 ppm (SE=0.056) in children's personal CO exposures for every additional hour spent cooking by their mothers. Children's age in years was centered at its mean across households to investigate the effect of age, and possible interactions between child's age and mother's time spent cooking. However, neither of these effects were statistically significant (P=0.7363 and P=0.1943), for the main and interaction effects, respectively. The interaction between cooking time and the time mothers spent playing with children also did not reach statistical significance (F test statistic=0.94, P=0.33).

Kitchen CO concentrations

Kitchen CO concentrations were marginally associated with the type of wood used for cooking (X^2=5.52, df=2, P=0.06, Table 5.5C). Study subjects used different types of wood as fuel including pine, *eucalypto* (eucalyptus), cypress, *talla*, *huayo* and *hualango*. After preliminary analysis, firewood types were grouped into *eucalypto* (n=82) , *hualango* (n=68) and other wood types (n=77). Households that used *hualango* had higher and statistically significant (P=0.02) kitchen CO whereas households that used *eucalypto* did not (P=0.22) when compared to other wood types (Table 5.5C). There was no difference in kitchen CO between households that used *eucalypto* compared to *hualango* (P=0.19). Households using *hualango* had 8.4 (7.2, 9.8) ppm (mean with 95% CI), those using *eucalypto* had 7.4 (6.4, 8.6) ppm and those using other wood types had 6.7 (5.6, 7.8) ppm of CO in the kitchen (Table 5.5C).

Passive tube correlations

Spearman correlation coefficients (r) between personal CO exposures and kitchen CO levels are presented by stove type used in households in Figure 5.1. All mother and child measurements were correlated (r=0.63 P<0.0001 n=299 Figure 5.1A). The correlation coefficient value between mother and child personal samples was larger for all control stoves when compared with all intervention stoves (control: r=0.67 P<0.0001 n=145, intervention: r=0.60 P<0.0001 n=154).

Among intervention households, correlations between personal mother and child CO exposures were moderate to low (Figure 5.1 B and C), with the strength of the correlation slightly increasing with decreasing stove quality (r=0.58 P<0.0001 n=93 for OPTIMA FL-I compared to r=0.66 P<0.0001 n=61 for OPTIMA FL-II). Personal mother and kitchen samples were moderately to weakly correlated (r=0.23 P<0.03 n=67 for OPTIMA FL-I compared to

r=0.29 P=0.02 n=46 for OPTIMA FL-II). Child and kitchen samples had a weak correlation (r=0.38 P=0.0002 n=67) for households with OPTIMA FL-I and a marginal statistically significant correlation for OPTIMA FL-II households (r=0.23 P=0.08 n=46).

For personal mother and child's correlation in the control arm of the intervention, households using traditional stoves (Figure 5.1F) had a larger correlation (r=0.70 P<0.0001 n=98) than households using NGO (Figure 5.1D) and self-improved by household (Figure 5.1E) stoves (r=0.56 P=0.003 n=25 and r=0.57 P=0.005 n=21 respectively). Kitchen CO levels were marginally correlated with mothers' personal exposures (r=0.47 P=0.06 n=17) and significantly correlated with children's exposures when stoves were self-improved by household (r=0.51 P=0.04 n=17).

DISCUSSION

Carbon monoxide measurements in this study did not demonstrate statistically significant differences across the various stove types in both arms of the intervention. The lack of differences in CO exposures between control and intervention households seems contrary to results reported in other chimney stove intervention studies (Chengappa et al. 2007; Cynthia et al. 2008; Dutta et al. 2007; Fitzgerald et al. 2012a; Masera et al. 2007; Smith et al. 2010). While some of the intervention studies mentioned above assessed HAP exposures before and soon after stove installation, other stoves were monitored frequently and stoves were routinely fixed. In this cross sectional study, we present data on HAP measurements of chimney stoves referred to as OPTIMA stoves that had been in use, on average, for several months. Some of these stoves had not been maintained, and may have been improperly used. Our results have potential implications for intervention studies in the developing world aiming to answer the question of stove performance months after installation and use.

Findings from RESPIRE demonstrate that a well-maintained stove decreased CO exposures by 50% and kitchen concentrations by 90% with a corresponding 22% decrease in physician diagnosed pneumonia in children (Smith et al. 2011). It must be noted that these households had stoves that had been installed for on average of 18 months, with weekly visits where repairs and maintenance were provided as needed (Smith et al. 2011). An ideal stove must be affordable and simultaneously have high heating efficiency and low, nonhealth-damaging emissions (Albalak et al. 2001; Martin et al. 2011). Lessons from current global stove intervention studies point to the fact that cookstove related woodsmoke exposures can be reduced, however these reductions need to be larger and must be sustained for several years to yield greater public health benefits (Smith et al. 2011; Smith and Peel 2010).

One goal of the c-RCT was to determine impact of the OPTIMA stoves in reducing acute lower respiratory infections (ALRI) in children between the ages of six and 36 months. Children in homes with OPTIMA FL-II stoves had CO exposures of 1.0 (0.9, 1.2) ppm. In the Gambia, a study of 1,115 children reported a mean CO exposure of 1.04 ±1.45 ppm (± SD) (Dionisio et al. 2012). Children in households using the plancha chimney stoves in the RESPIRE study had a geometric mean of 1.0 (2.4) ppm (GSD) (Smith et al. 2010). These CO tube measurements from children in the above mentioned studies are similar to results in the current study; however the levels of HAP experienced by OPTIMA stove users may not result in significant health improvements compared to control stove users due to two main reasons. First, there were no significant differences between control and intervention household measurements hence health impacts between the two groups are expected to be similar. Second, findings from an exposure-response analysis from the RESPIRE study suggest that larger HAP exposure reduction is needed to observe reductions in child mortality from ALRI.

Our results also suggest children's CO exposures decreased (marginally) with increasing time mothers' spent during cooking. This finding could be spurious although possible reasons could be due to decreased CO emissions from the fire presumably after cooking has occurred or after cooking, children were further away from the cookstoves. A third reason for decreased child exposure with increased cooking time could be due to maternal mis-reporting, that children were further away from cookstoves during cooking events. However, we do not have data from time activity diaries of subjects to corroborate these possible explanations.

All personal mother and child CO exposures were correlated amongst our study population. This agrees with the literature that suggests that when children data are unavailable, data from their mothers can be used to estimate exposures of the children especially in a high HAP setting (Naeher et al. 2001). As expected kitchen CO measurements were higher compared to personal measurements; and mothers' personal exposures were higher compared to children's. Also as seen from our results, kitchen measurements need to be used with caution where personal measurements are unavailable, since kitchen levels can inform but can overestimate personal exposures (Armendáriz-Arnez et al. 2010; Northcross et al. 2010; Smith et al. 2010).

Aside from the significant correlation between personal exposures, our results showed an increase in the value of the correlation coefficient with a corresponding increase in stove deterioration. For example, among intervention stove users, the correlation between personal mother and child exposures among OPTIMA FL-II stove users had a slightly higher value compared to OPTIMA FL-I. Also among control stove users, the correlation among traditional stove users had a higher value compared to households with chimney stoves. This suggests stronger correlation between personal exposures with increasing HAP levels (Naeher et al. 2001), and is a finding which needs to be corroborated by other studies.

A number of reasons may have led to high HAP levels in intervention households in our study. Adequate stove design, manner of stove use (i.e. whether it is used continuously, properly and exclusively), as well as maintenance over time are key factors in HAP mitigation. Design and construction of efficient cookstoves is also key to reducing and sustaining low exposure levels (Smith et al. 2011). It must be noted that cookstoves, with use, are expected to degrade with time even with adequate maintenance (Lokras 2012). Hence there is the need to design and construct cookstoves which factor in the high temperatures and pressure factors that will impact its degradation. Additionally it is important to recognize the effect, if any, of altitude on the combustion efficiency of cookstoves.

Improper stove use is another important factor. If fitted pots are not placed in tightly sealed pot holes on the stove top during cooking, combustion emissions can leak into the indoor environment. The same is true for openings designed for fuel insertion. Any uncovered chimney stove opening may introduce into the household environment emissions akin to an open fire. Conversely, although OPTIMA stove users were not specifically asked whether they had used open fires during the sampling period, this is a possibility given that even gas stove users in this study reported the use of firewood during the sampling period. Findings from participatory observational surveys revealed a reported 90% (212/236) daily use of the OPTIMA-improved stove after about 7 months (median 7.4 IQR = 6.6-8.1 month) (Hartinger in review).

However there is the possibility these households used open fire stoves throughout that period. Lack of exclusive and continuous stove usage can introduce more HAP into the kitchen environment and needs to be addressed if an intervention program is to be successful and sustained over time.

It has been documented that intervention stoves can improve health when properly used (Diaz et al. 2008; Romieu et al. 2009; Smith et al. 2011). It is important then to determine the stove's performance at the time of installation (Fitzgerald et al. 2012a) and also months and years after installation, as the intervention stove may possibly introduce greater HAP if improperly maintained and used. Also the importance of functionality levels within stove type is important in HAP exposure assessment. Clark et al. (2010) suggest the utility of stove functionality levels to be more representative of HAP exposures and indoor levels. They note the importance of assessing the condition of the stoves rather than a mere comparison between traditional and improve stove type (Clark et al 2010). Our results indicate that after an average of seven months of use, OPTIMA stoves (whether they were in need of repairs or not) did result in significantly lower personal CO exposures and kitchen levels when compared to control stoves. Hence stove maintenance and functionality are both essential in understanding HAP exposures (Albalak et al. 2001).

Results from our study seem to suggest that stoves which were self-improved by households had lower HAP measurements, almost akin to gas stove measurements, although this was not statistically significant. We do not know the reason for this finding.

However, we can surmise that these stoves may have had better durability, lower emissions or perhaps the subjects took more responsibility for the maintenance of these personally constructed stoves. The qualities of any control stove type must also be assessed in future studies as they could provide insight on potential stove designs in local communities.

Firewood type is another important factor in the quest to reduce HAP (Lisouza, Owuor, and Lalah 2011). In our study, households using *hualango* (Acacia sp) as firewood had higher mean kitchen CO compared to other wood types used. High biomass combustion by-products such as PM and CO are associated with biomass fuel use (Andreae and Merlet 2001; Smith 1987) hence this finding is expected. With the move to decrease HAP on the international horizon, the need for utilizing cleaner energy (from wood to eventually using gas and electricity) should be considered in conjunction with the design of cookstoves.

This study is apt even as The Global Alliance for Clean Cookstoves (GACC) continues to build momentum in the effort to reduce HAP and the adverse health effects associated with it. The GACC, led by the United Nations Foundation, has the goal of 100 million households adopting clean and efficient cookstoves by the year 2020 (GACC 2011). The success of household air pollution mitigation programs will depend not just on the number of disseminated stoves, but on the number of stoves that are adequately designed, continually, exclusively and properly used, as well as maintained over time (Armendáriz-Arnez et al. 2010; Dutta et al. 2007; Romieu et al. 2009).

Limitations

Although valuable lessons can be gleaned from our study, single 48hr measurements limit our ability to detect the temporal and within household variability in exposure (Dionisio et al. 2012; McCracken et al. 2009; Saksena et al. 1992). This is important for a site such as San Marcos, which is subject to considerable seasonal climate changes that may impact the combustion efficiency of cookstoves, and the types of available cooking fuel. Future studies should consider taking repeated measurements over time (Dionisio et al. 2012; Smith et al. 2010). Also information from time activity diaries may help future studies to derive better estimates of exposure.

It is also essential to be able to make population inferences based on larger sample sizes for each stove type. Control groups in this study, by design of the parent study, consisted of a diverse range of stoves with varying air pollution levels. Future studies with the primary aim of assessing HAP exposure need to limit the number of control groups or ensure adequate sample sizes in each stove category.

Another limitation is the timing of the HAP exposure assessment. Study households were not sampled prior to and immediately after chimney stove installation and this prevented evaluation of the effectiveness of the OPTIMA stoves soon after installation. Additionally a change in kitchen sampling procedure led to the loss of nearly 30% of kitchen samples and demonstrated the importance of accurately quantifying exposure.

Finally, air pollution levels in some study households may have contributed to some tubes reaching maximum stain length. Ideally, the tubes should be monitored after deployment to detect any high levels of exposure or other sampling problems (Dionisio et al. 2012) and replaced if the upper limits of detection is reached. However this was a hard feat to accomplish given substantial traveling distances to study households in the 51 community clusters.

CONCLUSION

After installation of study promoted chimney stoves in San Marcos, Cajamarca region, Peru, personal CO exposures and kitchen levels measured with passive diffusion tubes did not differ significantly between intervention and control households. Personal mother CO exposures were correlated with children's exposures. These results point to the fact that where data are unavailable, mothers' exposures can be used to predict children's exposures especially in high pollution settings. Results suggest that proper and exclusive chimney stove use, maintenance of stoves as well as changes to fuel types may be necessary in reducing CO and more generally, HAP exposures.

REFERENCES

Adetona, O., D. B. Hall, and L. P. Naeher. 2011. "Lung function changes in wildland firefighters working at prescribed burns." *Inhalation Toxicology* 23(13): 835-41.

Aggarwal, R. K. and S. S. Chandel. 2004. "Review of Improved Cookstoves Programme in Western Himalayan State of India." *Biomass and Bioenergy* 27(2): 131-44.

Alam, S., S. J. Chowdhury, A. Begum, and M. Rahman. 2006. "Effect of improved earthen stoves: improving health for rural communities in Bangladesh." *Energy for Sustainable Development* 10(3): 46-53.

Albalak, R., N. Bruce, J. P. McCracken, K. R. Smith, and T. De Gallardo. 2001. "Indoor respirable particulate matter concentrations from an open fire, improved cookstove, and LPG/open fire combination in a rural Guatemalan community." *Environmental science & technology* 35(13): 2650-55.

Allen, R. W., C. Carlsten, B. Karlen, S. Leckie, S. van Eeden, S. Vedal, I. Wong, and M. Brauer. 2011. "An air filter intervention study of endothelial function among healthy adults in a woodsmoke-impacted community." *American journal of respiratory and critical care medicine* 183(9): 1222-30.

Andreae, M. O. and P. Merlet. 2001. "Emission of trace gases and aerosols from biomass burning." *Global biogeochemical cycles* 15(4): 955-66.

Armendáriz-Arnez, C., R. D. Edwards, M. Johnson, I. A. Rosas, F. Espinosa, and O. R. Masera. 2010. "Indoor particle size distributions in homes with open fires and improved Patsari cook stoves." *Atmospheric Environment* 44(24): 2881-86.

Arnett, S. D., D. M. Osbourn, K. D. Moore, S. S. Vandaveer, and C. E. Lunte. 2005. "Determination of 8-oxoguanine and 8-hydroxy-2′-deoxyguanosine in the rat cerebral cortex using microdialysis sampling and capillary electrophoresis with electrochemical detection." *Journal of Chromatography B* 827(1): 16-25.

Avakian, M. D., B. Dellinger, H. Fiedler, B. Gullet, C. Koshland, S. Marklund, G. Oberdörster, S. Safe, A. Sarofim, and K. R. Smith. 2002. "The origin, fate, and health effects of combustion by-products: a research framework." *Environmental health perspectives* 110(11): 1155.

Balakrishnan, K., P. Ramaswamy, S. Sambandam, G. Thangavel, S. Ghosh, P. Johnson, K. Mukhopadhyay, V. Venugopal, and V. Thanasekaraan. 2011. "Air pollution from household solid fuel combustion in India: an overview of exposure and health related information to inform health research priorities." *Global health action* 4.

Balakrishnan, K., P. Ramaswamy, and S. Sankar. 2004. "Biomass Smoke and Health Risks–The Situation in Developing Countries." *Air Pollution*: 219-39.

Ballard-Tremeer, G. and H. Jawurek. 1996. "Comparison of five rural, wood-burning cooking devices: efficiencies and emissions." *Biomass and Bioenergy* 11(5): 419-30.

Banerjee, A., N. K. Mondal, D. Das, and M. R. Ray. 2011. "Neutrophilic inflammatory response and oxidative stress in premenopausal women chronically exposed to indoor air pollution from biomass burning." *Inflammation*: 1-13.

Barregard, L., G. Sällsten, L. Andersson, A. C. Almstrand, P. Gustafson, M. Andersson, and A. C. Olin. 2008. "Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress." *Occupational and Environmental Medicine* 65(5): 319.

Barregard, L., G. Sällsten, P. Gustafson, L. Andersson, L. Johansson, S. Basu, and L. Stigendal. 2006. "Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation." *Inhalation Toxicology* 18(11): 845-53.

Bates, E., N. Bruce, A. Doig, and S. Gitonga. 2002. "Participatory approaches for alleviating indoor air pollution in rural Kenyan kitchens." *Boiling point*: 12-15.

Bølling, A. K., J. Pagels, K. E. Yttri, L. Barregard, G. Sallsten, P. E. Schwarze, and C. Boman. 2009. "Health effects of residential wood smoke particles: the importance of combustion conditions and physicochemical particle properties." *Particle and fibre toxicology* 6(1): 29.

Bruce, N., J. McCracken, R. Albalak, M. Schei, K. R. Smith, V. Lopez, and C. West. 2004. "Impact of improved stoves, house construction and child location on levels of indoor air pollution exposure in young Guatemalan children." *Journal of Exposure Science and Environmental Epidemiology* 14: S26-S33.

Bruce, N. G. 2010. "Symposium: The RESPIRE randomized control trial: preventing child ALRI by reducing household air pollution and the implications for pneumonia prevention globally. Presented at the Health Effects Institute 2010 annual conference, April 27th 2010."

Bryce, J., C. Boschi-Pinto, K. Shibuya, and R. E. Black. 2005. "WHO estimates of the causes of death in children." *The Lancet* 365(9465): 1147-52.

Chengappa, C., R. Edwards, R. Bajpai, K. N. Shields, and K. R. Smith. 2007. "Impact of improved cookstoves on indoor air quality in the Bundelkhand region in India." *Energy for Sustainable Development* 11(2): 33-44.

Chuang, C. Y., C. C. Lee, Y. K. Chang, and F. C. Sung. 2003. "Oxidative DNA damage estimated by urinary 8-hydroxydeoxyguanosine: influence of taxi driving, smoking and areca chewing." *Chemosphere* 52(7): 1163-71.

Clark, M. L., J. L. Peel, J. B. Burch, T. L. Nelson, M. M. Robinson, S. Conway, A. M. Bachand, and S. J. Reynolds. 2009. "Impact of improved cookstoves on indoor air pollution and adverse health effects among Honduran women." *International Journal of Environmental Health Research* 19(5): 357-68.

Clark, M. L., S. J. Reynolds, J. B. Burch, S. Conway, A. M. Bachand, and J. L. Peel. 2010. "Indoor air pollution, cookstove quality, and housing characteristics in two Honduran communities." *Environmental research* 110(1): 12-18.

Cynthia, A. A., R. D. Edwards, M. Johnson, M. Zuk, L. Rojas, R. D. Jiménez, H. Riojas-Rodriguez, and O. Masera. 2008. "Reduction in personal exposures to particulate matter and carbon monoxide as a result of the installation of a Patsari improved cook stove in Michoacan Mexico." *Indoor air* 18(2): 93-105.

Danielsen, P. H., E. V. Bräuner, L. Barregard, G. Sällsten, M. Wallin, R. Olinski, R. Rozalski, P. Møller, and S. Loft. 2008. "Oxidatively damaged DNA and its repair after experimental exposure to wood smoke in healthy humans." *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis* 642(1-2): 37-42.

Danielsen, P. H., S. Loft, N. R. Jacobsen, K. A. Jensen, H. Autrup, J. L. Ravanat, H. Wallin, and P. Møller. 2010. "Oxidative stress, inflammation, and DNA damage in rats after intratracheal instillation or oral exposure to ambient air and wood smoke particulate matter." *Toxicological Sciences* 118(2): 574-85.

Danielsen, P. H., S. Loft, A. Kocbach, P. E. Schwarze, and P. Møller. 2009. "Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines." *Mutation Research/Genetic Toxicology and Environmental Mutagenesis* 674(1-2): 116-22.

Danielsen, P. H., P. Møller, K. A. Jensen, A. K. Sharma, H. Wallin, R. Bossi, H. Autrup, L. Mølhave, J. L. Ravanat, and J. J. Briedé. 2011. "Oxidative stress, DNA damage, and inflammation induced by ambient air and wood smoke particulate matter in human A549 and THP-1 cell lines." *Chemical Research in Toxicology*.

Dennis, R. J., D. Maldonado, S. Norman, E. Baena, and G. Martinez. 1996. "Woodsmoke exposure and risk for obstructive airways disease among women." *Chest* 109(1): 115-19.

Diaz, E., N. Bruce, D. Pope, A. Diaz, K. Smith, and T. Smith-Sivertsen. 2008. "Self-rated health among Mayan women participating in a randomised intervention trial reducing indoor air pollution in Guatemala." *BMC International Health and Human Rights* 8(1): 7.

Dionisio, K., S. Howie, K. Fornace, O. Chimah, R. Adegbola, and M. Ezzati. 2008. "Measuring the exposure of infants and children to indoor air pollution from biomass fuels in The Gambia." *Indoor air* 18(4): 317-27.

Dionisio, K. L., S. R. C. Howie, F. Dominici, K. M. Fornace, J. D. Spengler, S. Donkor, O. Chimah, C. Oluwalana, R. C. Ideh, and B. Ebruke. 2011. "The exposure of infants and children to carbon monoxide from biomass fuels in The Gambia: a measurement and modeling study." *Journal of Exposure Science and Environmental Epidemiology*.

Dionisio, K. L., S. R. C. Howie, F. Dominici, K. M. Fornace, J. D. Spengler, S. Donkor, O. Chimah, C. Oluwalana, R. C. Ideh, B. Ebruke, R. A. Adegbola, and M. Ezzati. 2012. "The exposure of infants and children to carbon monoxide from biomass fuels in The Gambia: a measurement and modeling study." *J Expos Sci Environ Epidemiol* 22(2): 173-81.

Dräger-Tubes & Chip-Measurement-System Handbook (PDF). 16th edition Soil, W., and Air Investigations as well as Technical Gas Analysis Carbon monoxide 50/a-D, 6733191.

Drost, E., K. Skwarski, J. Sauleda, N. Soler, J. Roca, A. Agusti, and W. MacNee. 2005. "Oxidative stress and airway inflammation in severe exacerbations of COPD." *Thorax* 60(4): 293.

Dutta, K., K. N. Shields, R. Edwards, and K. R. Smith. 2007. "Impact of improved biomass cookstoves on indoor air quality near Pune, India." *Energy for Sustainable Development* 11(2): 19-32.

Edwards, R. D., Y. Liu, G. He, Z. Yin, J. Sinton, J. Peabody, and K. Smith. 2007. "Household CO and PM measured as part of a review of China's National Improved Stove Program." *Indoor air* 17(3): 189-203.

Ekici, A., M. Ekici, E. Kurtipek, A. Akin, M. Arslan, T. Kara, Z. Apaydin, and S. Demir. 2005. "Obstructive airway diseases in women exposed to biomass smoke." *Environmental research* 99(1): 93-98.

El Tayeb Muneer, S. and E. W. Mukhtar Mohamed. 2003. "Adoption of biomass improved cookstoves in a patriarchal society: an example from Sudan." *Science of the Total Environment* 307(1): 259-66.

England, T., E. Beatty, A. Rehman, J. Nourooz-Zadeh, P. Pereira, J. O'Reilly, H. Wiseman, C. Geissler, and B. Halliwell. 2000. "The steady-state levels of oxidative DNA damage and of lipid peroxidation (F2-isoprostanes) are not correlated in healthy human subjects." *Free Radical Research* 32(4): 355-62.

Ezzati, M. and D. M. Kammen. 2002. "The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps, and data needs." *Environmental health perspectives* 110(11): 1057.

Fan, R., D. Wang, C. Mao, S. Ou, Z. Lian, S. Huang, Q. Lin, R. Ding, and J. She. 2011. "Preliminary study of children's exposure to PAHs and its association with 8-hydroxy-2'-deoxyguanosine in Guangzhou, China." *Environment international*.

Fitzgerald, C., M. Aguilar-Villalobos, A. R. Eppler, S. C. Dorner, S. L. Rathbun, and L. P. Naeher. 2012a. "Testing the effectiveness of two improved cookstove interventions in the Santiago de Chuco Province of Peru." *Science of the Total Environment* 420(0): 54-64.

Fitzgerald, C., M. Aguilar-Villalobos, A. R. Eppler, S. C. Dorner, S. L. Rathbun, and L. P. Naeher. 2012b. "Testing the effectiveness of two improved cookstove interventions in the Santiago de Chuco Province of Peru." *Science of the Total Environment*.

Fullerton, D. G., N. Bruce, and S. B. Gordon. 2008. "Indoor air pollution from biomass fuel smoke is a major health concern in the developing world." *Transactions of the Royal Society of Tropical Medicine and Hygiene* 102(9): 843-51.

GACC. 2011. "The Global Alliance for Clean Cookstoves: Overview [Document on the Internet] [updated 2011; cited 2011 Oct 23]. Available from http://cleancookstoves.org/."

Ghio, A. J., J. M. Soukup, M. Case, L. A. Dailey, J. Richards, J. Berntsen, R. B. Devlin, S. Stone, and A. Rappold. 2011. "Exposure to wood smoke particles produces inflammation in healthy volunteers." *Occupational and Environmental Medicine*.

Ghio, A. J., J. M. Soukup, M. Case, L. A. Dailey, J. Richards, J. Berntsen, R. B. Devlin, S. Stone, and A. Rappold. 2012. "Exposure to wood smoke particles produces inflammation in healthy volunteers." *Occupational and Environmental Medicine* 69(3): 170-75.

Gniwotta, C., J. D. Morrow, L. J. Roberts II, and H. Kühn. 1997. "Prostaglandin F2-like compounds, F2-isoprostanes, are present in increased amounts in human atherosclerotic lesions." *Arteriosclerosis, thrombosis, and vascular biology* 17(11): 3236-41.

Gopalan, H. N. B., S. Saksena, I. Tata Energy Research, and P. United Nations Environment. 1999. *Domestic environment and health of women and children*. New Delhi: Tata Energy Research Institute [and] United Nations Environment Programme, New Delhi, PNUMA, 253 p.

Hartinger, S., Commodore, AA, Hattendorf, J, Lanata, CF, Gil, AI, Verastegui, H, Aguilar-Villalobos, M, Mäusezahl, D, Naeher, LP. in review. "Chimney stoves in an intervention study do not improve household air pollution when compared with traditional open fire stoves in rural Peru." *Indoor air*.

Hartinger, S., C. Lanata, J. Hattendorf, A. Gil, H. Verastegui, T. Ochoa, and D. Mäusezahl. 2011. "A community randomised controlled trial evaluating a home-based environmental intervention package of improved stoves, solar water disinfection and kitchen sinks in rural Peru: Rationale, trial design and baseline findings." *Contemporary Clinical Trials* 32(6): 864-73.

Hartinger, S., Lanata, CF, Gil, AI, Hattendorf, J, Verastegui, H, Mäusezahl, D. . 2012. "Combining interventions: improved stove, kitchen sinks and solar disinfection of drinking water and kitchen cloths to improve hygiene in rural Peru." *Field Actions Science Reports [Online], Special Issue 6 | 2012, Online since 31 May 2012, Connection on 24 September 2012. URL : http://factsreports.revues.org/1627*.

Hartinger SM, L. C., Gil, AI, Hattendorf J, Verastegui H, Mäusezahl, D "Combining interventions: improved stove, kitchen sinks and solar disinfection of drinking water and kitchen cloths to improve hygiene in rural Peru." *Field Actions Science Reports [Online], Special Issue 6*

| *2012, Online since 31 May 2012, Connection on 24 September 2012. URL : http://factsreports.revues.org/1627*.

Henkle, J., C. Mandzuk, E. Emery, L. Schrowe, and J. Sevilla-Martir. 2010. "Global Health and International Medicine: Honduras Stove Project." *Hispanic Health Care International* 8(1): 36-46.

Hosgood, H. D., H. Wei, A. Sapkota, I. Choudhury, N. Bruce, K. R. Smith, N. Rothman, and Q. Lan. 2011. "Household coal use and lung cancer: systematic review and meta-analysis of case–control studies, with an emphasis on geographic variation." *International Journal of Epidemiology* 40(3): 719-28.

IARC. 2010. "Household use of solid fuels and high-temperature frying." *IARC Monogr Eval Carcinog Risks Hum.* 95: 1-430.

INEI. 2007. "Instituto Nacional de Estadística e Informática[Document on the Internet] [updated 2007; cited 2012 May 10]. Available from http://www.inei.gob.pe."

Jalava, P. I., R. O. Salonen, K. Nuutinen, A. S. Pennanen, M. S. Happo, J. Tissari, A. Frey, R. Hillamo, J. Jokiniemi, and M. R. Hirvonen. 2010. "Effect of combustion condition on cytotoxic and inflammatory activity of residential wood combustion particles." *Atmospheric Environment* 44(13): 1691-98.

Khushk, W., Z. Fatmi, F. White, and M. Kadir. 2005. "Health and social impacts of improved stoves on rural women: a pilot intervention in Sindh, Pakistan." *Indoor air* 15(5): 311-16.

Kim, J. Y., S. Mukherjee, L. C. Ngo, and D. C. Christiani. 2004. "Urinary 8-hydroxy-2'-deoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to fine particulates." *Environmental health perspectives* 112(6): 666.

Kim, K. H., S. A. Jahan, and E. Kabir. 2011a. "A review of diseases associated with household air pollution due to the use of biomass fuels." *Journal of hazardous materials* 192(2): 425-31.

Kim, K. H., S. A. Jahan, and E. Kabir. 2011b. "A review of diseases associated with household air pollution due to the use of biomass fuels." *Journal of hazardous materials*.

Kimura, S., H. Yamauchi, Y. Hibino, M. Iwamoto, K. Sera, and K. Ogino. 2006. "Evaluation of urinary 8-hydroxydeoxyguanine in healthy Japanese people." *Basic & Clinical Pharmacology & Toxicology* 98(5): 496-502.

Kinnula, V. L., H. Ilumets, M. Myllärniemi, A. Sovijärvi, and P. Rytilä. 2007. "8-Isoprostane as a marker of oxidative stress in nonsymptomatic cigarette smokers and COPD." *European Respiratory Journal* 29(1): 51-55.

Kipen, H., D. Rich, W. Huang, T. Zhu, G. Wang, M. Hu, S. Lu, P. Ohman-Strickland, P. Zhu, and Y. Wang. 2010. "Measurement of inflammation and oxidative stress following drastic

changes in air pollution during the Beijing Olympics: a panel study approach." *Annals of the New York Academy of Sciences* 1203(1): 160-67.

Kocbach, A., J. I. Herseth, M. Låg, M. Refsnes, and P. E. Schwarze. 2008a. "Particles from wood smoke and traffic induce differential pro-inflammatory response patterns in co-cultures." *Toxicology and applied pharmacology* 232(2): 317-26.

Kocbach, A., E. Namork, and P. E. Schwarze. 2008b. "Pro-inflammatory potential of wood smoke and traffic-derived particles in a monocytic cell line." *Toxicology* 247(2-3): 123-32.

Kurmi, O. P., S. Semple, P. Simkhada, W. C. S. Smith, and J. G. Ayres. 2010. "COPD and chronic bronchitis risk of indoor air pollution from solid fuel: a systematic review and meta-analysis." *Thorax* 65(3): 221-28.

Lai, C., S. Liou, H. Lin, T. Shih, P. Tsai, J. Chen, T. Yang, J. Jaakkola, and P. Strickland. 2005. "Exposure to traffic exhausts and oxidative DNA damage." *Occupational and Environmental Medicine* 62(4): 216-22.

Lai, C. H., S. H. Liou, J. J. K. Jaakkola, H. B. Huang, T. Y. Su, and P. T. Strickland. 2012. "Exposure to Polycyclic Aromatic Hydrocarbon Associated with Traffic Exhaust Increases Lipid Peroxidation and Reduces Antioxidant Capacity."

Lee, M.-W., M.-L. Chen, S.-C. C. Lung, C.-J. Tsai, X.-J. Yin, and I. F. Mao. 2010. "Exposure assessment of PM2.5 and urinary 8-OHdG for diesel exhaust emission inspector." *Science of the Total Environment* 408(3): 505-10.

Lewtas, J. 2007. "Air pollution combustion emissions: characterization of causative agents and mechanisms associated with cancer, reproductive, and cardiovascular effects." *Mutation Research/Reviews in Mutation Research* 636(1-3): 95-133.

Li, C., S. Kang, P. Chen, Q. Zhang, J. Guo, J. Mi, P. Basang, Q. Luosang, and K. R. Smith. 2012a. "Personal PM< sub> 2.5</sub> and indoor CO in nomadic tents using open and chimney biomass stoves on the Tibetan Plateau." *Atmospheric Environment*.

Li, N., T. Xia, and A. E. Nel. 2008. "The role of oxidative stress in ambient particulate matter-induced lung diseases and its implications in the toxicity of engineered nanoparticles." *Free Radical Biology and Medicine* 44(9): 1689-99.

Li, Z., A. Commodore, S. Hartinger, E. Pittman, D. Trinidad, R. Ostovar, K. Hubbard, A. Sjodin, C. Lanata, A. Gil, D. Mäusezahl, and L. Naeher. 2012b. "O-046: Assessment of Exposure to PAHs in Cook Stove Intervention Projects in Peru by Urinary Biomonitoring." *Epidemiology* 23(5S): 10.1097/01.ede.0000416704.62764.3d.

Li, Z., L. C. Romanoff, A. Debra, N. Hussain, R. S. Jones, E. N. Porter, D. G. Patterson Jr, and A. Sjödin. 2006. "Measurement of urinary monohydroxy polycyclic aromatic hydrocarbons using automated liquid-liquid extraction and gas chromatography/isotope dilution high-resolution mass spectrometry." *Analytical chemistry* 78(16): 5744-51.

Li, Z., A. Sjödin, L. C. Romanoff, K. Horton, C. L. Fitzgerald, A. Eppler, M. Aguilar-Villalobos, and L. P. Naeher. 2011. "Evaluation of exposure reduction to indoor air pollution in stove intervention projects in Peru by urinary biomonitoring of polycyclic aromatic hydrocarbon metabolites." *Environment international* 37(7): 1157-63.

Limmeechokchai, B. and S. Chawana. 2007. "Sustainable energy development strategies in the rural Thailand: The case of the improved cooking stove and the small biogas digester." *Renewable and Sustainable Energy Reviews* 11(5): 818-37.

Ling, S. H. and S. F. Van Eeden. 2009. "Particulate matter air pollution exposure: role in the development and exacerbation of chronic obstructive pulmonary disease." *International journal of chronic obstructive pulmonary disease* 4: 233.

Lisouza, F. A., O. P. Owuor, and J. O. Lalah. 2011. "Variation in indoor levels of polycyclic aromatic hydrocarbons from burning various biomass types in the traditional grass-roofed households in Western Kenya." *Environmental Pollution* 159(7): 1810-15.

Liu, S., Y. Zhou, X. Wang, D. Wang, J. Lu, J. Zheng, N. Zhong, and P. Ran. 2007. "Biomass fuels are the probable risk factor for chronic obstructive pulmonary disease in rural South China." *Thorax* 62(10): 889-97.

Lodovici, M., C. Casalini, R. Cariaggi, L. Michelucci, and P. Dolara. 2000. "Levels of 8-hydroxydeoxyguanosine as a marker of DNA damage in human leukocytes." *Free Radical Biology and Medicine* 28(1): 13-17.

Loft, S., K. Vistisen, M. Ewertz, A. Tjønneland, K. Overvad, and H. E. Poulsen. 1992. "Oxidative DNA damage estimated by 8-hydroxydeoxyguanosine excretion in humans: influence of smoking, gender and body mass index." *Carcinogenesis* 13(12): 2241.

Lokras, S. 2012. "Development and Dissemination of Fuel-Efficient Biomass Burning Devices." *Journal of the Indian Institute of Science* 92(1): 99-110.

Lu, C. Y., Y. C. Ma, J. M. Lin, C. Y. Chuang, and F. C. Sung. 2007. "Oxidative DNA damage estimated by urinary 8-hydroxydeoxyguanosine and indoor air pollution among non-smoking office employees." *Environmental research* 103(3): 331-37.

Martin, W. J., R. I. Glass, J. M. Balbus, and F. S. Collins. 2011. "A Major Environmental Cause of Death." *Science* 334(6053): 180-81.

Masera, O., R. Edwards, C. A. Arnez, V. Berrueta, M. Johnson, L. R. Bracho, H. Riojas-Rodríguez, and K. R. Smith. 2007. "Impact of Patsari improved cookstoves on indoor air quality in Michoacán, Mexico." *Energy for Sustainable Development* 11(2): 45-56.

McCracken, J., K. R. Smith, P. Stone, A. Díaz, B. Arana, and J. Schwartz. 2011. "Intervention to Lower Household Wood Smoke Exposure in Guatemala Reduces ST-Segment Depression on Electrocardiograms." *Environmental health perspectives* 119(11): 1562.

McCracken, J. P., J. Schwartz, N. Bruce, M. Mittleman, L. M. Ryan, and K. R. Smith. 2009. "Combining individual-and group-level exposure information: child carbon monoxide in the Guatemala woodstove randomized control trial." *Epidemiology* 20(1): 127.

McCracken, J. P. and K. R. Smith. 1998. "Emissions and efficiency of improved woodburning cookstoves in Highland Gatemala." *Environment international* 24(7): 739-47.

McCracken, J. P., K. R. Smith, A. Díaz, M. A. Mittleman, and J. Schwartz. 2007. "Chimney stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women." *Environmental health perspectives* 115(7): 996.

Montuschi, P., M. Corradi, G. CIABATTONI, J. Nightingale, S. A. Kharitonov, and P. J. Barnes. 1999. "Increased 8-isoprostane, a marker of oxidative stress, in exhaled condensate of asthma patients." *American journal of respiratory and critical care medicine* 160(1): 216-20.

Naeher, L., K. Smith, B. Leaderer, L. Neufeld, and D. Mage. 2001. "Carbon monoxide as a tracer for assessing exposures to particulate matter in wood and gas cookstove households of highland Guatemala." *Environmental science & technology* 35(3): 575-81.

Naeher, L. P., M. Brauer, M. Lipsett, J. T. Zelikoff, C. D. Simpson, J. Q. Koenig, and K. R. Smith. 2007. "Woodsmoke Health Effects: A Review." *Inhalation Toxicology* 19(1): 67-106.

Nazmul Alam, S. and S. J. Chowdhury. 2010. "Improved earthen stoves in coastal areas in Bangladesh: Economic, ecological and socio-cultural evaluation." *Biomass and Bioenergy* 34(12): 1954-60.

Northcross, A., Z. Chowdhury, J. McCracken, E. Canuz, and K. R. Smith. 2010. "Estimating personal PM2. 5 exposures using CO measurements in Guatemalan households cooking with wood fuel." *J. Environ. Monit.* 12(4): 873-78.

Nuernberg, A. M., P. D. Boyce, J. M. Cavallari, S. C. Fang, E. A. Eisen, and D. C. Christiani. 2008. "Urinary 8-isoprostane and 8-OHdG concentrations in boilermakers with welding exposure." *Journal of occupational and environmental medicine* 50(2): 182.

Oanh, N. T. K., L. B. Reutergårdh, and N. T. Dung. 1999. "Emission of polycyclic aromatic hydrocarbons and particulate matter from domestic combustion of selected fuels." *Environmental science & technology* 33(16): 2703-09.

Orozco-Levi, M., J. Garcia-Aymerich, J. Villar, A. Ramirez-Sarmiento, J. Anto, and J. Gea. 2006. "Wood smoke exposure and risk of chronic obstructive pulmonary disease." *European Respiratory Journal* 27(3): 542-46.

Palmer, K., R. McNeill-Love, J. Poole, D. Coggon, A. Frew, C. Linaker, and J. Shute. 2006. "Inflammatory responses to the occupational inhalation of metal fume." *European Respiratory Journal* 27(2): 366-73.

Patel, P. R., R. J. Bevan, N. Mistry, and J. Lunec. 2007. "Evidence of oligonucleotides containing 8-hydroxy-2′-deoxyguanosine in human urine." *Free Radical Biology and Medicine* 42(4): 552-58.

Pennise, D., S. Brant, S. M. Agbeve, W. Quaye, F. Mengesha, W. Tadele, and T. Wofchuck. 2009. "Indoor air quality impacts of an improved wood stove in Ghana and an ethanol stove in Ethiopia." *Energy for Sustainable Development* 13(2): 71-76.

Perez-Padilla, R., A. Schilmann, and H. Riojas-Rodriguez. 2010. "Respiratory health effects of indoor air pollution [Review article]." *The International Journal of Tuberculosis and Lung Disease* 14(9): 1079-86.

Peterson, J., M. MacDonell, L. Haroun, F. Monette, R. D. Hildebrand, and A. Taboas. 2007. "Radiological and chemical fact sheets to support health risk analyses for contaminated areas." *Human Health Fact Sheet, Argonne*: 38-39.

Pilger, A., D. Germadnik, K. Riedel, I. Meger-Kossien, G. Scherer, and H. W. Rüdiger. 2001. "Longitudinal study of urinary 8-hydroxy-2'-deoxyguanosine excretion in healthy adults." *Free Radical Research* 35(3): 273-80.

Pilger, A., S. Ivancsits, D. Germadnik, and H. Rudiger. 2002. "Urinary excretion of 8-hydroxy-2'-deoxyguanosine measured by high-performance liquid chromatography with electrochemical detection." *Journal of Chromatography B* 778(1-2): 393-401.

Pope III, C. A., R. T. Burnett, M. C. Turner, A. Cohen, D. Krewski, M. Jerrett, S. M. Gapstur, and M. J. Thun. 2011. "Lung Cancer and Cardiovascular Disease Mortality Associated with Ambient Air Pollution and Cigarette Smoke: Shape of the Exposure–Response Relationships." *Environmental health perspectives* 119(11): 1616.

Regalado, J., R. Pérez-Padilla, R. Sansores, J. I. P. Ramirez, M. Brauer, P. Paré, and S. Vedal. 2006. "The effect of biomass burning on respiratory symptoms and lung function in rural Mexican women." *American journal of respiratory and critical care medicine* 174(8): 901-05.

Rehfuess, E., S. Mehta, and A. Prüss-Üstün. 2006. "Assessing household solid fuel use: multiple implications for the Millennium Development Goals." *Environmental health perspectives* 114(3): 373.

Roberts II, L. J. and J. D. Morrow. 2000. "Measurement of F2-isoprostanes as an index of oxidative stress in vivo." *Free Radical Biology and Medicine* 28(4): 505-13.

Romieu, I., H. Riojas-Rodriguez, A. T. Marron-Mares, A. Schilmann, R. Perez-Padilla, and O. Masera. 2009. "Improved biomass stove intervention in rural Mexico: impact on the respiratory health of women." *American journal of respiratory and critical care medicine*: 200810-1556OCv1.

Saha, A., P. Kulkarni, A. Shah, M. Patel, and H. Saiyed. 2005. "Ocular morbidity and fuel use: an experience from India." *Occupational and Environmental Medicine* 62(1): 66.

Sakano, N., D. H. Wang, N. Takahashi, B. Wang, R. Sauriasari, S. Kanbara, Y. Sato, T. Takigawa, J. Takaki, and K. Ogino. 2009. "Oxidative stress biomarkers and lifestyles in Japanese healthy people." *Journal of clinical biochemistry and nutrition* 44(2): 185.

Saksena, S., R. Prasad, R. Pal, and V. Joshi. 1992. "Patterns of daily exposure to TSP and CO in the Garhwal Himalaya." *Atmospheric Environment. Part A. General Topics* 26(11): 2125-34.

Smith, K. R. 1987. *Biofuels, air pollution, and health: a global review*: Plenum press.

Smith, K. R., J. P. McCracken, L. Thompson, R. Edwards, K. N. Shields, E. Canuz, and N. Bruce. 2010. "Personal child and mother carbon monoxide exposures and kitchen levels: Methods and results from a randomized trial of woodfired chimney cookstoves in Guatemala (RESPIRE)." *J Expos Sci Environ Epidemiol* 20(5): 406-16.

Smith, K. R., J. P. McCracken, M. W. Weber, A. Hubbard, A. Jenny, L. M. Thompson, J. Balmes, A. Diaz, B. Arana, and N. Bruce. 2011. "Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial." *The Lancet* 378(9804): 1717-26.

Smith, K. R. and S. Mehta. 2003. "The burden of disease from indoor air pollution in developing countries: comparison of estimates." *International Journal of Hygiene and Environmental Health* 206(4-5): 279-89.

Smith, K. R. and J. L. Peel. 2010. "Mind the gap." *Environmental health perspectives* 118(12): 1643.

Smith, K. R., J. M. Samet, I. Romieu, and N. Bruce. 2000. "Indoor air pollution in developing countries and acute lower respiratory infections in children." *Thorax* 55(6): 518-32.

Swiston, J. R., W. Davidson, S. Attridge, G. T. Li, M. Brauer, and S. F. van Eeden. 2008. "Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters." *European Respiratory Journal* 32(1): 129-38.

Tamura, S., H. Tsukahara, M. Ueno, M. Maeda, H. Kawakami, K. Sekine, and M. Mayumi. 2006. "Evaluation of a urinary multi-parameter biomarker set for oxidative stress in children, adolescents and young adults." *Free Radical Research* 40(11): 1198-205.

Thompson, L. M., N. Bruce, B. Eskenazi, A. Diaz, D. Pope, and K. R. Smith. 2011. "Impact of Reduced Maternal Exposures to Wood Smoke from an Introduced Chimney Stove on Newborn Birth Weight in Rural Guatemala." *Environmental health perspectives* 119(10): 1489.

Tuohy, K. M., D. J. S. Hinton, S. J. Davies, M. J. C. Crabbe, G. R. Gibson, and J. M. Ames. 2006. "Metabolism of Maillard reaction products by the human gut microbiota–implications for health." *Molecular nutrition & food research* 50(9): 847-57.

Van Eeden, S., J. Leipsic, S. F. P. Man, and D. D. Sin. 2012. "The relationship between lung inflammation and cardiovascular disease." *American journal of respiratory and critical care medicine* 186(1): 11-16.

van Eeden, S. F. and D. D. Sin. 2008. "Chronic obstructive pulmonary disease: a chronic systemic inflammatory disease." *Respiration* 75(2): 224-38.

Venkataraman, C., A. Sagar, G. Habib, N. Lam, and K. Smith. 2010. "The Indian national initiative for advanced biomass cookstoves: the benefits of clean combustion." *Energy for Sustainable Development* 14(2): 63-72.

Wang, Z., D. Neuburg, C. Li, L. Su, J. Y. Kim, J. C. Chen, and D. C. Christiani. 2005. "Global gene expression profiling in whole-blood samples from individuals exposed to metal fumes." *Environmental health perspectives* 113(2): 233.

WHO. 2011. "Indoor air pollution and health; fact sheet 292.".

Wu, L. L., C.-C. Chiou, P.-Y. Chang, and J. T. Wu. 2004. "Urinary 8-OHdG: a marker of oxidative stress to DNA and a risk factor for cancer, atherosclerosis and diabetics." *Clinica Chimica Acta* 339(1-2): 1-9.

Y von Schirnding, N. B., K Smith, G Ballard-Tremeer, M Ezzati, K Lvovsky. 2002. *Addressing the Impact of Household Energy and Indoor Air Pollution on the Health of Poor: Implications for Policy Action and Intervention Measures—paper prepared for the Commission on Macroeconomics and HealthWHO, Geneva (2002)*: World Health Organization.

Zhang, J. J. and K. R. Smith. 2007. "Household air pollution from coal and biomass fuels in China: measurements, health impacts, and interventions." *Environmental health perspectives* 115(6): 848.

Table 5.1. Description of passive diffusion CO tubes deployed in intervention and control households during household air pollution exposure assessment in rural Peru

	Intervention Homes			**Control Homes**		
	Child	Mother	Kitchen	Child	Mother	Kitchen
Total number of tubes	172	182	182	173	197	197
Broken	9	5	0	10	18	2
Lost	5	14	1	11	17	0
Unavailable during pickup	4	2	2	4	8	4
Sampling error	0	0	55	0	0	42
Tubes with stains	0	0	11	0	0	29
Final sample size	154	161	113	148	154	120

Table 5.2. Demographic and household information by stove type for study households in rural Peru

Characteristic	Stove type*				
	OPTIMA FL-I†(n=92)	OPTIMA FL-II† (n=64)	NGO‡ (n=25)	Self-improved by household (n=21)	Traditional (n=96)
Mothers' demography					
Mean age (SD), in years	30 (7.1)	31 (9.3)	31 (8.0)	27 (7.4)	29 (6.6)
Mean time spent playing with child (SD), in hours	0.9 (0.8)	0.8 (0.6)	0.7 (0.4)	0.7 (0.5)	0.8 (0.6)
Mean cooking time (SD), in hours	3 (1.0)	3 (0.9)	3 (0.6)	3 (1.0)	3 (1.1)
Household characteristics					
Mean number of people in household (SD)	5 (1.5)	5 (1.8)	5 (1.4)	4 (1.3)	5 (1.5)
Number of households with smokers n (%)	6 (7%)	4 (7%)	2 (8%)	4 (19%)	8 (8%)
Own a television set n (%)	22 (29%)	7 (17%)	1 (5%)	1 (6%)	25 (30%)
Kitchen characteristics					
Length of stove use n (%)					
<1 year	92 (100%)	64 (100%)	9 (53%)	11 (69%)	16 (21%)
1 - 2 years	0	0	5 (29%)	1 (6%)	15 (20%)
3 - 5 years	0	0	2 (12%)	3 (19%)	16 (21%)
>5 years	0	0	1 (6%)	1 (6%)	29 (38%)
Most common wood type n (%)					
Eucalypto	32 (34%)	24 (41%)	9 (39%)	13 (65%)	38 (40%)
Kitchen environment: number of kitchen walls n (%)					
Four walls	69 (75%)	40 (63%)	18 (72%)	17 (85%)	77 (88%)

*Total sample sizes for number of people in the household, mother's age, cooking time and time spent playing with child for the various stove types are as follows: OPTIMA FL-I (n=92), OPTIMA FL-II (n=64), NGO (n=25), self-improved by household (n=21) and traditional (n=96). For all other variables, the sample sizes and percentage reflect the total number of responders for each stove category only

†Functionality level (FL) I refers to an OPTIMA stove in good conditions, and FL-II refers to an OPTIMA stove in need of repairs (eg re-plastering).

‡ NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO and ADIAR are NGOs that work in nearby communities.

Table 5.3. 48hr Unadjusted personal CO exposures and kitchen CO concentrations measured in all control and intervention homes

Stove type	Statistics	Child	Mother	Kitchen
Intervention	GM§ (95% CI) ppm	1.0 (0.9, 1.1)	1.4 (1.3, 1.6)	7.3 (6.4, 8.2)
	n	154	161	113
OPTIMA FL-I*	GM (95% CI) ppm	1.0 (0.6, 1.4)	1.5 (1.1, 1.9)	7.2 (6.6, 7.8)
	n	93	97	67
OPTIMA FL-II*	GM (95% CI) ppm	1.1 (0.8, 1.5)	1.5 (1.1, 2.0)	7.4 (6.2, 8.9)
	n	61	64	46
Control†	GM (95% CI) ppm	1.1 (0.9, 1.2)	1.4 (1.3, 1.6)	7.3 (6.4, 8.3)
	n	148	154	120
NGO‡	GM (95% CI) ppm	1.1 (0.3, 1.9)	1.5 (0.7, 2.3)	6.3 (5.3, 7.3)
	n	25	25	18
Gas	GM (95% CI) ppm	0.8 (0, 3.5)	0.9 (0, 3.8)	4.0 (0, 9.4)
	n	4	4	4
Self-improved by household	GM (95% CI) ppm	0.7 (0, 1.7)	1.2 (0.3, 2.1)	7.0 (5.8, 8.2)
	n	21	23	17
Traditional	GM (95% CI) ppm	1.1 (0.7, 1.5)	1.5 (1.1, 1.9)	7.6 (7.1, 8.1)
	n	98	102	81

*Functionality level (FL) I refers to an OPTIMA stove in good conditions, and FL-II refers to an OPTIMA stove in need of repairs (eg re-plastering).

† Geometric mean for all control stoves does not include gas stoves

‡NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO and ADIAR are NGOs that work in nearby communities.

§GM refer to geometric mean

Sample sizes represent the total number of subjects from whom CO measurements were taken. For mothers n=154 and 161, and for children n=148 and 154 in control and intervention homes respectively

Table 5.4. Effects of all variables in the full model for each sample type. Test statistics and P values for modeled effects are provided for personal exposures and kitchen concentrations of logCO. All covariates listed in the table were included in an initial model and then backward elimination was used to arrive at the final model for each sample type.

Variable	**Child**		**Mother**		**Kitchen**	
	F test statistic	P value	F test statistic	P value	Chi square statistic	P value
Mother's age	0.70	0.41	1.22	0.27	0.05	0.82
Time spent playing with child	1.50	0.22	0.26	0.61	0.43	0.51
Cooking time	4.57	**0.03**	6.04	**0.02**	3.82	**0.05**
Number of people in household	0.22	0.64	0.84	0.36	1.39	0.24
Presence of smoker in household	0.02	0.90	0.07	0.80	0.10	0.75
Age of stove	0.06	0.81	2.06	0.15	0.03	0.86
Wood type	0.26	0.77	0.34	0.71	1.83	0.40
Kitchen environment	1.54	0.22	2.31	**0.04**	9.26	0.16
Mother's frequency of cleaning ashes from stove	0.55	0.70	0.76	0.55	1.82	0.77
Distance of household to road	0.33	0.92	1.26	0.28	1.91	0.93
Stove type	0.57	0.68	0.70	0.60	2.75	0.60

Table 5.5. Model derived analysis of variance and geometric means (with 95% confidence intervals) for 48hr time integrated personal CO exposures and kitchen concentrations.

a. Mothers' personal CO exposures

Variable		Num DF, Dem Df§	F statistic	P value
Stove type		4, 285	0.24	0.92
Variable	n	Mean CO (ppm)	95% CI (ppm)	Ho: mean = OPTIMA I‖
OPTIMA FL-I*	92	1.5	1.3, 1.7	-
OPTIMA FL-II*	59	1.5	1.2, 1.8	1.00
NGO†	23	1.5	1.1, 2.0	1.00
Self-improved by household	20	1.3	0.9, 1.7	0.85
Traditional	96	1.5	1.3, 1.7	1.00

b. Children's personal CO exposures

Variable		Num DF, Dem Df	F statistic	P value
Stove type		4, 281	1.67	0.16
Cooking time‡		1, 281	3.86	**0.05**
Variable	n	Mean CO (ppm)	95% CI (ppm)	Ho: mean = OPTIMA I
OPTIMA FL-I*	92	1.0	0.9, 1.2	-
OPTIMA FL-II*	59	1.0	0.8, 1.2	1.00
NGO†	22	1.2	0.9, 1.6	0.79
Self-improved by household	19	0.7	0.5, 1.0	0.34
Traditional	95	1.2	1.0, 1.3	0.56

c. Kitchen CO concentrations

Variable		Df§	Chi-Square (X^2)	P value
Stove type		4	1.68	0.79
Wood type		2	5.52	0.06
Variable	**n**	**Mean CO (ppm)‖**	**95% CI (ppm)**	**P value**
Stove (reference = OPTIMA FL-I*)	67	7.2	6.1, 8.5	-
OPTIMA FL-II*	46	7.4	6.2, 8.9	0.81
NGO†	18	6.3	4.7, 8.5	0.55
Self-improved by household	17	7.0	4.9, 10.2	0.53
Traditional	81	7.6	6.5, 8.9	0.43
Wood type (reference = other types of wood‡)	77	6.7	5.6, 7.8	-
Eucalypto (Eucalyptus sp)	82	7.4	6.4, 8.6	0.22
Hualango (Acacia sp)	68	8.4	7.2, 9.8	**0.02**

*Functionality level (FL) I refers to an OPTIMA stove in good conditions, and FL-II refers to an OPTIMA stove in need of repairs (eg re-plastering).

†NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO and ADIAR are NGOs that work in nearby communities.

‡Cooking time refers to the estimated cooking time of mothers in study region that have been centered by subtracting the mean cooking time from individual cooking times

§Num DF and Dem DF refer to numerator and denominator degrees of freedom respectively

‖Ho: mean = OPTIMA FL-I refers to the probability of the mean personal CO exposure from other stove type = OPTIMA FL-I stove users mean CO exposure using Dunnett's test

- denotes nonapplicable results

Sample size represents subjects who had complete questionnaire data on wood type and stove type only rather than the total number of subjects from whom CO measurements were taken. For mothers n=151 and 139, and for children n=151 and 136 in control and intervention homes respectively

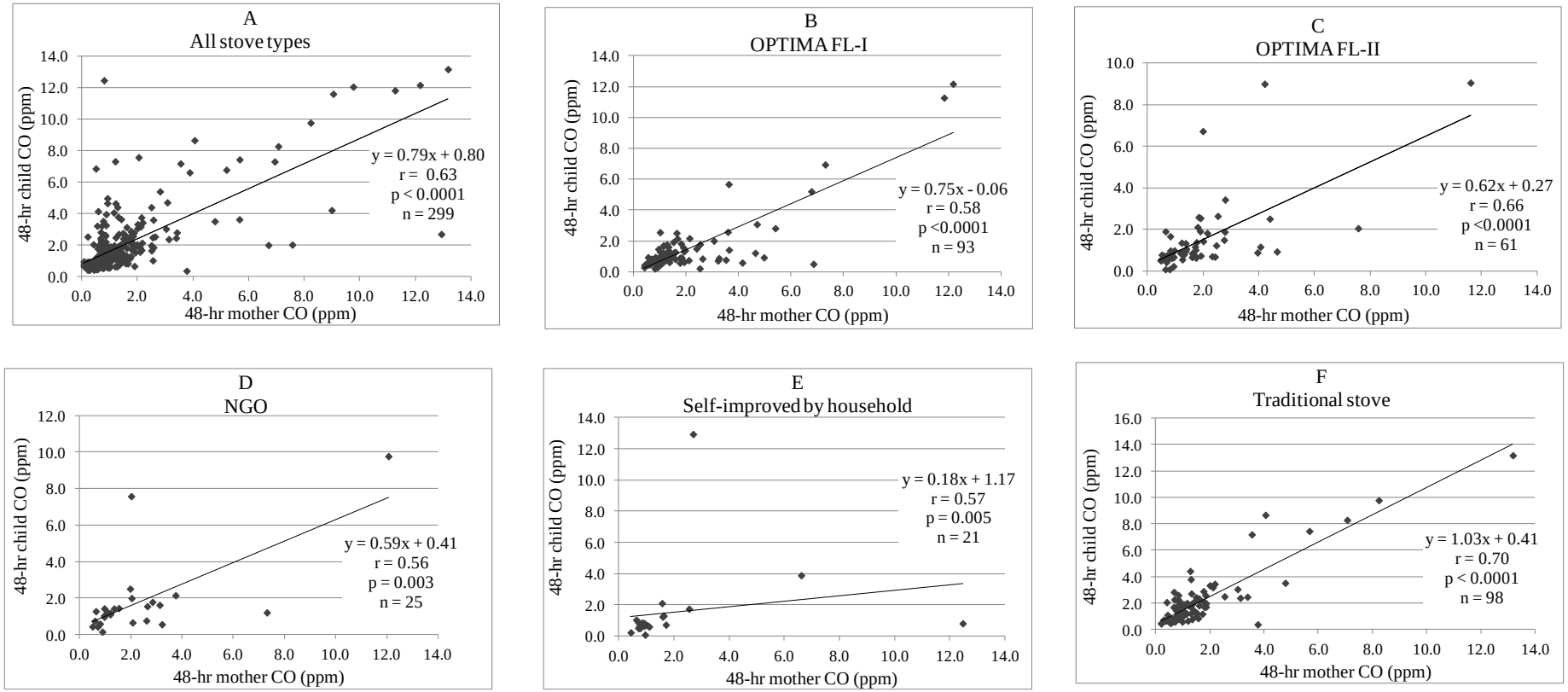


Figure 5.1.

Spearman correlation coefficients (r) between personal mother and child CO exposures for all intervention and control households (A). Then separate plots are presented by stove type: OPTIMA FL-I (B), OPTIMA FL-II (C), NGO (D), self-improved by household (E) and traditional (F) stoves.

CHAPTER 6

CONCENTRATIONS OF URINARY 8-OHDG AND 8-ISOPROSTANE IN WOMEN EXPOSED TO WOODSMOKE IN A COOKSTOVE INTERVENTION STUDY IN SAN MARCOS, PERU[1]

[1] Commodore, A. A., Zhang, J., Hartinger, S.M., Lanata, C.F., Mäusezahl, D., Gil, A.I., Hall, D.B., Aguilar-Villalobos, M., Vena, J.E., Wang, JS., Naeher, L.P. To be submitted to *Environment International*.

ABSTRACT

Background: Nearly half of the world's population is exposed to household air pollution (HAP) due to long hours spent in close proximity to unvented cooking fires. We aimed to investigate whether woodsmoke exposure induces oxidative stress by examining the relationship between woodsmoke exposure and biomarkers of DNA and lipid oxidation among control and intervention stove users.

Methods: The first morning voids after 48hr HAP exposure assessment were taken from 45 control stove users and 39 intervention stove users in San Marcos, Cajamarca region, Peru and analyzed for 8-OHdG and 8-isoprostane. Control households used a number of stoves including open fire and chimney stoves while intervention households used study-promoted OPTIMA chimney stoves.

Results: Creatinine corrected urinary oxidative stress biomarkers ranged from 11.2 to 2270.0 ug/g creatinine (median: 132.6 ug/g creatinine) for 8-OHdG and from 0.1 to 4.5 ug/g creatinine (median: 0.8 ug/g creatinine) for 8-isoprostane among all study subjects (n=84). There were no statistically significant differences in urinary oxidative stress biomarkers between the two arms of the intervention. Mean (95% CI) urinary 8-OHdG and 8-isoprostane levels among intervention stove users were 132.9 (97.7, 180.8) ug/g creatinine and 0.8 (0.6, 1.1) ug/g creatinine respectively. Likewise, among control stove users, these levels were 139.3 (108.7, 178.4) ug/g creatinine and 0.7 (0.6, 0.9) ug/g creatinine respectively. After controlling for the effects of traffic in the community and eating food exposed to fire, cooking time was positively correlated with urinary 8-OHdG (correlation coefficient (r)=0.29, p=0.01, n=80). Subjects' real time personal CO exposures were negatively correlated with 8-OHdG, particularly the maximum 30 second CO exposure during the sampling period (r=-0.32, p=0.001, n=73). After controlling

for the effect of distance of road to subjects' homes, 1-hyroxyphenanthrene and 2-hyroxyphenanthrene were both positively correlated with 8-isoprostane (r for both hydroxy-PAH biomarkers=0.21, p=0.05 and 0.06 respectively, n=84). 48hr time integrated personal $PM_{2.5}$ was negatively, but marginally correlated with urinary 8-isoprostane after controlling for the effect of distance of homes to the road (r=-0.21, p=0.09, n=69).

Conclusion: Both urinary oxidative stress biomarkers did not differ significantly between the two arms of the stove intervention. This cross sectional study conducted within the framework of a community based randomized control trial shows relatively high levels of urinary 8-OHdG compared to data in the literature for 8-OHdG excretion. Results suggest that there is a sustained systemic oxidative stress status among these women.

INTRODUCTION

The use of solid fuels occurs mostly in the developing world where wood and crop residues are employed by households for cooking and heating (Smith and Mehta 2003). These fuels are used in improperly vented stoves and this creates high levels of household air pollution (Rehfuess et al. 2006). Biomass combustion in the indoor environment is considered to be probably carcinogenic for humans (IARC 2010). Although numerous pollutants including carbon monoxide, polycyclic aromatic hydrocarbons benzo[a]pyrene, formaldehyde and benzene are produced from biomass combustion, particulate matter is considered the best indicator of exposure (Naeher et al. 2007; Perez-Padilla et al. 2010).

Mounting evidence points to the mutagenic, genotoxic and cytotoxic properties of biomass smoke, particularly for woodsmoke particulate matter (Danielsen et al. 2009) and this is similar to PM from urban sources (Kocbach, Namork, and Schwarze 2008b). One toxicological mechanism by which PM has been shown to induce health effects in in-vitro studies is through the pathway of oxidative stress (Danielsen et al. 2009). Oxidative stress can cause damage to deoxyribonucleic acid (DNA) including 8-hydroxylated guanine species such as 8-hydroxy-2'-deoxyguanosine (8-OHdG) (Arnett et al. 2005). Another toxicological effect of PM observed in in-vitro studies, specifically of woodsmoke PM, involves the generation of reactive oxygen species (ROS) and lipid peroxidation products such as 8-isoprostaglandin $F_{2\alpha}$ (8-isoprostane) (Danielsen et al. 2009). Both 8-OHdG and 8-isoprostane are known markers of oxidative stress (Barregard et al. 2006; Loft et al. 1992).

8-Isoprostane is a prostaglandin (PG)-F2-like compound belonging to the F2 isoprostane class that is produced in vivo by the free radical-catalyzed peroxidation of arachidonic acid (Montuschi et al. 1999). On the other hand 8-OHdG is an oxidized nucleoside of DNA and it is considered as the most frequently detected and studied DNA lesion (Wu et al. 2004). 8-OHdG is excreted during the repair of DNA damage (Wu et al. 2004). Measurements of these compounds have been performed in biologic fluid, particularly in urine and are capable of providing quantitative indices of oxidative stress (Montuschi et al. 1999). Aside experimental studies where the effect of woodsmoke on oxidative stress has been studied (Barregard et al. 2006); the literature is scant on studies on biomass smoke exposure and resulting health effects, particularly regarding women in the developing world who cook with biomass fuels. Given the continuous exposure experienced by these women over a lifetime, there is the need to better understand the effect of woodsmoke exposure on oxidative stress in this vulnerable population.

We investigated the changes in the concentrations of oxidative stress biomarkers that are related to the mechanism by which woodsmoke induces oxidative stress. Study subjects from San Marcos used wood as fuel for cooking and high PM levels have been measured among this population (Hartinger et al in review). Therefore this study examined the use of biomarkers in investigating a health end-point, namely oxidative stress, which is an indicator of possible chronic effects. Additionally, it allowed for the investigation of this health end-point in the natural environment, without experimentally introducing the exposure.

The aim of this study was twofold (1) to determine whether cookstove related biomass smoke exposure was positively associated increased urinary levels of 8-OHdG and 8-isoprostane among control and intervention subjects (2) investigate the factors that are associated with urinary 8-OHdG and 8-isoprostane concentrations among study subjects. We assessed woodsmoke exposure with personal and kitchen measurements of particulate matter and carbon monoxide, as well as urinary hydroxylated polycyclic aromatic hydrocarbons (hydroxy-PAH) metabolites.

METHODS

Study Design and Study Homes

From June to August 2009, a cross sectional study was conducted within the framework of a community based randomized control trial (c-RCT) by the Instituto de Investigación Nutricional (IIN) and the Swiss Tropical and Public Health Institute (Hartinger et al. in review; Commodore et al. in review). The May–August period in the study region is characterized by dry conditions and cold nights. HAP exposure assessment occurred during this season, no follow up during the rainy season. The altitude in the region ranges between 2200 and 3900 meters above sea level. Mean altitudes ± SD for intervention and control households are 2684 ± 284 and 2727 ± 438 meters above sea level respectively.

For this cross sectional study, control and intervention households were from participating households in the parent c-RCT (n=250 and 253 for intervention and control homes respectively). The c-RCT involved 51 community clusters who used solid fuels in the Province of San Marcos, Cajamarca region, Peru (Hartinger et al. 2011, Hartinger et al. 2012). The intervention was randomized at the community level, with the 51 community clusters allocated into the intervention arms by using covariate-based constrained randomization (Hartinger et al.

in review). Field workers for the c-RCT visited all study homes during this 3 month period; however subjects' availability, willingness to participate, as well as time and budget constraints limited the total sample size of the present study.

The aim of the parent study was to evaluate an integrated home-based environmental intervention package (IHIP) against childhood diarrhoea and respiratory infections. A pilot study was conducted in seven communities outside the study area, where several potential stove designs were tested, and subjects were consulted on cooking habits and preferences to provide a user-friendly stove design which met their household and cooking needs (Hartinger et al. 2012). The final stove model, for the c-RCT was called the OPTIMA-improved stove (hereafter OPTIMA stove). Kitchen performance tests of the OPTIMA stoves revealed a 15% reduction in daily fuel and energy use and a 16% reduction in fuel and energy use per capita compared with the traditional open fire stoves, although there was wide variability (Hartinger et al. 2011, Hartinger et al. 2012). The OPTIMA stove was built with red burnt bricks plastered with a mixture of mud, straw and donkey manure (Hartinger et al. 2012). It has three pot holes for cooking, a closed combustion chamber, metal chimney with a regulatory valve, a hood, and metal rods for support.

OPTIMA stoves were installed between October 2008 and January 2009 in 250 households (hereafter intervention households). There were no emissions tests or HAP exposure assessment prior to installation of the intervention stoves. The current study reports the only exposure assessment conducted for these stoves 6 to 8 months after installation (median 7.4 IQR = 6.6-8.1 month) (Hartinger et al. in review). OPTIMA stoves were later stratified (after exposure assessment had occurred) into two categories based on their levels of functionality

(FL). FL-I stoves were in good running conditions at the time of the assessment (plastered stove and no visible leaks when in use) and FL-II stoves were in need of repairs (re-plastering, filling small cracks, cleaning the chimney, chimney valve replacement). Field workers, during monthly visits, instructed OPTIMA stove users in the correct use of the stoves including cleaning and removing ashes and wood residues. Although surveillance occurred in all study homes, stove repair and maintenance were not addressed during home visits until after air quality monitoring had occurred. Households with OPTIMA-improved stoves were re-visited 9 months (median 9.3 IQR= 9.0-9.7 month) after installation and repaired as needed by the original stove builders (Hartinger et al. in review).

The control arm of the c-RCT included households with a diversity of stove types (Hartinger et al 2011). As such control households in this study had a wide range of stove types including (1) chimney stoves whose raw materials were provided by nongovernmental organizations (hereafter referred to as NGO), (2) chimney stoves built by the households themselves (hereafter referred to as self-improved by household), (3), and (4) non-vented stoves with pot holes for cooking including the common three stone open fire stove (hereafter referred to as traditional). At the time of sampling, control households had stoves which had been in use between 4 months to over 10 years. Lastly, households in each arm of the intervention were classified according to the primary stove in use and it is possible that some chimney stoves were used together with traditional stoves in some households, particularly for cooking animal feed or other meals which required substantial cooking times.

Urine Sampling

First morning urine voids were provided by the subjects between 5 and 7 o'clock in the morning. Samples were collected in 100ml polypropylene containers by study subjects and

stored in an insulated lunch bag with ice packs. Containers were then transported on ice to the study base and transferred into 50 ml polypropylene centrifuge tubes and frozen at -20^{o}C until the end of the study. Samples were subsequently placed on dry ice and shipped to the United States where they were stored at -80^{o}C. The samples were later aliquoted at the National Center for Environmental Health (NCEH) laboratory at the Centers for Disease Control and Prevention (CDC) in Atlanta, GA and shipped to the Keck School of Medicine, University of Southern California, CA for 8-OHdG and 8-isoprostane analyses.

In this cross sectional study, 50 control and 43 intervention households were selected from participating households in the c-RCT (Hartinger et al. in review). A total of 85 subjects provided urine samples (n=45 and 40 for control and intervention households respectively). Eight subjects did not provide urine samples (n=5 and 3 for control and intervention homes respectively). Of the 85 urine samples provided, only 84 had sufficient urine volume for analysis.

Exposure Monitoring

Forty eight-hr time integrated $PM_{2.5}$ and real time CO measurements were collected to assess the personal exposures of the household cook (usually the mother). Study subjects wore exposure monitoring equipment placed in vests which were worn in their breathing zones. Area 48-hr integrated $PM_{2.5}$ and real time CO measurements were also taken in the kitchen (Hartinger et al. in review). Additionally, passive carbon monoxide (CO) diffusion tubes were used to obtain 48-hour time integrated CO measurements in study kitchens and personal CO exposures of the mother and a child under the age of five (Commodore et al. in review). Questionnaires were administered on the second day of air sampling to obtain data on household air pollution, respiratory health-related symptoms, demographics, daily activities and commuting habits

Analysis of 8-OHdG

Urinary 8-OHdG concentrations were determined using the HPLC-ECD method. In brief, a solution of 2 ml aliquot of the urine sample and 2 ml potassium dihydrogen phosphate buffer (KH_2PO_4) (0.1 M, pH 6) was applied to a solid phase extraction cartridge (Bond Elut-Certify, Varian) already conditioned with methanol, deionized (DI) water and KH_2PO_4 (0.1 M, pH 6). The cartridge was then washed with DI water and KH_2PO_4 (0.1 M, pH 6) and vacuum dried for 10 minutes. 8-OHdG was eluted by 2 ml solution of 30% methanol in DI water and 20 µl of eluted solution was injected into the HPLC (Alliance Waters 2695 with 2465 Electron-Chemical Detector). 8-OHdG was detected at a potential of +0.6 V at a range of 50 nA and a time constant of 1.0 sec. A linear calibration curve was obtained using aqueous solutions of 8-OHdG standard. The recovery rate of the analytical procedure was 99.6% with an analytic precision of 4.41% and a detection limit of 0.46 ng/ml.

Analysis of 8-isoprostane

Urinary 8-iso-prostaglandin-$F_{2\alpha}$ (8-iso-$PGF_{2\alpha}$) was analyzed using a modified LC-MS/MS method (Liang et al, 2003). One milliliter of urine sample was spiked with 5 ng of 8-iso-$PGF_{2\alpha}$-D4, and diluted with 1 mL of water. After vortexing and centrifugation, the sample was purified using solid phase extraction. A Bond Elut C18 cartridge was prewashed with 5 mL of methanol and 5 mL of water; then the sample was loaded and washed with 5 mL of water, 5 mL of methanol:water (5:95), and 1 mL of hexane, and then eluted with 2 mL of ethyl acetate. Following solvent evaporation, the sample extract was reconstituted with 100 µL of acetonitrile: water (15:85) and 20 µL was injected to LC-MS/MS for analysis. The LC-MS/MS was performed on TSQ Quantum Access MAX triple stage quadrupole mass specitrometer, coupled with Accela 1250 pump and Accela Open Autosampler.

Chromatographic separation was achieved on a Phenomenex Luna 3µ C18 (50 x 2 mm) column, with 0.1% formic acid in water (solvent A) and acetonitrile (solvent B) as mobile phase. The flow rate was 100 µL/min, and the gradient elution was programmed as follows: hold at 85% A for 1 min, decreasing to 20% A over 10 mins, hold for 3 mins then increasing to 85% at 13.5 mins. The mass spectrometer was operated in the negative ESI mode. The capillary temperature and vaporizer temperature were at 270˚C and 320˚C, respectively. The ion spray voltage was set to 3000. Nitrogen sheath and auxiliary gases were set to 40 and 20 arbitrary units, respectively. The ion pairs of m/z 353/193, m/z357/197 were used to monitor 8-iso-$PGF_{2\alpha}$ and 8-iso- $PGF_{2\alpha}$-D4, respectively. The calibration curve was constructed over a range of 50 pg/mL-5000 pg/mL. The recovery was 98.4% with analytic precision (%CV) of 3.27% and a detection limit of 50 pg/mL.

Creatinine and hydroxy-PAH analysis

Urinary creatinine was measured on a Roche Hitachi 912 Chemistry Analyzer (Hitachi Inc., Pleasanton, CA) using the Creatinine Plus Assay, as described in Roche's Creatinine Plus Product Application # 03631761003. Additionally, 2mL urine aliquots were analyzed by the NCEH laboratory for polycyclic aromatic hydrocarbon metabolites, hydroxyl-substituted naphthalene, fluorene, phenanthrene, and pyrene from urine samples of 365 subjects participating in the c-RCT (Li et al. 2012b). A semi-automated liquid–liquid extraction and isotope dilution gas chromatography/high-resolution mass spectrometry (GC/HRMS) method were used (Li et al. 2006). We present hydroxylated polycyclic aromatic hydrocarbons (hydroxy-PAH) metabolite data for the 39 and 45 intervention and control subjects in the current oxidative stress study. The specific hydroxy-PAH metabolites include 1-naphthol (1-NAP), 2-naphthol (2-NAP), 2-hydroxyfluorene (2-FLU), 3-hydroxyfluorene (3-FLU), 9-hydroxyfluorene (9-FLU), 1-

hydroxyphenanthrene (1-PHE), 2-hydroxyphenanthrene (2-PHE), and 1-hydroxypyrene (1-PYR).

Statistical Analysis

Data were analyzed using SAS 9.2 (Cary, NC). All two-sided probability values less than 0.05 were considered statistically significant. Oxidative stress markers, woodsmoke exposure measurements (CO and $PM_{2.5}$) and hydroxy-PAHs were then log transformed for all analysis. The relationship log transformed creatinine corrected urinary oxidative stress biomarker concentrations and woodsmoke exposure measurements were first determined using Pearson correlation coefficients.

The following exposure measurements were used in the correlation analysis: 48hr time integrated personal $PM_{2.5}$, 48hr time integrated personal real time CO, highest 30 second CO measurement obtained during sampling, highest 15 minute average CO and highest 90 minute average CO. Subject's personal 48hr integrated personal CO data from passive diffusion tubes were available for 81/84 of study subjects, as such these CO measurements were also tested in the analysis.

Data were then analyzed with linear models (PROC GLM) to (1) test for a dose-response relationship between the oxidative stress biomarkers and woodsmoke exposure and (2) investigate factors that are associated with creatinine corrected urinary 8-OHdG and 8-isoprostane concentrations among study subjects. Predictor variables include the weight and age of study subjects, cooking time, amount of time mother's spent playing with children, volume of kitchen, stove type and wood type. The time mothers spent in playing with their children during the day was also assessed to determine whether this affected their respective exposures. This variable was chosen as a proxy for how often the mother and child are together on any given day,

which was thought to be important. This latter variable was considered to be potentially important because if playing time did not overlap with cooking time, it could impact personal exposures. Other variables included the distance of the subject's homes to the road, the frequency of traffic in subject's neighborhood and whether subject had eaten food exposed to fire. Mother's weight, age, cooking time and the amount of time spent playing with children were centered on their respective means before inclusion in the models.

Predictors were included in the model individually to test for significant associations with each oxidative stress biomarker. Stepwise regression (PROC GLMSELECT) was then used for multiple effect selection with the significance levels of the F statistic for entering or removing effects set at 0.2 and 0.1 respectively. Second order interaction terms were also investigated but none were significant. Finally simple logistic regression models were fit to test whether each of self-reported respiratory symptoms were associated with urinary oxidative stress markers. These respiratory systems include chest congestion, chest pains, chest pressure, phlegm coughing, lack of air runny nose and wheezing.

RESULTS

Household characteristics

Household specific information is presented according to stove type (Table 6.1). Intervention households used OPTIMA FL-I (n=22) or OPTIMA FL-II (n=17) stoves and control households used NGO stoves (n=7), traditional stoves (n=29) or stoves that were self-improved by the household (n=9). Subjects' mean ages were similar among all study households (Table 6.1). Mean cooking time for study subjects was above 3 hrs (Table 6.1). *Eucalipto* (eucalyptus sp) was the most popular wood types used as cooking fuel by study subjects during the sampling period (Table 6.1).

Creatinine corrected urinary 8-OHdG and 8-isoprostane measurements for each study subject together with personal and kitchen $PM_{2.5}$ and CO measurements are provided (Table 6.2). Urinary 8-OHdG levels ranged from 100.4 ug/g creatinine (44.3, 227.8) ug/g creatinine (mean with 95% CI) to 157.0 (60.1, 410.0) ug/g creatinine (Table 6.2). Urinary 8-isoprostane levels ranged from 0.7 (0.5, 0.9) ug/g creatinine to 1.1 (0.7, 1.6) ug/g creatinine (Table 6.2). There were no statistically significant differences in urinary oxidative stress biomarkers between the two arms of the intervention. Mean (95% CI) urinary 8-OHdG and 8-isoprostane levels among intervention stove users were 132.9 (97.7, 180.8) ug/g creatinine and 0.8 (0.6, 1.1) ug/g creatinine respectively. Likewise, among control stove users, these levels were 139.3 (108.7, 178.4) ug/g creatinine and 0.7 (0.6, 0.9) ug/g creatinine respectively.

Association between hydroxy-PAH metabolites and oxidative stress biomarkers

Correlation analysis involving creatinine corrected hydroxy-PAH metabolites (Li et al. 2012b), oxidative stress biomarkers as well as CO and $PM_{2.5}$ are presented (Table 6.3). When correlation analysis was performed between each specific hydroxy-PAH and the oxidative stress markers, 1-hyroxyphenanthrene (1-PHE) and 2-hyroxyphenanthrene (2-PHE) were marginally correlated with creatinine corrected 8-isoprostane levels, p=0.099 and 0.06 respectively (Table 6.3). 8-isoprostane was positively associated with the sum of the hydroxy-PAH metabolites although this association was not statistically significant (Figure 6.2).

Association between CO and $PM_{2.5}$, and oxidative stress biomarkers

Forty eight-hr time integrated personal $PM_{2.5}$ was negatively associated with 8-isoprostane (p=0.04) and 48hr personal CO was negatively associated with 8-OHdG (p=0.01) (Table 6.3 and Table 6.4). Kitchen levels of $PM_{2.5}$, CO and 48hr time integrated personal CO measurements using passive diffusion tubes were not correlated with either oxidative stress biomarker (Table 6.3).

As seen in Table 6.4, the highest 30 second personal CO measured during the sampling period for each subject was significantly but negatively associated with both 8-OHdG (p=0.001) and 8-isoprostane (p=0.04) while 15 minute and 90 minute averages of real time CO were negatively associated with 8-isoprostane only (p=0.005 and 0.01). Results indicate a pattern of decreasing urinary oxidative stress biomarker levels with increasing woodsmoke exposure measurements (Figure 6.1). Scatter plots of $PM_{2.5}$ exposures against oxidative stress biomarkers allude to this and the trend suggest decreasing urinary oxidative stress biomarker levels with increasing $PM_{2.5}$ exposures (Figure 6.1).

Factors associated with oxidative stress biomarkers

Individual variables included in statistical models are presented in Table 6.4. All variables found to be associated with the oxidative stress markers were entered for the final model selection process using stepwise regression. The test statistics of the modeled effects of individual variables are listed (Table 6.4). Table 6.5 presents the final models for both oxidative stress markers. Subjects who resided in communities with frequent traffic (a car every few minutes) had mean (95% CI) urinary 8-OHdG concentrations of 333.5 (199.0, 559.0) ug/g creatinine (n=12) compared to those who lived where cars passed by every hour: 148.3 (102.8, 214.1) ug/g creatinine (n=27) or seldomly (every few days): 133.6 (93.7, 190.5) ug/g creatinine (n=23) (Table 5). Also for 8-OHdG, subjects who reported consuming food exposed to fire had higher urinary levels of 250.4 (151.0, 415.2) ug/g creatinine (n=11) compared to those who had not consumed food exposed to fire: 140.7 (111.7, 177.0) ug/g creatinine (n=51).

After controlling for the effects of traffic in the community and eating food exposed to fire, cooking time was positively correlated with urinary 8-OHdG (correlation coefficient (r) = 0.29, p= 0.01, n=80) (Table 6.5). On the other hand, subjects' real time personal CO exposures were negatively correlated with 8-OHdG (Table 6.5). The highest 30 second personal CO measured in real time during the sampling period for each subject was significantly but negatively correlated with 8-OHdG (r=-0.32, p=0.001, n=73) while 15 minute and the mean CO exposure over the 48hr period were also significantly correlated with 8-OHdG (r=-0.25, p=0.04 and r=-0.26, p=0.03, n=73) (Table 6.5). Ninety minute averages of real time CO were also negatively, but marginally correlated with 8-isoprostane only (r=-0.23, p=0.09, n=73) (Table 6.5).

For urinary 8-isoprostane, subjects who lived ≤ 20 meters to the road had levels of 0.8 (0.6, 1.1) ug/g creatinine (n=25) compared to those who lived > 20 meters away with levels of

0.6 (0.5, 0.7) ug/g creatinine (n=38) (Table 6.5). After controlling for the effect of distance of road to subjects' homes, 1-hyroxyphenanthrene and 2-hyroxyphenanthrene were both positively correlated with 8-isoprostane (correlation coefficients for both hydroxy-PAH biomarkers=0.21, p values=0.05 and 0.06 respectively, n=84) (Table 6.5). 48hr time integrated personal $PM_{2.5}$ was also marginally and negatively correlated with urinary 8-isoprostane after controlling for the effect of distance of homes to the road (r=-0.21, p=0.09, n=69)

Simple logistic regression models used to investigate whether self-reported respiratory symptoms (for example wheezing, coughing, etc) were associated with urinary oxidative stress markers revealed an association between the reporting of phlegm (p=0.043) and wheezing (p=0.047) with increasing urinary 8-isoprostane levels. Subjects experiencing phlegm (n=11/78) and wheezing (n=8/78) were 2.90 (1.04, 8.14) and 3.28 (1.02, 10.58) times as likely to have higher urinary 8-isoprostane. All other self-reported respiratory symptoms: chest congestion, chest pains, chest pressure, coughing, lack of air and runny nose were not associated with either 8-OHdG or 8-isoprostane concentrations (all p>0.05).

DISCUSSION

It has been observed in in-vivo and human experiment studies that oxidative stress, in response to wood smoke, may play an important role in airway and alveolar epithelium injury (Barregard et al. 2008; Barregard et al. 2006; Danielsen et al. 2009; Danielsen et al. 2011). 8-Isoprostane is produced in vivo by the free radical-catalyzed peroxidation of arachidonic acid (Montuschi et al. 1999) while 8-OHdG is an oxidized nucleoside of DNA and is the most frequently detected and studied DNA lesion (Wu et al. 2004). Measurements of these compounds have been performed in biologic fluid, particularly in urine and are capable of providing quantitative indices of oxidative stress (Montuschi et al. 1999). In the present study, we

investigated the effects of cookstove related biomass smoke exposure, subject characteristics and other factors on urinary concentrations of 8-OHdG and 8-isoprostane among women in rural Peru.

Urinary 8-isoprostane levels reported in the literature are comparable to results reported in the current study. Levels of this metabolite in normal individuals have been measured at 0.39 ± 0.18 ug/g creatinine (mean ± 2 SD) (Roberts II and Morrow 2000). This biomarker has been shown to increase with increasing exposure. Lai et al (2012) found median concentrations (interquartile range) of urinary 8-isoprostane among 47 Taiwanese female highway toll station workers exposed to traffic exhausts to be 3.69 (3.26 – 4.39) ug/g creatinine among exposed smokers (n=5) and 3.00 (2.63 – 4.17) ug/g creatinine among exposed nonsmokers (n=42).

The elimination half-life of isoprostanes in the circulation has been noted to be relatively short, less than 20 min (Gniwotta et al. 1997). Therefore once the exposure is no longer available, it is expected that levels of the biomarker will fall, particularly from nighttime until the following morning (Nuernberg et al. 2008). Also, 24-hr urine samples may have been more informative since a single sample will provide only an index of isoprostane formation and only in chronic disease states is there expected to be a relatively steady rate of formation (Gniwotta et al. 1997). However, this is not the case among our study population as these women are mainly young women of child bearing age with no reported chronic diseases.

Urinary 8-OHdG levels reported in the literature are lower compared to results reported in the current study. Among 344 nonsmoking office women in offices in Taiwan where CO_2 levels were in the range of 467–2810 ppm, mean measurements of urinary 8-OHdG ranged from 3.10 to 6.27 μg/g creatinine (Lu et al. 2007). Among a healthy Japanese population, women had mean (± SD) urinary 8-OHdG levels of 15.58 ± 5.49 μg/g creatinine (Kimura et al. 2006). Mean

concentrations of urinary 8-OHdG were substantially higher among the 47 female highway toll station workers in Taiwan in the study by Lai et al (2005). Exposed non-smokers had mean urinary 8-OHdG of 13.6 μg/g creatinine while exposed smokers had 10.2 μg/g creatinine (Lai et al. 2005).

Elevated 8-OHdG levels have also been detected in the urine samples of smokers and occupational workers (Chuang et al. 2003; Kim et al. 2004; Lodovici et al. 2000). It must be noted though that the mean urinary 8-OHdG concentration recorded in the literature have been between 3 - 20 ug/g creatinine (Chuang et al. 2003; Kim et al. 2004; Lai et al. 2005; Lee et al. 2010; Tamura et al. 2006). Given the continuous exposures experienced by subjects in the current study over the course of their lifetime, this cross sectional study shows high levels of urinary 8-OHdG and indicates that there is a sustained systemic oxidative stress status among these women.

Our results showed that study subjects are exposed to high levels of $PM_{2.5}$ and CO from cookstove related woodsmoke. We postulate that presence of highly reactive electrophilic compounds in biomass smoke (Lewtas 2007) and or the continual induction of intracellular ROS as a result of inhaling such exposures (Avakian et al. 2002) leads to high urinary oxidative stress in our study population. Results also show that increasing biomass smoke exposure was not positively correlated with urinary levels of 8-OHdG and 8-isoprostane among study subjects. This was unexpected. However, but it must be noted that the exposures experienced by the women are high compared to EPA's air quality standard of 35 ug/m^3 and WHO recommended air quality guideline of 25 ug/m^3 for a 24hr period. Additionally the exposures are also continuous; and this allows little recovery time from one exposure period to the next.

In our study there were no statistically significant differences in urinary 8-OHdG and 8-isoprostane among control and intervention stoves users (see stove effect in Table 6.4). This finding is supported by earlier studies showing that exposure did not differ significantly among intervention and control stove users (Hartinger et al. review). Possible reasons for high exposure measurements and chronically high urinary oxidative stress biomarkers among chimney stove users in our study include lack of maintenance over time, inadequate stove design, improper and nonexclusive stove use (Commodore et al. in review).

In our study, 48hr mean CO was weakly but negatively associated with 8-OHdG and 48hr mean $PM_{2.5}$ was weakly but negatively associated with 8-isoprostane. These findings are surprising. Possible reasons may be as a result of decreased DNA damage or decreased DNA repair activity (Nuernberg et al. 2008). As has been hypothesized by others in the literature concerning workers exposed to metal fumes (Palmer et al. 2006; Wang et al. 2005) this finding may be due to a 'muted response' to inhaled exposure, in this case, HAP constituents. Future studies need to be designed to determine whether this response truly occurs, the mechanism by which it occurs, and most importantly, whether this response is damaging or protective (Nuernberg et al. 2008).

In our study, metabolites of hyroxyphenanthrene, 1-PHE and 2-PHE, were correlated with 8-isoprostane. Urinary 8-OHdG levels were not correlated with individual urinary hydroxy-PAHs or the summed total of the PAH metabolites. A study among children exposed to heavy traffic in Guangzhou, China did not also observe any strong correlations between hydroxy-PAHs and 8-OHdG (Fan et al. 2011). Our results indicated no correlation between urinary 8-OHdG and 8-isoprostane. As suggested by others in the literature (England et al. 2000; Sakano et al. 2009),

there appears to be no correlation between oxidative DNA damage products and F_2-isoprostanes in healthy individuals.

Cooking time was weakly, but positively correlated with urinary 8-OHdG concentrations. This finding is expected since longer cooking times implies longer exposures to HAP. Increasing cooking time increases the potential for oxidative DNA adduct formation due to continuous exposure to biomass smoke combustion products. Likewise eating food exposed to fire was also positively and significantly correlated with urinary 8-OHdG. Eating food exposed to fires also leads to the generation of reactive oxygen and nitrogen species which are precursors of oxidative stress (Tuohy et al. 2006).

In this study, subjects who self-reported wheezing and the presence of phlegm had higher urinary 8-isoprostane levels compared to those who did not report these symptoms. Given this preliminary study, self-reported symptoms may be a useful indicator of health. Other studies have also observed adverse symptoms from subjects exposed to high levels of HAP (Clark et al. 2009; Diaz et al. 2008).

In the questionnaire given to study subjects, none reported a chronic respiratory illness. There is an implication that subjective expressions of health can be sensitive indicators of health status (Clark et al. 2009), and there is the need for future studies to explore this tool.

Results from our study indicate that proximity of subjects' homes to the road side and the frequency of vehicular traffic lead to increased urinary oxidative stress in urine samples. Subjects who lived less than 20 meters to the road side and those whose communities had frequent traffic had higher urinary 8-isoprostane and 8-OHdG levels respectively. Elevated levels of both markers have been recorded in the literature among subjects exposed to diesel and petroleum by-products (Lai et al. 2005; Lee et al. 2010). In the cross-sectional study of 47 Taiwanese female highway toll station workers exposed to traffic exhaust, there was a 9.32 mg/g creatinine increase of urinary 8-OHdG per 1000 cars/hour increase in average traffic density (Lai et al. 2005). The corresponding estimate for trucks and buses was of greater magnitude; 16.76 mg/g creatinine (Lai et al. 2005). This finding may help explain the elevated 8-OHdG levels in the current population. It implies that aside biomass smoke combustion products from the indoor environment, subjects in this population are also exposed to traffic air pollution which represents an additional physiologic burden.

As the Global Alliance for Clean Cookstoves (GACC) aims to reduce HAP and the adverse health effects associated with it, these results add to the limited body of literature on the health effects experienced by populations exposed to HAP. The GACC, led by the United Nations Foundation, has the goal of 100 million households adopting clean and efficient cookstoves by the year 2020 (GACC 2011). Certain subjects must be addressed before these clean and efficient stoves are adopted globally. It is essential to determine the amount of HAP reduction necessary to improve health (Pope III et al. 2011; Smith et al. 2011; Smith and Peel

2010), as well as develop new and rigorous means to evaluate the health benefits of worldwide stove implementation programs (Martin et al. 2011). In the advent of national cookstove programs in Peru and other countries, evaluation of new stove models is also an important step to understanding the impact of cookstove related woodsmoke exposure (Fitzgerald et al. 2012b).

Strengths and Limitations

To our best knowledge, this is the first study looking at understanding systemic oxidative stress in urine samples among women exposed to cookstove related biomass combustion in Peru. The use of two measures of systemic oxidative stress represents a significant strength of this study. Other strengths of our study include measurement of biomarkers in urine rather than blood, which avoids potential artifactual oxidation (Patel et al. 2007). Additionally, study subjects were healthy, although chronically exposed to woodsmoke, and this may have minimized the effects of preclinical or overt disease regarding the concentrations of oxidative stress biomarkers.

The use of spot urine samples rather than 24hr urine samples did not allow us to examine the intra-individual variability in the biomarkers (Pilger et al. 2001; Pilger et al. 2002). Future studies can consider taking repeated simultaneous exposure (Dionisio et al. 2011; Smith et al. 2010) and urine samples (Gniwotta et al. 1997; Roberts II and Morrow 2000) over extended periods of time to better characterize the exposure-response relationship.

In our study we did not have information on alcohol drinking habits and antioxidant capacity, factors that potential confounders in lipid peroxidation (Lai et al. 2012). Future studies may need this information for further exposure characterization.

Households in our study were also not sampled prior to and immediately after improved stove installation and this prevented evaluation of the effectiveness of the OPTIMA stoves at improving health soon after installation. Even more challenging, although the current measurements inform, the results suggest that the OPTIMA stove may not be effective over the long term at reducing HAP exposures and health effects.

CONCLUSION

We characterized urinary 8-OHdG and 8-isoprostane among intervention and control households in San Marcos Peru. Although both oxidative stress biomarkers did not differ significantly between the two intervention arms, 8-OHdG levels were higher among subjects when compared to values of urinary 8-OHdG reported in the scientific literature.

This study is the first to investigate the effect of woodsmoke exposure on systemic oxidative stress among women exposed to high levels of cookstove related woodsmoke in rural Peru. Given the continuous exposures experienced by subjects in the current study over the course of their lifetime, this cross sectional study shows high levels of urinary 8-OHdG and indicates that there is a sustained systemic oxidative stress status among these women. Further investigation on the cumulative effects of constant exposure to HAP and other activities that increase systemic oxidative stress among such populations is needed.

REFERENCES

Arnett, S. D., D. M. Osbourn, K. D. Moore, S. S. Vandaveer, and C. E. Lunte. 2005. "Determination of 8-oxoguanine and 8-hydroxy-2′-deoxyguanosine in the rat cerebral cortex using microdialysis sampling and capillary electrophoresis with electrochemical detection." *Journal of Chromatography B* 827(1): 16-25.

Avakian, M. D., B. Dellinger, H. Fiedler, B. Gullet, C. Koshland, S. Marklund, G. Oberdörster, S. Safe, A. Sarofim, and K. R. Smith. 2002. "The origin, fate, and health effects of combustion by-products: a research framework." *Environmental health perspectives* 110(11): 1155.

Barregard, L., G. Sällsten, L. Andersson, A. C. Almstrand, P. Gustafson, M. Andersson, and A. C. Olin. 2008. "Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress." *Occupational and Environmental Medicine* 65(5): 319.

Barregard, L., G. Sällsten, P. Gustafson, L. Andersson, L. Johansson, S. Basu, and L. Stigendal. 2006. "Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation." *Inhalation Toxicology* 18(11): 845-53.

Chuang, C. Y., C. C. Lee, Y. K. Chang, and F. C. Sung. 2003. "Oxidative DNA damage estimated by urinary 8-hydroxydeoxyguanosine: influence of taxi driving, smoking and areca chewing." *Chemosphere* 52(7): 1163-71.

Clark, M. L., J. L. Peel, J. B. Burch, T. L. Nelson, M. M. Robinson, S. Conway, A. M. Bachand, and S. J. Reynolds. 2009. "Impact of improved cookstoves on indoor air pollution and adverse health effects among Honduran women." *International Journal of Environmental Health Research* 19(5): 357-68.

Danielsen, P. H., S. Loft, A. Kocbach, P. E. Schwarze, and P. Møller. 2009. "Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines." *Mutation Research/Genetic Toxicology and Environmental Mutagenesis* 674(1-2): 116-22.

Danielsen, P. H., P. Møller, K. A. Jensen, A. K. Sharma, H. Wallin, R. Bossi, H. Autrup, L. Mølhave, J. L. Ravanat, and J. J. Briedé. 2011. "Oxidative stress, DNA damage, and inflammation induced by ambient air and wood smoke particulate matter in human A549 and THP-1 cell lines." *Chemical Research in Toxicology*.

Diaz, E., N. Bruce, D. Pope, A. Diaz, K. Smith, and T. Smith-Sivertsen. 2008. "Self-rated health among Mayan women participating in a randomised intervention trial reducing indoor air pollution in Guatemala." *BMC International Health and Human Rights* 8(1): 7.

Dionisio, K. L., S. R. C. Howie, F. Dominici, K. M. Fornace, J. D. Spengler, S. Donkor, O. Chimah, C. Oluwalana, R. C. Ideh, and B. Ebruke. 2011. "The exposure of infants and children

to carbon monoxide from biomass fuels in The Gambia: a measurement and modeling study." *Journal of Exposure Science and Environmental Epidemiology*.

England, T., E. Beatty, A. Rehman, J. Nourooz-Zadeh, P. Pereira, J. O'Reilly, H. Wiseman, C. Geissler, and B. Halliwell. 2000. "The steady-state levels of oxidative DNA damage and of lipid peroxidation (F2-isoprostanes) are not correlated in healthy human subjects." *Free Radical Research* 32(4): 355-62.

Fan, R., D. Wang, C. Mao, S. Ou, Z. Lian, S. Huang, Q. Lin, R. Ding, and J. She. 2011. "Preliminary study of children's exposure to PAHs and its association with 8-hydroxy-2'-deoxyguanosine in Guangzhou, China." *Environment international*.

Fitzgerald, C., M. Aguilar-Villalobos, A. R. Eppler, S. C. Dorner, S. L. Rathbun, and L. P. Naeher. 2012. "Testing the effectiveness of two improved cookstove interventions in the Santiago de Chuco Province of Peru." *Science of the Total Environment*.
GACC. 2011. "The Global Alliance for Clean Cookstoves: Overview [Document on the Internet] [updated 2011; cited 2011 Oct 23]. Available from http://cleancookstoves.org/."

Gniwotta, C., J. D. Morrow, L. J. Roberts II, and H. Kühn. 1997. "Prostaglandin F2-like compounds, F2-isoprostanes, are present in increased amounts in human atherosclerotic lesions." *Arteriosclerosis, thrombosis, and vascular biology* 17(11): 3236-41.

IARC. 2010. "Household use of solid fuels and high-temperature frying." *IARC Monogr Eval Carcinog Risks Hum.* 95: 1-430.

Kim, J. Y., S. Mukherjee, L. C. Ngo, and D. C. Christiani. 2004. "Urinary 8-hydroxy-2'-deoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to fine particulates." *Environmental health perspectives* 112(6): 666.

Kimura, S., H. Yamauchi, Y. Hibino, M. Iwamoto, K. Sera, and K. Ogino. 2006. "Evaluation of urinary 8-hydroxydeoxyguanine in healthy Japanese people." *Basic & Clinical Pharmacology & Toxicology* 98(5): 496-502.

Kocbach, A., E. Namork, and P. E. Schwarze. 2008. "Pro-inflammatory potential of wood smoke and traffic-derived particles in a monocytic cell line." *Toxicology* 247(2-3): 123-32.

Lai, C., S. Liou, H. Lin, T. Shih, P. Tsai, J. Chen, T. Yang, J. Jaakkola, and P. Strickland. 2005. "Exposure to traffic exhausts and oxidative DNA damage." *Occupational and Environmental Medicine* 62(4): 216-22.
Lai, C. H., S. H. Liou, J. J. K. Jaakkola, H. B. Huang, T. Y. Su, and P. T. Strickland. 2012. "Exposure to Polycyclic Aromatic Hydrocarbon Associated with Traffic Exhaust Increases Lipid Peroxidation and Reduces Antioxidant Capacity."

Lee, M.-W., M.-L. Chen, S.-C. C. Lung, C.-J. Tsai, X.-J. Yin, and I. F. Mao. 2010. "Exposure assessment of PM2.5 and urinary 8-OHdG for diesel exhaust emission inspector." *Science of the Total Environment* 408(3): 505-10.

Lewtas, J. 2007. "Air pollution combustion emissions: characterization of causative agents and mechanisms associated with cancer, reproductive, and cardiovascular effects." *Mutation Research/Reviews in Mutation Research* 636(1-3): 95-133.

Li, Z., A. Commodore, S. Hartinger, E. Pittman, D. Trinidad, R. Ostovar, K. Hubbard, A. Sjodin, C. Lanata, A. Gil, D. Mäusezahl, and L. Naeher. 2012. "O-046: Assessment of Exposure to PAHs in Cook Stove Intervention Projects in Peru by Urinary Biomonitoring." *Epidemiology* 23(5S): 10.1097/01.ede.0000416704.62764.3d.

Li, Z., L. C. Romanoff, A. Debra, N. Hussain, R. S. Jones, E. N. Porter, D. G. Patterson Jr, and A. Sjödin. 2006. "Measurement of urinary monohydroxy polycyclic aromatic hydrocarbons using automated liquid-liquid extraction and gas chromatography/isotope dilution high-resolution mass spectrometry." *Analytical chemistry* 78(16): 5744-51.

Lodovici, M., C. Casalini, R. Cariaggi, L. Michelucci, and P. Dolara. 2000. "Levels of 8-hydroxydeoxyguanosine as a marker of DNA damage in human leukocytes." *Free Radical Biology and Medicine* 28(1): 13-17.

Loft, S., K. Vistisen, M. Ewertz, A. Tjønneland, K. Overvad, and H. E. Poulsen. 1992. "Oxidative DNA damage estimated by 8-hydroxydeoxyguanosine excretion in humans: influence of smoking, gender and body mass index." *Carcinogenesis* 13(12): 2241.

Lu, C. Y., Y. C. Ma, J. M. Lin, C. Y. Chuang, and F. C. Sung. 2007. "Oxidative DNA damage estimated by urinary 8-hydroxydeoxyguanosine and indoor air pollution among non-smoking office employees." *Environmental research* 103(3): 331-37.

Martin, W. J., R. I. Glass, J. M. Balbus, and F. S. Collins. 2011. "A Major Environmental Cause of Death." *Science* 334(6053): 180-81.

Montuschi, P., M. Corradi, G. CIABATTONI, J. Nightingale, S. A. Kharitonov, and P. J. Barnes. 1999. "Increased 8-isoprostane, a marker of oxidative stress, in exhaled condensate of asthma patients." *American journal of respiratory and critical care medicine* 160(1): 216-20.

Naeher, L. P., M. Brauer, M. Lipsett, J. T. Zelikoff, C. D. Simpson, J. Q. Koenig, and K. R. Smith. 2007. "Woodsmoke Health Effects: A Review." *Inhalation Toxicology* 19(1): 67-106.
Nuernberg, A. M., P. D. Boyce, J. M. Cavallari, S. C. Fang, E. A. Eisen, and D. C. Christiani. 2008. "Urinary 8-isoprostane and 8-OHdG concentrations in boilermakers with welding exposure." *Journal of occupational and environmental medicine* 50(2): 182.

Palmer, K., R. McNeill-Love, J. Poole, D. Coggon, A. Frew, C. Linaker, and J. Shute. 2006. "Inflammatory responses to the occupational inhalation of metal fume." *European Respiratory Journal* 27(2): 366-73.

Patel, P. R., R. J. Bevan, N. Mistry, and J. Lunec. 2007. "Evidence of oligonucleotides containing 8-hydroxy-2′-deoxyguanosine in human urine." *Free Radical Biology and Medicine* 42(4): 552-58.

Perez-Padilla, R., A. Schilmann, and H. Riojas-Rodriguez. 2010. "Respiratory health effects of indoor air pollution [Review article]." *The International Journal of Tuberculosis and Lung Disease* 14(9): 1079-86.

Pilger, A., D. Germadnik, K. Riedel, I. Meger-Kossien, G. Scherer, and H. W. Rüdiger. 2001. "Longitudinal study of urinary 8-hydroxy-2'-deoxyguanosine excretion in healthy adults." *Free Radical Research* 35(3): 273-80.

Pilger, A., S. Ivancsits, D. Germadnik, and H. Rudiger. 2002. "Urinary excretion of 8-hydroxy-2'-deoxyguanosine measured by high-performance liquid chromatography with electrochemical detection." *Journal of Chromatography B* 778(1-2): 393-401.

Pope III, C. A., R. T. Burnett, M. C. Turner, A. Cohen, D. Krewski, M. Jerrett, S. M. Gapstur, and M. J. Thun. 2011. "Lung Cancer and Cardiovascular Disease Mortality Associated with Ambient Air Pollution and Cigarette Smoke: Shape of the Exposure–Response Relationships." *Environmental health perspectives* 119(11): 1616.

Rehfuess, E., S. Mehta, and A. Prüss-Üstün. 2006. "Assessing household solid fuel use: multiple implications for the Millennium Development Goals." *Environmental health perspectives* 114(3): 373.

Roberts II, L. J. and J. D. Morrow. 2000. "Measurement of F2-isoprostanes as an index of oxidative stress in vivo." *Free Radical Biology and Medicine* 28(4): 505-13.

Sakano, N., D. H. Wang, N. Takahashi, B. Wang, R. Sauriasari, S. Kanbara, Y. Sato, T. Takigawa, J. Takaki, and K. Ogino. 2009. "Oxidative stress biomarkers and lifestyles in Japanese healthy people." *Journal of clinical biochemistry and nutrition* 44(2): 185.

Smith, K. R., J. P. McCracken, L. Thompson, R. Edwards, K. N. Shields, E. Canuz, and N. Bruce. 2010. "Personal child and mother carbon monoxide exposures and kitchen levels: Methods and results from a randomized trial of woodfired chimney cookstoves in Guatemala (RESPIRE)." *J Expos Sci Environ Epidemiol* 20(5): 406-16.

Smith, K. R., J. P. McCracken, M. W. Weber, A. Hubbard, A. Jenny, L. M. Thompson, J. Balmes, A. Diaz, B. Arana, and N. Bruce. 2011. "Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial." *The Lancet* 378(9804): 1717-26.

Smith, K. R. and S. Mehta. 2003. “The burden of disease from indoor air pollution in developing countries: comparison of estimates.” *International Journal of Hygiene and Environmental Health* 206(4-5): 279-89.

Smith, K. R. and J. L. Peel. 2010. “Mind the gap.” *Environmental health perspectives* 118(12): 1643.

Tamura, S., H. Tsukahara, M. Ueno, M. Maeda, H. Kawakami, K. Sekine, and M. Mayumi. 2006. “Evaluation of a urinary multi-parameter biomarker set for oxidative stress in children, adolescents and young adults.” *Free Radical Research* 40(11): 1198-205.

Tuohy, K. M., D. J. S. Hinton, S. J. Davies, M. J. C. Crabbe, G. R. Gibson, and J. M. Ames. 2006. “Metabolism of Maillard reaction products by the human gut microbiota–implications for health.” *Molecular nutrition & food research* 50(9): 847-57.

Wang, Z., D. Neuburg, C. Li, L. Su, J. Y. Kim, J. C. Chen, and D. C. Christiani. 2005. “Global gene expression profiling in whole-blood samples from individuals exposed to metal fumes.” *Environmental health perspectives* 113(2): 233.

Wu, L. L., C.-C. Chiou, P.-Y. Chang, and J. T. Wu. 2004. “Urinary 8-OHdG: a marker of oxidative stress to DNA and a risk factor for cancer, atherosclerosis and diabetics.” *Clinica Chimica Acta* 339(1-2): 1-9.

Table 6.1. Mother's demographic information and household characteristics by stove type

Subject's stove type	Age (in years) Mean ± SD (n)	Cooking time (in hours) Mean ± SD (n)	Kitchen volume (in m^3) Mean ± SD (n)	Most common wood type used n (%)	Homes ≤ 20 meters from road n (%)	n (%) who have completed at least elementary school
Intervention	**30.5 ± 9.3 (39)**	**3.2 ± 1.4 (39)**	**27.5 ± 11.0 (39)**	**Eucalyptus: 16 (45.7)**	**14 (37.8)**	**22 (56.4)**
OPTIMA FL-I[1]	29.2 ± 7.9 (22)	3.1± 1.3 (22)	24.8 ± 9.8 (22)	Other: 9 (45.0)	7 (33.3)	13 (59.1)
OPTIMA FL-II[2]	32.1 ± 10.7 (17)	3.3 ± 1.6 (17)	30.9 ± 11.8 (17)	Eucalyptus: 6 (60.0)	7 (43.8)	9 (52.9)
Control	**30.0 ± 6.7 (38)**	**3.3 ± 1.4 (43)**	**36.0 ± 26.4 (38)**	**Eucalyptus: 17(39.5)**	**19 (44.2)**	**28 (73.7)**
NGO[3]	35.4 ± 3.3 (5)	3.2 ± 1.1 (5)	29.0 ± 13.9 (4)	Other: 3 (42.9)	4 (80.0)	3 (60.0)
Self-improved by household	25.3 ± 3.8 (6)	3.3 ± 1.7 (9)	50.3 ± 40.1 (8)	Other: 5 (55.6)	4 (44.4)	4 (66.7)
Traditional	30.1 ± 7.0 (27)	3.3 ± 1.4 (29)	32.7 ± 21.8 (26)	Eucalyptus: 14 (48.3)	11 (37.9)	21 (77.8)

[1]Functionality level I refers to an OPTIMA stove in good conditions

[2]Functionality level II refers to an OPTIMA stove in need of minor repairs (eg re-plastering) or major repairs (eg chimney valve replacement)

[3]NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO & ADIAR are NGOs that work in nearby communities.

Total sample sizes for all stove types: OPTIMA FL-I=22, OPTIMA FL-II=17, NGO=7, self-improved by household=9 and traditional=29. Information on the total number of subjects who responded to questions on each of the demographic information and characteristics are presented in the table.

Table 6.2. Creatinine corrected urinary oxidative stress measures together with personal and kitchen $PM_{2.5}$ and CO measurements

Sample type	Stove type	8-OHdG (ug/g creatinine)	8-isoprostane (ug/g creatinine)	Kitchen $PM_{2.5}$ (ug/m^3)	Personal $PM_{2.5}$ (ug/m^3)	Kitchen Real Time CO (ppm)	Personal Real Time CO (ppm)	Personal Passive Tube CO (ppm)
Intervention	OPTIMA FL-I[1]	66.7	1.3	70.3	25.4	3.8	2.2	4.9
	OPTIMA FL-I	277.2	1.1	-	22.7	0.4	0.1	0.7
	OPTIMA FL-I	63.5	0.9	453.0	46.1	-	0.3	1.9
	OPTIMA FL-I	154.9	0.4	103.5	65.4	1.1	0.2	0.8
	OPTIMA FL-I	99.4	1.2	-	117.2	1.1	1.3	3.0
	OPTIMA FL-I	80.4	1.6	90.5	37.8	1.5	0.3	0.8
	OPTIMA FL-I	395.3	0.6	-	159.0	2.1	0.3	1.8
	OPTIMA FL-I	34.1	1.3	90.3	84.1	2.8	4.7	0.9
	OPTIMA FL-I	39.1	1.5	-	-	-	-	0.6
	OPTIMA FL-I	198.4	0.3	401.9	170.4	4.7	1.5	-
	OPTIMA FL-I	41.6	0.1	26.2	37.4	0.7	0.1	0.4
	OPTIMA FL-I	557.4	0.8	21.3	39.6	0.3	0.3	0.4
	OPTIMA FL-I	135.3	4.5	9.0	5.1	1.7	1.1	0.7
	OPTIMA FL-I	379.8	1.0	-	-	-	-	12.5
	OPTIMA FL-I	107.1	0.8	195.1	313.3	3.1	-	3.2
	OPTIMA FL-I	184.9	1.3	245.9	275.8	3.6	4.1	6.9
	OPTIMA FL-I	145.5	0.8	-	44.5	-	-	1.0
	OPTIMA FL-I	2270.0	3.2	56.3	55.0	0.9	0.1	0.6
	OPTIMA FL-I	29.0	0.2	674.7	183.2	22.0	4.2	7.5
	OPTIMA FL-I	130.4	0.5	-	40.9	0.4	0.4	0.4
	OPTIMA FL-I	111.9	0.3	35.2	-	2.7	1.0	1.0
	OPTIMA FL-I	65.2	4.5	-	-	3.6	1.2	2.2
	GM (95% CI)*	**130.1 (82.5,**	**0.9 (0.6, 1.3)**	**92.8 (44.9,**	**62.5 (37.7,**	**1.8 (1.0,**	**0.6 (0.3,**	**1.4 (0.9,**

		205.2)		**191.8)**	**103.6)**	**3.0)**	**1.2)**	**2.3)**
Intervention	OPTIMA FL-II[2]	140.7	0.6	114.3	36.3	2.3	1.0	1.0
	OPTIMA FL-II	89.2	0.6	118.0	30.1	2.3	0.2	0.8
	OPTIMA FL-II	220.0	2.5	145.4	17.5	4.0	1.9	1.5
	OPTIMA FL-II	163.3	0.5	23.0	44.5	0.9	0.5	1.0
	OPTIMA FL-II	451.6	1.1	117.8	27.9	7.6	1.7	1.0
	OPTIMA FL-II	132.4	2.2	50.0	92.5	-	0.9	0.8
	OPTIMA FL-II	45.0	2.3	161.0	209.6	3.3	1.4	3.2
	OPTIMA FL-II	93.3	0.6	-	20.3	0.4	0.2	0.8
	OPTIMA FL-II	463.1	1.3	238.5	85.8	6.4	1.5	1.7
	OPTIMA FL-II	91.6	0.2	154.3	84.7	4.6	3.4	1.9
	OPTIMA FL-II	220.3	0.5	40.9	67.8	3.6	1.0	-
	OPTIMA FL-II	158.0	0.2	-	75.2	1.8	1.1	0.9
	OPTIMA FL-II	227.3	0.9	223.4	162.4	3.5	1.3	1.7
	OPTIMA FL-II	134.5	1.2	436.3	375.4	6.4	3.4	4.0
	OPTIMA FL-II	11.2	1.2	-	360.5	12.2	4.5	7.8
	OPTIMA FL-II	132.9	0.6	-	531.3	22.9	-	7.2
	OPTIMA FL-II	263.0	0.8	-	159.1	24.0	7.4	8.0
	GM (95% CI)*	**136.6 (87.4, 213.5)**	**0.8 (0.5, 1.2)**	**116.5 (69.3, 195.9)**	**85.1 (49.8, 145.4)**	**4.0 (2.3, 7.2)**	**1.3 (0.8, 2.2)**	**1.9 (1.2, 2.9)**
Control	NGO[3]	103.5	0.3	539.2	391.4	4.9	2.6	3.9
	NGO	43.8	0.4	-	-	6.6	3.6	4.0
	NGO	193.9	0.9	-	-	3.6	-	3.0
	NGO	263.1	0.8	74.9	27.0	24.0	3.0	2.1
	NGO	155.0	1.0	-	-	-	-	7.3
	NGO	133.9	0.6	43.5	8.1	-	0.4	1.0

NGO	21.4	0.8	-	597.3	32.4	9.4	12.1
GM (95% CI)*	**100.4(44.3, 227.8)**	**0.7 (0.5, 0.9)**	**120.7 (4.5, 3239.7)**	**84.7 (3.1, 2309.5)**	**9.8 (2.9, 33.3)**	**2.6 (0.6, 10.5)**	**3.7 (1.7, 7.7)**
Self-improved by household	104.1	1.6	83.3	73.2	-	-	1.0
Self-improved by household	72.6	0.3	547.7	77.7	29.9	0.8	2.1
Self-improved by household	25.1	1.0	111.4	46.7	1.3	0.4	0.7
Self-improved by household	946.1	1.4	298.3	70.3	3.1	0.1	0.7
Self-improved by household	347.1	0.6	37.0	111.6	0.3	0.5	0.6
Self-improved by household	132.8	1.2	540.1	21.2	5.3	0.6	7.2
Self-improved by household	54.5	1.9	17.4	24.0	8.8	0.4	1.6
Self-improved by household	133.1	1.8	-	11.5	2.1	0.2	1.1
Self-improved by household	969.1	0.9	100.5	112.8	1.1	0.5	1.7
GM (95% CI)*	**157.0 (60.1, 410.0)**	**1.1 (0.7, 1.6)**	**123.2 (43.9, 345.8)**	**48.1 (25.9, 89.3)**	**2.8 (0.8, 9.2)**	**0.3 (0.2, 0.7)**	**1.3 (0.7, 2.4)**
Traditional	97.9	0.5	366.6	298.4	8.2	5.0	6.8
Traditional	315.8	0.8	225.5	43.0	14.1	0.5	3.0
Traditional	45.4	0.4	-	720.7	23.4	8.4	7.6
Traditional	262.8	1.3	107.4	135.0	1.8	0.5	4.1
Traditional	693.4	0.1	257.1	244.6	3.7	2.9	3.8
Traditional	189.8	0.6	-	-	-	-	13.2
Traditional	69.3	2.4	-	-	-	-	12.6

Traditional	104.7	0.4	179.4	-	0.2	0.9	1.0
Traditional	107.7	0.7	150.9	98.8	2.8	0.8	1.5
Traditional	50.1	0.4	54.9	95.2	0.5	0.8	0.8
Traditional	241.3	1.1	54.6	195.6	1.0	0.3	0.4
Traditional	86.8	0.6	146.7	-	0.1	0.2	0.6
Traditional	65.9	0.4	-	-	-	-	7.0
Traditional	112.9	0.5	-	52.0	0.6	0.5	0.9
Traditional	126.5	0.8	-	149.1	2.3	0.7	1.7
Traditional	120.2	1.0	360.7	391.3	4.6	3.6	3.8
Traditional	99.5	0.8	-	-	8.0	2.0	3.0
Traditional	120.6	0.5	53.2	63.6	1.4	0.4	0.5
Traditional	173.6	1.0	717.1	195.0	16.1	0.7	-
Traditional	162.8	0.6	207.0	128.5	3.7	-	2.0
Traditional	209.3	0.9	82.1	29.3	1.1	0.1	0.4
Traditional	555.3	0.7	-	206.2	2.5	2.2	0.8
Traditional	214.6	0.7	152.7	101.8	2.6	2.2	3.1
Traditional	98.3	0.9	930.9	162.8	17.0	2.0	3.1
Traditional	254.8	1.0	-	-	-	0.3	0.6
Traditional	328.1	1.1	-	275.9	24.2	4.2	8.7
Traditional	122.2	0.8	-	-	1.5	1.5	1.9
Traditional	99.2	1.2	233.4	149.5	2.1	2.4	3.3
Traditional	131.8	0.5	67.4	130.4	0.9	0.5	0.9
GM (95% CI)*	**145.2 (113.5, 185.8)**	**0.7 (0.5, 0.9)**	**170.1 (111.9, 258.7)**	**141.5 (100.5, 199.3)**	**2.6 (1.4, 4.6)**	**1.0 (0.7, 1.6)**	**2.1 (1.4, 3.2)**

*GM (95% CI) refers to geometric mean (95% confidence interval)

[1]Functionality level I refers to an OPTIMA stove in good conditions

[2]Functionality level II refers to an OPTIMA stove in need of minor repairs (eg re-plastering) or major repairs (eg chimney valve replacement)

[3]NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO & ADIAR are NGOs that work in nearby communities.

Table 6.3. Correlation analysis between urinary oxidative stress measures, exposure measurements and hydroxylated PAHs. Pearson's correlation coefficients are provided, with p values in parenthesis and sample sizes italized

Creatinine corrected (ug/g creatinine)										
		log8-OHdG	log8-isoprostane	log1-NAP	log2-NAP	log2-FLU	log3-FLU	log9-FLU	log1-PHE	log2-PHE
log8-OHdG (ug/g creatinine)	r (p) n	1	0.08 (0.49) 84	-0.02 (0.87) 83	-0.05 (0.64) 84	-0.07 (0.55) 84	-0.02 (0.81) 84	-0.03 (0.81) 84	-0.01 (0.94) 84	-0.01 (0.91) 84
log8-isoprostane (ug/g creatinine)	r (p) n		1	0.03 (0.77) 83	0.11 (0.33) 84	0.14 (0.19) 84	0.10 (0.34) 84	0.10 (0.38) 84	0.18 (0.10) 84	0.20 (0.06) 84

Creatinine corrected (ug/g creatinine)						logKPM[1]	logMPM[2]	logMRCO[3]	logKRCO[4]	logMTCO[5]
		log8-OHdG	log8-isoprostane	log3-PHE	log1-PYR	(ug/m3)	(ug/m3)	ppm	ppm	ppm
log8-OHdG (ug/g creatinine)	r (p) n	1	0.08 (0.49) 84	-0.02 (0.89) 84	-0.03 (0.77) 84	-0.07 (0.62) 55	-0.11 (0.36) 69	**-0.29** **(0.01)** **73**	-0.17 (0.14) 73	-0.19 (0.09) 81
log8-isoprostane (ug/g creatinine)	r (p) n		1	0.18 (0.11) 84	0.11 (0.31) 84	-0.17 (0.22) 55	-0.25 **(0.04)** 69	-0.08 (0.50) 73	0.04 (0.72) 73	0.01 (0.94) 81

2 MPM refers to 48hr personal PM2.5 exposures

3 MRCO refers to 48hr real time personal CO exposures

4 KRCO refers to 48hr real time kitchen CO concentrations

5 MTCO refers to 48hr time integrated personal CO exposures

1-NAP, 2-NAP, 2-FLU, 3-FLU, 9-FLU, 1-PHE, 2-PHE, 3-PHE and 1-PYR refer to 1-naphthol, 2-naphthol, 2-hydroxyfluorene, 3-hydroxyfluorene, 9-hydroxyfluorene, 1-hydroxyphenanthrene, 2-hydroxyphenanthrene, and 1-hydroxypyrene respectively.

Statistically significant p values are in bold

Table 6.4. Test statistics from individual general linear models for variables associated with urinary 8-OHdG and 8-isoprostane. Only one predictor variable is included in each model, together with a specific oxidative stress biomarker as a response variable.

Parameter	Degrees of freedom[1]	8-OHdG		8-isoprostane	
		Overall F statistic	P value	Overall F statistic	P value
Continuous variables					
Cooking time	1, 78	5.55	**0.02**	0.62	0.43
48hr time integrated personal $PM_{2.5}$*	1, 67	0.87	0.36	4.54	**0.04**
30-second maximum personal CO*	1, 72	12.86	**0.001**	4.39	**0.04**
15-minute maximum personal CO*	1, 72	8.46	**0.005**	1.65	0.20
90-minute maximum personal CO*	1, 72	6.25	**0.01**	1.85	0.18
48hr time integrated personal CO*	1, 72	6.59	**0.01**	0.46	0.50
Categorical variables					
Age	2, 72	2.36	0.10	0.24	0.79
≤ 25 years (reference: ≥ 36 years)			0.51		1.00
26 - 35 years			**0.04**		0.57
Distance of home to road	1, 78	0.85	0.36	4.56	**0.04**
≤ 20 meters (reference: > 20 meters)					
Eating food exposed to fire	1, 78	3.27	0.07	0.19	0.66
No (reference: yes)					
Stove type	4, 79	0.31	0.87	1.00	0.41
OPTIMA FL-II[2] (reference: OPTIMA FL-I)			0.87		0.60
NGO[3]			0.51		0.31
Self-improved by household			0.60		0.54
Traditional			0.66		0.18
Traffic in the community[1]	2, 67	4.58	**0.01**	2.51	0.09
Frequently (reference: seldomly)			**0.004**		0.16
Hourly			0.34		**0.03**
Wood type	2, 75	0.16	0.86	1.39	0.26
Eucalyptus (reference: other wood			0.86		0.15

types)

Hualango	0.70	0.97

[1]Degrees of freedom for model and error respectively

[2]Functionality levels I and II refer to OPTIMA stoves in need of minor repairs (eg re-plastering) or major repairs (eg chimney valve replacement)

[3]NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO & ADIAR are NGOs that work in nearby communities.

*Indicates negative regression coefficients for exposure measurements

Table 6.5. Variables associated with urinary biomarkers of oxidative stress

Variables that are significantly associated with urinary 8-OHdG.

Variable	Estimate, in ug/g creatinine	95% CI, in ug/g creatinine
Traffic in the community[1]		
Frequently (n=12)	333.5	199.0, 559.0
Hourly (n=27)	148.3	102.8, 214.1
Seldomly (n=23)	133.6	93.7, 190.5
Eating food exposed to fire		
No (n=51)	140.7	111.7, 177.0
Yes (n=11)	250.4	151.0, 415.2

After controlling for traffic and eating food exposed to fire	Correlation coefficent	P value
Cooking time[2] (n=80)	0.29	0.01
48hr time integrated personal CO (n=73)	-0.26	0.03
90-minute maximum personal CO (n=73)	-0.23	0.06
15-minute maximum personal CO (n=73)	-0.25	0.04
30-second maximum personal CO (n=73)	-0.32	0.01

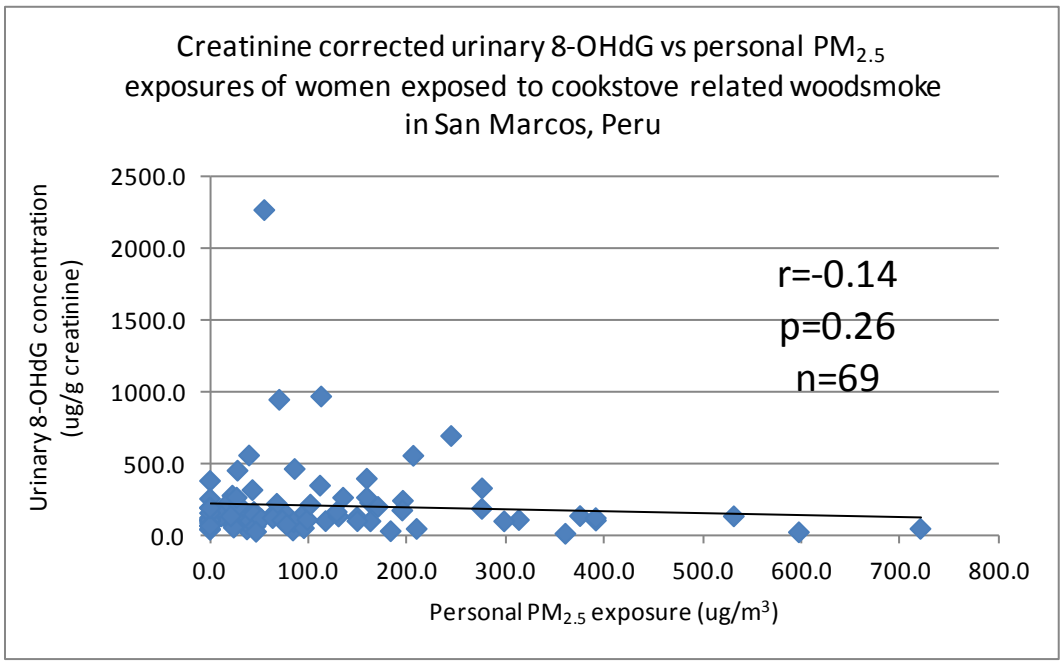
Variables that are significantly associated with urinary 8-isoprostane.

Variable	Estimate, in ug/g creatinine	95% CI, in ug/g creatinine
Distance of home to road		
≤ 20 meters (n=25)	0.8	0.6, 1.1
> 20 meters (n=38)	0.6	0.5, 0.7

After controlling for distance of home to road	Correlation coefficent	P value
1-hyroxyphenanthrene (n=84)	0.21	0.05
2-hyroxyphenanthrene (n=84)	0.21	0.06
48hr time integrated personal $PM_{2.5}$ (n=69)	-0.21	0.09

[1]Traffic in the community: frequently refers to a car every few minutes; Hourly refers to a car every hour; Seldomly refers a car every few days.

[2]Cooking time refers to the estimated cooking time of mothers in study region spent cooking (in hours). Each subject's time has been centered around the mean cooking time.



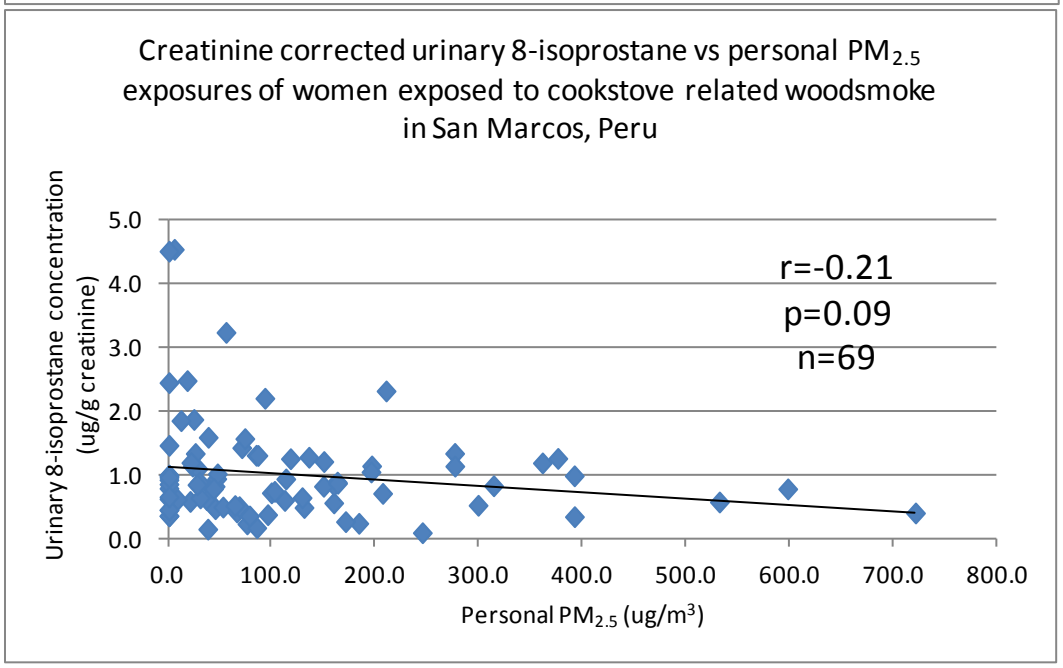


Figure 6.1. Urinary oxidative stress measures vs. $PM_{2.5}$ exposures of women exposed to woodsmoke in San Marcos, Peru. The association has been adjusted for distance of subject's home to the road.

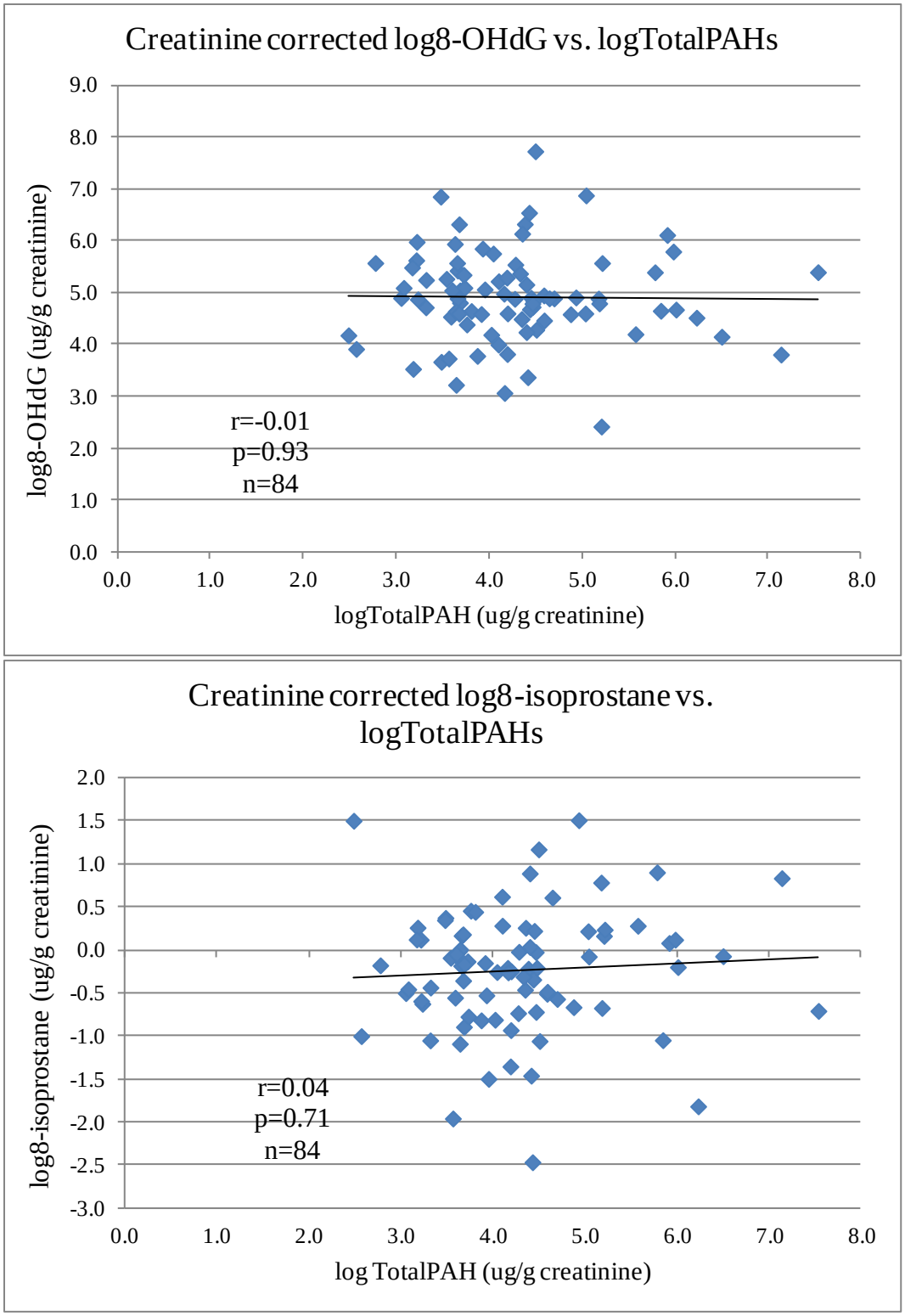


Figure 6.2. Urinary oxidative stress measures vs. total urinary hydroxy-PAH exposures of women exposed to woodsmoke in San Marcos, Peru. The association has been adjusted for distance of subject's home to the road.

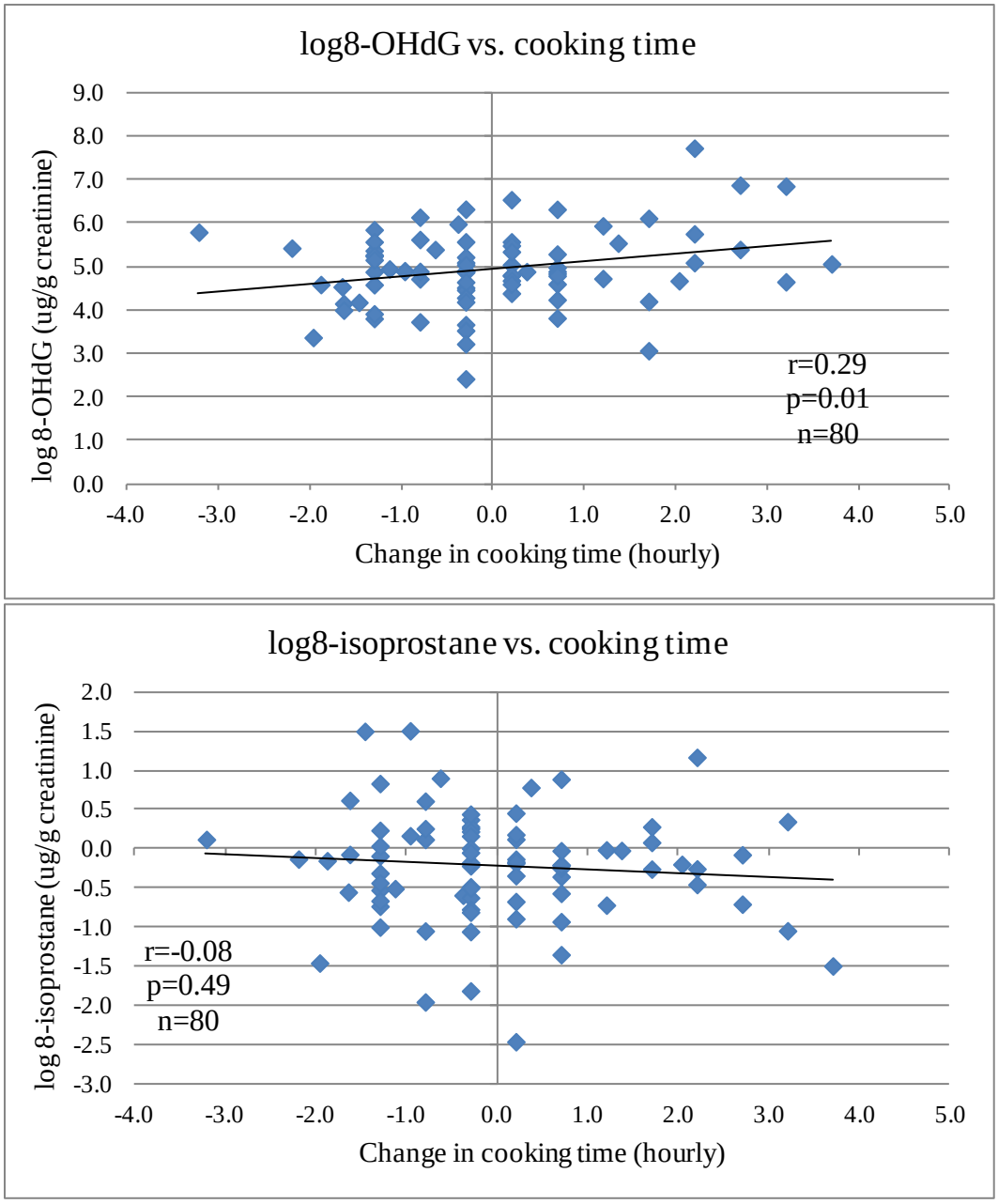


Figure 6.3. Urinary oxidative stress measures vs. cooking time of women exposed to woodsmoke in San Marcos, Peru. The association has been adjusted for the amount of vehicular traffic in subjects' communities and eating food exposed to fire.

CHAPTER 7

A PILOT STUDY CHARACTERIZING REAL TIME EXPOSURES TO PARTICULATE MATTER AND CARBON MONOXIDE FROM COOKSTOVE RELATED WOODSMOKE IN RURAL PERU[1]

[1] Commodore, A. A., Hartinger, S.M., Lanata, C.F., Mäusezahl, D., Gil, A.I., Hall, D.B., Aguilar-Villalobos, M., Naeher, L.P. To be submitted to *Atmospheric Environment*.

ABSTRACT

Background: Nearly half of the world's population is exposed to household air pollution (HAP) due to long hours spent in close proximity to unvented cooking fires.

Objectives: We aimed to use $PM_{2.5}$ and CO measurements from households in San Marcos, Peru to characterize exposure to cookstove generated woodsmoke in real time among control and intervention households.

Methods: Real time personal particulate matter with an aerodynamic diameter ≤2.5 μm ($PM_{2.5}$), and personal and kitchen carbon monoxide (CO) samples were taken in control (n=10) and intervention households (n=9) in San Marcos, Cajamarca region, Peru. Control households used a number of stoves including open fire and chimney stoves while intervention households used study-promoted chimney stoves. Measurements were categorized into lunch (9am – 1pm) and dinner (3pm – 7pm) periods, where applicable, to adjust for a wide range of sampling periods (2.8–13.1hrs).

Results: There were no statistically significant differences in exposure measurements between control and intervention stove users. Mean (95% CI) personal $PM_{2.5}$ exposures were 557.9 (0, 1404.7) $\mu g/m^3$ and 396.8 (0, 840.1) $\mu g/m^3$ in intervention and control homes respectively. Likewise personal CO exposures were 4.9 (0, 12.5) ppm and 2.8 (1.0, 5.0) ppm, and kitchen CO concentrations were 12.3 (0, 30.0) ppm and 10.8 (2.0, 15.2) ppm in intervention and control homes respectively.

For all study subjects, median (95% CI) personal $PM_{2.5}$ exposures ranged from 0.8 (0, 0.8) $\mu g/m^3$ to 1579.3 (1321.3, 1737.1) $\mu g/m^3$ during lunch (n=11) and 0.8 (0.8, 0.8) $\mu g/m^3$ to 4267.7 (3545.9, 5413.1) $\mu g/m^3$ during dinner (n=17). Likewise personal CO exposures ranged from 0 ppm to 12.0 (12.0, 13.0) ppm during lunch (n=11) and from 0 ppm to 31.5 (26.0, 37.0) ppm

during dinner (n=17). Kitchen CO levels ranged from 0 ppm to 85.5 ppm (71.0, 95.0) ppm during lunch (n=9) and from 0 ppm to 46.0 (41.0, 51.0) ppm during dinner (n=15).

During the 4hr time periods, mean personal $PM_{2.5}$ exposures were correlated with personal CO exposures during lunch (r=0.67 p=0.024 n=11) and dinner (r=0.72 p=0.0011 n=17) in all study households. Personal $PM_{2.5}$ exposures and kitchen CO concentrations were also correlated during lunch (r=0.76 p=0.018 n=9) and dinner (r=0.60 p=0.018 n=15). Median measurements during the same time periods were also either significantly or marginally correlated ($p<0.1$).

Conclusion: Although there were no statistically significant differences in exposure measurements between control and intervention stove users, results suggest that CO may be a useful indicator of PM during 4hr time scales measured in real time and particularly during high PM exposures.

INTRODUCTION

Cooking with solid fuels such as wood over inefficient stoves leads to exposure to products of incomplete combustion in the domestic environment (World Health Organization: [WHO] 2011). Nearly half of the world's population employs biomass as fuel for cooking (Kurmi et al 2010, Naeher et al 2007, Smith 1987). Particularly, women and their young infants experience high household air pollution (HAP) exposures due to long hours spent in close proximity to improperly vented cooking fires (Ezzati and Kammen 2002, Martin et al 2011). Adequate characterization of exposure to residential biomass combustion is crucial in vulnerable populations which use biomass fuels on a daily basis and spend a majority of time indoors.

HAP resulting from incomplete biomass combustion contains health-damaging pollutants such as polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), aldehydes, carbon monoxide (CO) and particulate matter with an aerodynamic diameter of $\leq$2.5 μm ($PM_{2.5}$) (Bølling et al 2009; Jalava et al 2010). CO and $PM_{2.5}$ are major constituents and are considered chief inhalation hazards of woodsmoke exposure (Naeher et al 2007). Adverse health effects associated with exposure to biomass combustion products include upper and lower respiratory symptoms, lung cancer, chronic obstructive pulmonary disease, lung function decline, cardiovascular disease, low birth weight (Fullerton et al 2008, Romieu et al 2009, WHO 2011). Recent studies have successfully demonstrated the use of time integrated personal $PM_{2.5}$ and real time CO monitoring instruments to quantify woodsmoke exposure in the indoor environment (Armendáriz-Arnez et al 2008, Chengappa et al 2007, Dutta et al 2007, Fitzgerald et al 2012, Fullerton et al 2009, Hartinger et al in review, Kurmi et al 2008, Masera et al 2007, Northcross et al 2010).

Studies on real time monitoring of personal $PM_{2.5}$ exposures are however lacking particularly during periods of highest exposures including meal preparation. There is also limited data on measurements of HAP surrogates that are correlated in real time (Li et al 2012).

The aim of this work is to use $PM_{2.5}$ and CO measurements from households in San Marcos, Peru to characterize exposure to cookstove generated woodsmoke in real time among control and intervention households. Additionally, we examine correlations between personal $PM_{2.5}$ and CO exposures and kitchen concentrations in this population during 4hr periods when subjects are involved in meal preparation. To the best of our knowledge this is the first study to employ real time personal $PM_{2.5}$ monitoring in this region in Peru.

METHODS

Study Design and Study Homes

Measurements presented in this paper occurred between June and August 2009 as part of a community based randomized control trial (c-RCT) (Hartinger et al 2011). Intervention homes used OPTIMA-improved stoves (hereafter OPTIMA stoves). After air sampling had occurred, OPTIMA stoves were categorized based on their levels of functionality. OPTIMA FL-I refers to OPTIMA stoves in good conditions and OPTIMA FL-II refers to OPTIMA stoves in need of minor repairs (e.g. re-plastering) or major repairs (e.g. chimney valve replacement). Air sampling did not take place to measure HAP levels post repairs.

Control homes in the c-RCT used various stoves including chimney stoves whose raw materials are provided by nongovernmental organizations (hereafter referred to as NGO), chimney stoves built by the households themselves (hereafter referred to as self-improved by household), gas stoves, and non-vented stoves with openings at the top for pots including the common three stone open fire stove (hereafter referred to as traditional) (Hartinger et al in review, Commodore et al in review).

In this cross sectional study, control and intervention households were selected from participating households in the c-RCT. Twenty subjects (11 control and 9 intervention stove users) were measured for cookstove related $PM_{2.5}$ exposures in real time. Data from one subject has been excluded from the final data set due to a short sampling duration (17 min). Subjects wore exposure monitoring equipment placed in vests which were worn in their breathing zones (Figure 1 A). These vests held real time CO monitors and 48hr time integrated $PM_{2.5}$ samplers for personal air sampling and the data for these measurements are presented elsewhere (Hartinger et al in review).

Exposure Monitoring

Real time $PM_{2.5}$ exposure was monitored with the Sidepak AM510 (TSI Inc, Shoreview, MN). The equipment is a size selective laser photometer designed to read the mass concentration of particulate matter (TSI Inc, 2006). Sidepaks used in this study were fitted with a 2.5 micron impactor, and set to log $PM_{2.5}$ concentrations every 30 seconds. Sidepaks were zero calibrated with a HEPA filter before each use. Data was only available for as long as the Sidepaks battery power lasted (range of sampling duration presented in Table 1). Logged data were retrieved from the equipment using *Trakpro v. 4.4.0*.5.

Real time exposure to CO was measured using Dräger Pac III single gas monitors (Draeger Safety Inc, Pittsburgh, PA) outfitted with CO sensors. The CO monitors were calibrated before the study at 0 and 50 ppm using pure nitrogen and 50 ppm CO gases respectively (Calgaz, Air Liquide America Corp, Cambridge, MA). CO monitors were also set to log data every 30 seconds. Personal CO monitors were worn in the breathing zones of subjects while kitchen monitors were placed 1.5m from the ground in the subject's kitchen. Logged data were retrieved from the equipment using *Gas Vision v. 4.5*. Unlike the Sidepak measurements, real time CO data was available for a total of 48hrs (Hartinger et al in review). Data presented for each household in this study (n=19) reports the sampling durations with corresponding real time $PM_{2.5}$ measurements. Baseline questionnaires were also administered (Hartinger et al in review, Commodore et al in review).

Statistical Analysis

SAS version 9.2 (SAS Institute, NC, USA) was used for all data analysis. Raw personal $PM_{2.5}$ exposures ranged from 0 to 20, 000 $\mu g/m^3$ (upper and lower limit of the Sidepak), and after a calibration factor of 0.77 was applied (Jiang et al 2011), the upper limit was 15, 400 $\mu g/m^3$. Real time exposures were averaged over the corresponding sampling duration in each household (hereafter termed mean). Measurements were also categorized into lunch (9am – 1pm) and dinner (3pm - 7pm) periods, where applicable, to adjust for a wide range of sampling periods (2.8–13.1hrs).

Study subjects in the c-RCT typically prepared breakfast from 5am – 8am. Typically there was an hour in between and then the next meal cycle would begin. Lunch preparation was typically from 9am to 1pm.

Dinner preparation then started by 3pm or later. We use descriptive measures to characterize personal $PM_{2.5}$ and CO exposures and kitchen concentrations by stove type. SAS PROC UNIVARIATE was used to calculate medians and 95^{th} percentiles along with corresponding 95% confidence intervals during lunch and dinner periods.

Finally, to examine how well $PM_{2.5}$ and CO measurements are correlated in this sampled population, of the following procedures were employed. First, to determine how well PM and CO are correlated in time, PROC RANK was used to create 4h mean and median measurement rankings and PROC CORR was used to calculate correlation coefficients. Spearman's rank correlation coefficients (r) between 4hr mean and median personal $PM_{2.5}$, CO exposures and CO concentrations are calculated separately for lunch and dinner sampling periods. Correlation coefficients are only presented for all stove types rather than by intervention arm or specific stove types due to sample size constraints.

RESULTS

Household characteristics

Characterization of the entire study population is found elsewhere (Hartinger et al 2011, Hartinger et al in review). Household information is presented according to stove type (Table 7.1). Mean sampling was 9.9 hours (SD: 2.3 hours, range: 2.8 – 13.1 hours). Figure 7.1 captures an OPTIMA stove user wearing vest which encases a Sidepak, a Pac III and a personal sampling pump (A). Stoves built by an NGO, self-improved by household and a traditional three stone open fire are shown in B, C and D respectively. Summary statistics of personal $PM_{2.5}$, personal CO and kitchen CO measurements are presented (Table 7.2) and during lunch and dinner sampling periods (Table 7.3).

Overall mean (95% CI) personal $PM_{2.5}$ exposures were 557.9 (0, 1404.7) µg/m^3 and 396.8 (0, 840.1) µg/m^3 in intervention and control homes respectively. Likewise personal CO exposures were 4.9 (0, 12.5) ppm and 2.8 (1.0, 5.0) ppm, and kitchen CO concentrations were 12.3 (0, 30.0) ppm and 10.8 (2.0, 15.2) ppm in intervention and control homes respectively. Mean and median concentrations for all measurements by intervention stove type (OPTIMA FL-I and OPTIMA FL-II) and control stove type (NGO, self-improved by household and traditional) are provided in the subsequent results.

Personal $PM_{2.5}$

Median (95% CI) personal $PM_{2.5}$ exposures among five OPTIMA FL-I stove users ranged from 6.2 (6.2, 7.9) µg/m^3 to 279.5 (224.8, 321.9) µg/m^3 during lunch and 0.8 (0.8, 0.8) µg/m^3 to 7.7 (6.9, 8.5) µg/m^3 during dinner (Table 7.3). One OPTIMA FL-II stove user had median personal $PM_{2.5}$ exposures of 653.7 (632.2, 703.1) µg/m^3 during one and other had 6.2 (6.2, 6.9) µg/m^3 during dinner (Table 7.3). The highest lunch and dinner median $PM_{2.5}$ exposures belonged in the OPTIMA FL-II group, with measurements of 1579.3 (1321.3, 1737.1) µg/m^3 and 2731.6 (2371.6, 3379.5) µg/m^3 respectively (Table 7.3).

Among control stove users, median personal $PM_{2.5}$ exposures among subjects who used traditional stoves (n=5) ranged from 0.8 (0, 0.8) µg/m^3 to 576.7 (459.7, 683.8) µg/m^3 during lunch and 8.5 (8.5, 8.5) µg/m^3 to 4267.7 (3545.9, 5413.1) µg/m^3 during dinner (Table 7.3). Measurements of 10 (9.2, 13.9) µg/m^3 and 50.4 (42.4, 65.5) µg/m^3 was obtained from the only home with a stove self-improved by the household during lunch and dinner respectively (Table 7.3). NGO stove users had median $PM_{2.5}$ exposures between 0 µg/m^3 (IQR=6.2 µg/m^3) to 148.6 (101.6, 177.1) µg/m^3 during dinner (Table 7.3).

Approximately 90.9% (10/11) of 4hr mean $PM_{2.5}$ exposures during lunch and 58.8% (10/17) during dinner (Table 7.3), were clearly above the 24hr US EPA ambient air quality standard of 35 $\mu g/m^3$ and the WHO's guideline of 25 $\mu g/m^3$. Table 7.4 presents 95^{th} percentiles of exposure for each meal time for each subject to give an indication not only of the typical exposure (Table 7.3), but also the extreme level of exposure resulting from each stove. These extremes are highly variable both within and across stove types and do not yield a clear superiority of one stove type over another.

Personal CO

Median (95% CI) personal CO exposures among three OPTIMA FL-I stove users ranged from 0 to 2.0 (2.0, 3.0) ppm during lunch. OPTIMA FL-II stove users (n=2) had median personal CO exposures ranging from 0 ppm to 1.0 (1.0, 1.0) ppm (Table 7.3). Dinner median CO exposures for OPTIMA FL-I and FL-II stove users were 0 ppm (Table 7.3). Again, an OPTIMA FL-II stove user had the highest lunch and dinner median personal CO exposures of 12.0 (12.0, 13.0) ppm and 31.5 (26.0, 37.0) ppm respectively.

Among control stove users, subjects who used NGO stoves (n=4) had no lunch measurements while dinner exposures ranged from 0 ppm to 3.0 (3.0, 3.0) ppm. Among traditional stove (n=5) users, lunch CO exposures ranged from 0 to 5.0 (5.0, 6.0) ppm while dinner exposures ranged from 0 ppm to 21.0 (18.0, 24.0). The only user with a stove which was self-improved by the household had 0 ppm lunch CO exposure and 1.0 (1.0, 1.0) ppm median dinner exposure (Table 7.3).

In all study homes 11.5% (3/26) of 4hr mean personal CO exposures were above 8hr ambient US EPA CO standard of 9 ppm and the WHO's 8hr guideline of 8.7 ppm. During lunch and dinner respectively, 9.1% (1/11) and 11.7% (2/15) of 4hr mean personal CO were above US

EPA and WHO limits (Table 7.3). The 95th percentiles of CO exposures also reveal the variability in household personal CO exposures and present the values of the top 5% of the measurements (Table 7.4).

Kitchen CO

Median (95% CI) kitchen CO concentrations among three households with OPTIMA FL-I stoves ranged from 0 to 10.0 (9.0, 11.0) ppm during lunch (Table 7.3). The only household with OPTIMA FL-II with both lunch and dinner measurements had the highest median kitchen CO of 85.5 (71.0, 95.0) ppm and 46.0 (41.0, 51.0) ppm respectively.

Among control households, kitchens with NGO stoves (n=4) had no lunch measurements while dinner kitchen CO levels ranged from 0 ppm to 13.0 (12.0, 15.0) ppm. In households with traditional stoves (n=5), kitchen lunch CO ranged from 0 to 22.0 (20.0, 24.0) ppm while dinner measurements ranged from 0 ppm to 33.5 (31.0, 35.0). The only kitchen with a self-improved by household stove had CO concentrations of 1.0 (0, 1.0) ppm and 0 ppm during lunch and dinner respectively (Table 7.3). In all study homes 29.2% (7/24) of 4hr mean kitchen CO concentrations were above US EPA and WHO limits of 9 ppm and 8.7 ppm respectively (Table 7.3). Specifically during lunch and dinner respectively, 33.3% (3/9) and 26.7% (4/15) of kitchen CO concentrations (Table 7.3), were above US EPA and WHO limits. The 95th percentiles of CO exposures also reveal the variability in kitchen CO among in different households and present the values of the top 5% of the measurements (Table 7.4).

Correlations between $PM_{2.5}$ and CO

In all study homes, 4hr mean personal $PM_{2.5}$ exposures were correlated with personal CO exposures during lunch (r=0.67 p=0.024 n=11) and dinner (r=0.72 p=0.0011 n=17). Personal $PM_{2.5}$ exposures and kitchen CO concentrations were also correlated during lunch (r=0.76

p=0.018 n=9) and dinner (r=0.60 p=0.018 n=15). Four hour median personal $PM_{2.5}$ exposures were marginally correlated with personal CO exposures during lunch (r=0.56 p=0.07 n=11) and significantly correlated during dinner (r=0.64 p=0.006 n=17) in all study households. Personal $PM_{2.5}$ exposures and kitchen CO concentrations were also marginally correlated during lunch (r=0.59 p=0.09 n=9) and significantly correlated dinner (r=0.81 p=0.0003 n=15).

Temporal profiles for personal $PM_{2.5}$ vs. personal CO and personal $PM_{2.5}$ vs. kitchen CO are presented for a traditional (Figure 7.2 A and B) and an OPTIMA FL-II stove user (Figure 7.2 C and D). The plots reveal the close similarity in the temporal pattern of exposure between the two air pollutants particularly during high exposure periods. In all study households, mean personal $PM_{2.5}$ exposures were marginally correlated with personal CO exposures (r=0.41 p=0.08 n=19) and significantly correlated with kitchen CO levels (r=0.56 p=0.03 n=16). On the other hand, median personal $PM_{2.5}$ exposures were correlated with personal or kitchen CO: r=0.27 p=0.27 n=19 and r=0.06 p=0.83 n=16 respectively.

DISCUSSION

We present personal $PM_{2.5}$ exposure, personal CO and kitchen CO measurements in real time from 9 chimney stove intervention and 10 control homes in San Marcos Peru. Although limited by small sample size, our results add to the growing evidence that the use of biomass fuels results in elevated HAP levels, which have been reported to be associated with adverse health effects (Kim et al 2011, Smith et al 2011, Smith and Peel 2010).

The levels observed in our study during cooking are typical when compared to measurements from around the world. Previous studies have measured kitchen CO between 10 and 500 ppm during stove use (Von Schirndig et al 2002). In India a range of 500 to 2000 $\mu g/m^3$ has been recorded for $PM_{2.5}$ during cooking (Balakrishnan et al 2002).

The feasibility of using stove functionality as a potential HAP exposure surrogate has been demonstrated among traditional and improved stove users in Honduras (Clark et al 2010). A four-level subjective scale revealed 8hr (geometric mean) personal $PM_{2.5}$ exposures of 60 ug/m^3, 71 ug/m^3, 108 ug/m^3 and 202 ug/m^3 for high quality, high mid quality, low-mid quality and low quality stoves respectively (Clark et al 2010).

The lack of statistically significant differences in measurements between control stove and intervention stove users however is atypical in comparison to other stove intervention studies. In Mexico, subjects who used the Patsari improved stove together with a traditional open fire had median real time personal 24hr $PM_{2.5}$ exposures of 190 µg/m^3 and 48hr kitchen median concentration of 300 µg/m^3 (Armendáriz-Arnez et al 2008). In Peru subjects using two types of chimney stoves had 48hr $PM_{2.5}$ exposures of 68.4 and 58.3 ug/m^3 (Fitzgerald et al 2012).

The study by Armendáriz-Arnez et al, together with other pre/post stove intervention studies (for example Fitzgerald et al), was shortly after stove installation (≤ one month). On the other hand, other stoves such as the improved Plancha stoves in Guatemala (Smith et al 2010) were maintained periodically. In the current study, the improved stoves had been in use for an average of seven months and some were not adequately maintained by subjects. Possible reasons for high HAP exposure among intervention stove users in our study include maintenance over time, stove design, proper and exclusive stove use (Commodore et al in review).

$PM_{2.5}$ has been identified as the best single indicator of the health effects of combustion of biomass such as wood (Naeher et al 2007, Perez-Padilla et al 2010). During the specified 4hr lunch and dinner periods in all study homes, approximately 71% of mean personal $PM_{2.5}$ exposures were higher than 24hr air quality limits for $PM_{2.5}$ set by regulatory and international agencies. These standards for PM may also be applied to the indoor environment, specifically in

the developing world, where large populations are exposed to high levels of combustion particles derived from indoor cookstoves (Martin et al 2011). Although results from our study are not within the same time frame as these air quality limits, the potential for cumulative exposures and decrease in recovery time on a daily basis exist in our study population. It then becomes evident meeting these limits for a 24hr period can protect against HAP peaks capable of causing substantial excess morbidity and mortality (WHO 2006).

Mean personal $PM_{2.5}$ and personal CO measurements were well correlated in all study homes, particularly during 4hr cooking periods where HAP exposures were high. This agrees with literature that over time, CO can be used as an indicator of PM depending on the HAP source or cooking activity (McCracken and Smith 1998; Naeher et al 2001; Northcross et al 2010). When median values were considered in the correlation analysis, there was marginal statistical significance. This is probably due to the smaller values of median measurements compared to mean values derived over the 4hr periods.

The values of the correlation coefficients were significantly higher in homes high cookstove activity whereas in homes with little cookstove activity, the correlations were lower and/or insignificant. A possible explanation could be due to some households having prepared and eaten earlier or later than the specified period used in the analysis and/or the sampling period. With the use of real time instruments, the temporal patterns of $PM_{2.5}$ and CO can be observed and subjects can be educated on when these peaks occur in order to adopt strategies to avoid exposure to these high levels of HAP generated during cooking.

In this study we present the 95^{th} percentiles of measurements from individual households. These values represent the upper extremes of exposures experienced during any given meal period.

Clearly, it is desirable for a stove to not only have low value of the typical exposure, but also a low extreme exposure. As expected from both 4hr periods, most of the highest exposures account for about 5% of the total exposures. Hence knowing when to mitigate these peak values can greatly reduce HAP exposures.

This study adds to the growing body of literature on real time HAP exposure even as the Global Alliance for Clean Cookstoves (GACC) aims to reduce HAP and its associated adverse health effects. The GACC, led by the United Nations Foundation, has the goal of 100 million households adopting clean and efficient cookstoves by the year 2020 (GACC 2011). In the advent of national cookstove programs in Peru and other countries, appropriate stove selection and evaluation of new stove models is an important step to understanding the impact of cookstove related woodsmoke exposure (McCracken and Smith 1998; Fitzgerald et al 2012). This can aid the global stove community to act together to make the necessary changes and implementation in improving billions of lives worldwide (Rehfuess et al 2006).

Limitations

Future studies can consider taking repeated measurements over extended periods of time (Smith et al 2010; Dionisio et al 2011), and together with information from time activity diaries better estimate exposures. Household observation studies also aid in the understanding of cooking patterns, behaviors and usage of improved stoves. Furthermore, it is important to be able to make population inferences based on larger sample sizes for each stove type. Control groups in this study consisted of a diverse range of stoves with varying air pollution levels. Future studies need to limit the number of control groups or ensure adequate sample sizes in each stove category.

Real time $PM_{2.5}$ data in this study were corrected with a calibration factor derived from another study (Jiang et al 2011). Jiang et al stated that the low density of wood smoke particles (p= 1.30 ± 0.02 g /cm3), a low real refractive index of 1.53 and the smaller size distribution compared to Arizona Road Dust contributed to the derived calibration factor of 0.77. Our results must be interpreted with caution in light of these. Future studies using Sidepaks should consider taking real time data and gravimetric data to account for this possible over-estimation of data. Finally, households in our study were not sampled prior to and immediately after improved stove installation and this prevented the evaluation of the OPTIMA stoves shortly after installation. Although the current measurements inform, we could not make inferences on the effectiveness of the intervention stove at reducing HAP.

CONCLUSION

We characterized personal $PM_{2.5}$ and personal CO exposures, and kitchen CO concentrations in real time among intervention and control households in San Marcos Peru. Although mean personal exposures and kitchen concentrations did not differ significantly between the two intervention arms, personal $PM_{2.5}$ exposures were well correlated with personal CO exposures and kitchen CO levels in study households. Results suggest that CO may be a useful indicator of PM in real time even during 4hr time scales and particularly during high PM exposures.

REFERENCES

Armendáriz-Arnez, C. Edwards R.D., Johnson M, Rosas I.A., Espinosa F, Masera O.R. (2010) Indoor particle size distributions in homes with open fires and improved Patsari cook stoves, *Atmospheric Environment*, 44, 2881-2886.

Balakrishnan, K., Parikh, J., Sankar, S., Padmavathi, R., Srividya, K., Venugopal, V., Prasad, S. and Laxmi Pandey, V. (2002) Daily average exposures to respirable particulate matter from combustion of biomass fuels in rural households of Southern India, *Enviromental Health Perspectives*, 110, 1069–1075.

Bølling, A.K., Pagels, J., Yttri, K.E., Barregard, L., Sallsten, G., Schwarze, P.E., Boman, C. (2009) Health effects of residential wood smoke particles: the importance of combustion conditions and physicochemical particle properties, *Particle and fibre toxicology,* 6: 29-48.

Bruce, N., McCracken, J., Albalak, R., Scheid, M., Smith, K.R., Lopez, V., West, C. (2004) Impact of improved stoves, house construction and child location on levels of indoor air pollution exposure in young Guatemalan children, *Journal of Exposure Science and Environmental Epidemiology,* 14: S26–S33.

Chengappa, C., Edwards, R., Bajpai, R., Naumoff Shields, K., Smith, K.R. (2007**)** Impact of improved cookstoves on indoor air quality in the Bundelkhand region in India, *Energy for Sustainable Development,* 11(2):33-44.

Clark ML, Reynolds SJ, Burch JB, Conway S, Bachand AM, Peel JL. (2010) Indoor air pollution, cookstove quality, and housing characteristics in two Honduran communities. *Environmental Research*, 110(1): 12-18.

Commodore, A. A., Hartinger, S.M., Lanata, C.F., Mäusezahl, D., Gil, A.I., Hall, D.B., Aguilar-Villalobos, M., Butler, C.J., Naeher, L.P. in review. "Carbon Monoxide Exposures and Kitchen Concentrations from Cookstove Related Woodsmoke in the Indoor Environment in San Marcos, Peru." *International Journal of Occupational and Environmental Health*.

Donkor, O. Chimah, C. Oluwalana, R. C. Ideh, B. Ebruke, R. A. Adegbola, and M. Ezzati. (2012). "The exposure of infants and children to carbon monoxide from biomass fuels in The Gambia: a measurement and modeling study." *Journal of Exposure Science and Environmental Epidemiology*, 22(2): 173-81.

Dutta, K., Naumoff Shields, K., Edwards, R., Smith, K.R. (2007) Impact of improved biomass cookstoves on indoor air quality near Pune, India, *Energy for Sustainable Development,* 11(2):19-32.

Environmental Protection Agency (EPA) (2005). *National Primary and Secondary Ambient Air Quality Standards*, US E.P.A.

Ezzati, M., Kammen, D.M., (2002). The Health Impacts of Exposure to Indoor Air Pollution from Solid Fuels in Developing Countries: Knowledge, Gaps, and Data Needs, *Enviromental Health Perspectives*, 110: 1057-1068.

Fitzgerald, C., M. Aguilar-Villalobos, A. R. Eppler, S. C. Dorner, S. L. Rathbun, and L. P. Naeher. (2012). "Testing the effectiveness of two improved cookstove interventions in the Santiago de Chuco Province of Peru." *Science of the Total Environment* 420(0): 54-64.

Fullerton, D. G., Semple, S., Kalambo, F., Suseno, A., Malamba, R., Henderson, G., Ayres, J. G., Gordon, S. B. (2009). Biomass fuel use and indoor air pollution in homes in Malawi. *Occupational and environmental medicine,* 66**,** 777.

Fullerton, D. G., N. Bruce, and S. B. Gordon. (2008). "Indoor air pollution from biomass fuel smoke is a major health concern in the developing world." *Transactions of the Royal Society of Tropical Medicine and Hygiene* 102(9): 843-51.

Hartinger. S., Lanata, C.F., Hattendorf, J., Gil, A.I., Verastegui, H., Ochoa, T.J., Mäusezahl, D. (2011) A community randomized controlled trial evaluating a home-based environmental intervention package of improved stoves, solar water disinfection and kitchen sinks in rural Peru: Rationale, trial design and baseline findings, *Contemporary. Clinical Trials*, doi:10.1016/j.cct.2011.06.006.

Hartinger S.M., Commodore, A.A., Hattendorf, J., Lanata, C.F., Gil, A.I., Verastegui, H., Aguilar-Villalobos, M., Mäusezahl, D., Naeher, L.P. Kitchen environment and personal exposure assessment of an improved stove in rural Peru. *Indoor Air*. (in review).

Jalava, P. I., Salonen, R.O., Nuutinen, K., Pennanen, A.S., Happoc, M.S., Tissari, J., Frey, A., Hillamo, R., Jokiniemi, J., Hirvonen, M. (2010) Effect of combustion condition on cytotoxic and inflammatory activity of residential wood combustion particles, *Atmospheric. Environment*, 44: 1691-1698.

Jiang, R.-T., V. Acevedo-Bolton, K.-C. Cheng, N. E. Klepeis, W. R. Ott, and L. M. Hildemann. (2011). "Determination of response of real-time SidePak AM510 monitor to secondhand smoke, other common indoor aerosols, and outdoor aerosol." *Journal of Environmental Monitoring* 13(6): 1695-702.

Kim K-H, Jahan SA, Kabir E. (2011). "A review of diseases associated with household air pollution due to the use of biomass fuels." *Journal of Hazardous Materials* 192(2): 425-431.

Kurmi, O. P., Semple, S., Simkhada, P., Smith, W. C. S., Ayres J. G. (2010). COPD and chronic bronchitis risk of indoor air pollution from solid fuel: a systematic review and meta-analysis. *Thorax,* 65**,** 221-228.

Kurmi, O. P., Semple, S., Steiner, M., Henderson, G. D., Ayres, J. G. (2008). Particulate Matter Exposure during Domestic Work in Nepal. *Annals of Occupational Hygiene*, 52**,** 509-517.

Li, C., S. Kang, P. Chen, Q. Zhang, J. Guo, J. Mi, P. Basang, Q. Luosang, and K. R. Smith. (2012). "Personal PM2.5 and indoor CO in nomadic tents using open and chimney biomass stoves on the Tibetan Plateau." *Atmospheric Environment* 59(0): 207-13.

Martin W.J., Glass R.I., Balbus J.M., Collins F.S. (2011) A Major Environmental Cause of Death. *Science*, 334, 180 -181.

Masera, O., R. Edwards, C. A. Arnez, V. Berrueta, M. Johnson, L. R. Bracho, H. Riojas-Rodríguez, and K. R. Smith. (2007). "Impact of Patsari improved cookstoves on indoor air quality in Michoacán, Mexico." *Energy for Sustainable Development* 11(2): 45-56.

McCracken, J.P., Smith, K.R. (1998) Emissions and efficiency of improved woodburning cookstoves in Highland Guatemala, *Environment International*, 24(7): 739-747.

Naeher, L.P., Brauer, M., Lipsett, M., Zelikoff, J.T., Simpson, C.D., Koenig, J.Q., Smith, K.R. (2007) Woodsmoke Health Effects: A Review, *Inhalation Toxicolology*, 19:67–106.

Naeher, L.P., Smith, K.R., Leaderer, B.P., Neufeld, L., Mage, D. (2001) Carbon monoxide as a tracer for assessing exposures to particulate matter in wood and gas cookstove households of highland Guatemala, *Environment Science & Technology*, 35:575-581.

Northcross, A., Chowdhury, Z., McCracken, J., Canuz, E., Smith, K.R. (2010) Estimating personal PM2.5 exposures using CO measurements in Guatemalan households cooking with wood fuel, *Journal of Environmental Monitoring*, 12: 873 – 878.

Rehfuess, E., Mehta, S., Pruss-Ustun, A. (2006) Assessing household solid fuel use: multiple implications for the Millennium Development Goals, *Environmental Health Perspectives*, 14:373-378.

Romieu, I., Riojas-Rodríguez H., Marrón-Mares A.T., Schilmann A., Perez-Padilla R., Masera O. (2009) Improved biomass stove intervention in rural Mexico: impact on the respiratory health of women. *American journal of respiratory and critical care medicine*, 180, 649-656.

Smith KR, McCracken JP, Weber MW, Hubbard A, Jenny A, Thompson LM, Balmes J, Diaz A, Arana B, Bruce N. (2011) Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial. *Lancet* 378:1717-1726.

Smith, K.R., McCracken, J.P., Thompson, L., Edwards, R., Shields K.N., Canuz, E. (2010) Personal child and mother carbon monoxide exposures and kitchen levels: Methods and

results from a randomized trial of woodfired chimney cookstoves in Guatemala (RESPIRE), *Journal of Exposure Science Environmental Epidemiology*, 20(5): 406-416.

Smith KR, Peel JL (2010). Mind the Gap. *Environmental Health Perspectives* 118:1643-1645.

Smith, K.R. (1987) Biofuels, Air Pollution, and Health. New York: Plenum. pp. 452.

The Global Alliance for Clean Cookstoves
Retrieved from http://cleancookstoves.org/ on October 23, 2011.

TSI Inc. SidePak™ AM510 Personal Aerosol Monitor Theory of Operation. Application Note ITI-085. 2006. [[accessed 1 May 2012]]. Available: http://www.tsi.com/uploadedFiles/_Site_Root/Products/Literature/Application_Notes/ITI-085.pdf.

Von Schirndig, Y., Bruce, N., Smith, K., Ballard-Treemer, G., Ezzati, M. and Lvovsky, K. (2002) Addressing the Impact of Household Energy and Indoor Air Pollution on the Health of the Poor. Implications for Policy Action and Intervention Measures. Geneva, WHO.

World Health Organization (WHO) (2008) *Air quality and health*; Fact Sheet 313.

World Health Organization (WHO) (2011) *Indoor Air Pollution and Health*; Fact Sheet 292.

Table 7.1. Study household characteristics and demographic information by stove type

Arm of Intervention	Stove Type	Type of stove repair needed	Age of subject (years)	Highest level of education completed by subject	Total # of people in household	Kitchen volume (m^3)	Wood type used in last 48 hrs
Intervention	Optima FL-I[a]	None	40	None	5	24.4	-
	Optima FL-I	None	26	Elementary	4	25.1	Eucalipto
	Optima FL-I	None	30	Preschool	7	14.6	Hualango
	Optima FL-I	None	28	Elementary	4	33.7	Other
	Optima FL-I	None	23	Preschool	4	21.3	Hualango
	Optima FL-I	None	30	Preschool	4	25.7	Eucalipto
	Optima FL-II	Minor repair	24	High School	6	33.6	Hualango
	Optima FL-II	Minor repair	46	Preschool	7	45.3	Eucalipto
	Optima FL-II	Major repair	24	High School	3	36.2	Eucalipto
	Built by NGO[b]	N/A	38	Elementary	7	49.7	Eucalipto
	Built by NGO	N/A	32	Preschool	6	21.0	Chamiza
	Built by NGO	N/A	-	-	-	39.5	Pauquillo
	Built by NGO	N/A	-	-	-	-	-
Control	Improved by household	N/A	24	Elementary	5	105.3	Chamana
	Traditional	N/A	24	Preschool	5	28.3	Hualango
	Traditional	N/A	36	Elementary	6	55.5	Eucalipto
	Traditional	N/A	38	High School	4	56.0	Eucalipto
	Traditional	N/A	26	High School	6	19.2	Eucalipto
	Traditional	N/A	38	Elementary	3	23.4	Hualango

[a]Functionality level (FL) I refers to a stove in good conditions, level II refers to a stove in need of minor repairs (re-plastering) or major repairs (e.g. chimney valve replacement).

[b]NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO & ADIAR are NGOs that work in nearby communities.

- denotes unavailable data

Table 7.2. Time weighted average (TWA) real time $PM_{2.5}$ and CO measurements from intervention and control households

Arm of Intervention	Stove Type	Sampling Duration (hrs)	Adjusted[c] Personal $PM_{2.5}$ ($\mu g/m^3$)			Personal CO (ppm)			Kitchen CO (ppm)		
			Mean (95% CI)	Median	Range	Mean (95% CI)	Median	Range	Mean (95% CI)	Median	Range
Intervention	Optima FL-I[a]	6.7	315.3 (250.8, 379.8)	10.8	0 - 14840	2.5 (2.2, 2.8)	0.5	0 - 56.0	10.1 (9.1, 11.1)	5.0	0 - 179.0
	Optima FL-I	13.1	63.1 (52.3, 115.4)	5.4	0 - 4198.8	0.8 (0.7, 1.6)	0	0 - 15.0	1.9 (1.7, 3.6)	0	0 - 50.0
	Optima FL-I	11.0	175.8 (147.6, 323.4)	12.3	1.5 - 7188.7	1.2 (1.0, 2.2)	0	0 - 19.0	4.5 (4.1, 8.6)	1.0	0 - 126.0
	Optima FL-I	10.3	7.6 (7.2, 8.0)	6.2	3.1 - 132.4	0.0	0	0	0	0	0 - 2.0
	Optima FL-I	11.1	19.7 (16.6, 22.8)	5.4	0 - 1096.5	0.3 (0.2, 0.3)	0	0 - 10.0	0.6 (0.5, 0.7)	0	0 - 12.0
	Optima FL-I	2.8	1911.4 (1624.3, 2198.5)	653.7	3.1 - 6545.0	0.8 (0.5, 1.1)	0	0 - 16	-	-	-
	Optima FL-II	8.1	15.5 (11.4, 19.7)	1.5	0 - 793.1	0.8 (0.7, 0.9)	0	0 - 22.0	2.9 (2.6, 3.2)	2.0	0 - 63.0
	Optima FL-II	11.3	303.2 (254.5, 557.7)	9.2	3.1 - 9389.4	1.7 (1.5, 3.2)	0	0 - 55.0	3.7 (3.4, 7.2)	1.0	0 - 60.0
	Optima FL-II	9.0	3562.7 (3263.1, 3862.2)	1.6	0 - 15400	31.9 (29.2, 34.6)	13.0	0 - 257.0	74.9 (69.2, 80.7)	40.0	0 - 812.0
	Built by NGO[b]	10.3	75.3 (62.5, 88.2)	9.2	0 - 2067.5	0.4 (0.3, 0.4)	0	0 - 6.0	-	-	-
	Built by NGO	12.4	526.1 (458.9, 593.2)	27.0	0 - 11665.5	5.0 (4.7, 5.4)	3.0	0 - 55.0	20.9 (19.0, 22.8)	10.0	0 - 383.0
	Built by NGO	9.4	83.7 (62.9, 104.5)	0.8	0 - 4118.0	2.0 (1.7, 2.2)	0	0 - 35.0	4.1 (3.8, 4.2)	3.0	0 - 60.0
	Built by NGO	9.5	9.7 (8.4, 11.0)	2.7	0 - 360.4	2.9 (2.5, 3.3)	0	0 - 61.0	5.3 (4.7, 5.9)	0	0 - 104.0
Control	Improved by household	11.2	92.2 (74.5, 109.8)	13.1	1.5 - 8130.0	0.6 (0.5, 0.6)	0	0 - 10.0	0.5 (0.4, 0.5)	0	0 - 16.0
	Traditional	9.1	324.9 (252.6, 397.2)	30.0	5.4 - 12657.3	1.2 (1.0, 1.4)	0	0 - 27.0	-	-	-
	Traditional	11.1	2365.9 (2165.6, 2566.2)	252.2	0 - 15400	11.3 (10.6, 12.1)	5.0	0 - 67.0	29.8 (28.5, 31.1)	24.0	0 - 148.0
	Traditional	9.2	15.4 (14.3, 16.6)	14.6	0 - 515.9	0.0	0	0 - 3.0	0	0	0
	Traditional	10.9	415.3 (363.5, 467.1)	98.2	2.3 - 11133.4	4.9 (4.2, 5.6)	2.0	0 - 210.0	4.9 (4.2, 5.6)	1.0	0 - 267.0
	Traditional	10.5	60.0 (41.8, 78.1)	7.7	0 - 5664.9	0.2 (0.2, 0.3)	0	0 - 15.0	2.9 (2.6, 3.1)	2.0	0 - 40.0

[a]Functionality level (FL) I refers to a stove in good conditions, level II refers to a stove in need of minor repairs (re-plastering) or major repairs (e.g. chimney valve replacement).

[b]NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO & ADIAR are NGOs that work in nearby communities.

[c]Sidepak $PM_{2.5}$ measurements adjusted with a correction factor of 0.77 according to measurements by Jiang et al (2011)

- denotes unavailable data

Table 7.3. Intervention and control household $PM_{2.5}$ and CO measurements during lunch and dinner

Arm of Intervention	Stove Type	Lunch (9am - 1pm)					
		Mean (95% Confidence Interval)			Median (95% Confidence Interval)		
		Personal $PM_{2.5}$ ($\mu g/m^3$)	Personal CO (ppm)	Kitchen CO (ppm)	Personal $PM_{2.5}$ ($\mu g/m^3$)	Personal CO (ppm)	Kitchen CO (ppm)
Intervention	Optima FL-I[a]	739.1 (549.6, 928.6)	2.3 (1.7, 2.9)	14.4 (12.0, 16.8)	279.5 (224.8, 321.9)	1.0 (1.0, 2.0)	10.0 (9.0, 11.0)
	Optima FL-I	-	-	-	-	-	-
	Optima FL-I	447.0 (376.4, 823.4)	2.8 (2.5, 5.3)	8.4 (7.5, 15.9)	123.2 (101.6, 160.2)	2.0 (2.0, 3.0)	6.0 (5.0, 7.0)
	Optima FL-I	-	-	-	-	-	-
	Optima FL-I	49.9 (39.5, 60.3)	0.2 (0.1, 0.3)	1.0 (0.8, 1.2)	14.6 (11.6, 22.3)	0	0
	Optima FL-I	1911.4 (1624.3, 2198.5)	0.8 (0.5, 1.1)	-	653.7 (632.2, 703.1)	0	-
	Optima FL-II	14.2 (6.8, 21.5)	1.8 (1.5, 2.1)	6.9 (6.1, 7.7)	6.2 (6.2, 6.9)	1.0 (1.0, 1.0)	5.0 (5.0, 5.0)
	Optima FL-II	-	-	-	-	-	-
	Optima FL-II	3823.6 (3349.1, 4298.1)	22.9 (20.1, 25.6)	115.8 (103.7, 128.0)	1579.3 (1321.3, 1737.1)	12.0 (12.0, 13.0)	85.5 (71.0, 95.0)
Control	Built by NGO[b]	-	-	-	-	-	-
	Built by NGO	-	-	-	-	-	-
	Built by NGO	-	-	-	-	-	-
	Built by NGO	-	-	-	-	-	-
	Improved by household	63.9 (40.7, 87.0)	0.6 (0.4, 0.8)	1.4 (1.0, 1.7)	10.0 (9.2, 13.9)	0	1.0 (0, 1.0)
	Traditional	669.5 (485.6, 853.5)	3.7 (3.0, 4.3)	-	134.8 (107.0, 206.4)	3.0 (2.0, 3.0)	-
	Traditional	1088.0 (943.7, 1232.3)	6.6 (5.9, 7.3)	27.9 (25.9, 30.0)	576.7 (459.7, 683.8)	5.0 (5.0, 6.0)	22.0 (20.0, 24.0)
	Traditional	-	-	-	-	-	-
	Traditional	456.1 (395.0, 517.2)	4.7 (3.3, 6.0)	1.8 (1.5, 2.2)	149.8 (116.3, 195.6)	2.0 (2.0, 3.0)	0
	Traditional	178.9 (97.3, 260.5)	0.4 (0.2, 0.6)	3.9 (3.4, 4.4)	0.8 (0, 0.8)	0	3.0 (3.0, 3.0)

Arm of Intervention	Stove Type	Dinner (3pm - 7pm)					
		Mean (95% Confidence Interval)			Median (95% Confidence Interval)		
		Personal $PM_{2.5}$ ($\mu g/m^3$)	Personal CO (ppm)	Kitchen CO (ppm)	Personal $PM_{2.5}$ ($\mu g/m^3$)	Personal CO (ppm)	Kitchen CO (ppm)
Intervention	Optima FL-I[a]	-	-	-	-	-	-
	Optima FL-I	47.0 (34.7, 81.8)	1.2 (0.8, 2.0)	0	7.7 (6.9, 8.5)	0	0
	Optima FL-I	7.9 (6.8, 14.7)	0	1.7 (1.2, 2.9)	3.9 (3.1, 3.9)	0	0
	Optima FL-I	8.7 (7.9, 9.5)	0	0	5.4 (5.4, 6.2)	0	0
	Optima FL-I	7.4 (5.7, 9.0)	0.1 (0, 0.1)	0.1 (0, 0.1)	1.5 (1.5, 1.5)	0	0
	Optima FL-I	-	-	-	-	-	-
	Optima FL-II	24.3 (16.6, 32.0)	0.4 (0.3, 0.5)	0.6 (0.4, 0.8)	0.8 (0.8, 0.8)	0	0
	Optima FL-II	309.4 (207.5, 516.9)	1.3 (0.8, 2.1)	2.1 (1.4, 3.5)	6.2 (6.2, 6.9)	0	0
	Optima FL-II	4937.3 (4399.0, 5475.6)	50.9 (45.1, 56.7)	63.1 (57.1, 69.0)	2731.6 (2371.6, 3379.5)	31.5 (26.0, 37.0)	46.0 (41.0, 51.0)
Control	Built by NGO[b]	198.8 (164.6, 232.9)	0.7 (0.5, 0.8)	-	19.3 (12.3, 37.0)	0	-
	Built by NGO	1017.2 (837.2, 1197.2)	6.5 (5.6, 7.3)	37.4 (32.3, 42.6)	148.6 (101.6, 177.1)	3.0 (3.0, 3.0)	13.0 (12.0, 15.0)
	Built by NGO	154.4 (104.1, 204.6)	2.1 (1.8, 2.4)	4.5 (3.7, 5.3)	17.3 (14.6, 26.2)	0	2.0 (2.0 - 3.0)
	Built by NGO	5.2 (3.6, 6.8)	5.2 (4.4, 6.0)	7.8 (6.7, 8.9)	0	1.0 (0, 1.0)	0
	Improved by household	191.8 (158.3, 225.2)	1.4 (1.2, 1.5)	0.8 (0.7, 0.9)	50.4 (42.4, 65.5)	1.0 (1.0, 1.0)	0
	Traditional	57.0 (47.6, 66.4)	0.4 (0.3, 0.5)	-	29.6 (27.0, 34.7)	0	-
	Traditional	5098.0 (4665.3, 5530.6)	20.8(19.2, 22.3)	36.7 (34.4, 38.9)	4267.7 (3545.9, 5413.1)	21.0 (18.0, 24.0)	33.5 (31.0, 35.0)
	Traditional	15.8 (14.9, 16.7)	0	0	14.6 (13.9, 14.9)	0	0
	Traditional	581.5 (460.3, 702.7)	7.2 (5.7, 8.6)	9.9 (8.2, 11.6)	166.3 (122.4, 202.5)	3.0 (3.0, 4.0)	6.0 (5.0, 7.0)
	Traditional	11.1 (9.8, 12.5)	0	2.6 (2.4, 2.9)	8.5 (8.5, 8.5)	0	2.0 (1.0, 2.0)

[a]Functionality level (FL) I refers to a stove in good conditions, level II refers to a stove in need of minor repairs (re-plastering) or major repairs (e.g. chimney valve replacement).

[b]NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO & ADIAR are NGOs that work in nearby communities.

[c]Sidepak $PM_{2.5}$ measurements adjusted with a correction factor of 0.77 according to measurements by Jiang et al (2011)

- denotes unavailable data

Table 7.4. 95th percentile of real time measurements during lunch and dinner periods. In this table the highest 5% of exposure measurements recorded. Hence each value represents PM or CO levels below which 95% of the total measurements during that meal period may be found.

Arm of Intervention	Stove Type	Lunch Period (9am - 1pm)			Dinner Period (3pm - 7pm)		
		Personal PM (95%CI)	Personal CO (95%CI)	Kitchen CO (95%CI)	Personal PM (95%CI)	Personal CO (95%CI)	Kitchen CO (95%CI)
Intervention	Optima FL-I[a]	2936.8 (2561.0, 4234.2)	7.0 (5.0, 8.0)	54.0 (34.0, 77.0)	-	-	-
	Optima FL-I	-	-	-	295.7 (216.4, 388.9)	10.0 (9.0, 11.0)	0.0
	Optima FL-I	1938.1 (1757.1, 2715.0)	8.0 (7.0 , 10.0)	23.0 (21.0, 29.0)	36.2 (28.5, 43.1)	0.0	25.0 (18.0, 82.0)
	Optima FL-I	-	-	-	23.1 (18.5, 31.6)	0.0	0.0
	Optima FL-I	183.3 (151.7, 267.2)	2.0 (0. 3.0)	5.0 (4.0, 6.0)	34.7 (27.0, 47.7)	0.0	0.0
	Optima FL-I	6047.6 (5811.2, 6357.9)	6.0 (3.0, 10.0)	-	-	-	-
	Optima FL-II	25.4 (13.9, 61.6)	5.0 (5.0 - 9.0)	17.0 (13.0, 27.0)	261.8 (137.8, 336.5)	3.0 (1.0, 3.0)	4.0 (4.0, 5.0)
	Optima FL-II	-	-	-	2502.5 (1560.8, 3883.1)	9.0 (7.0, 15.0)	15.0 (10.0, 21.0)
	Optima FL-II	15400 (14871.8, 15400)	84.0 (76.0, 106.0)	375.0 (284.0, 450.0)	15400.0 (15400.0, 15400.0)	205.0 (197.0, 218.0)	212.0 (193.0, 229.0)
	Built by NGO[b]	-	-	-	973.3 (820.1, 1361.4)	4.0 (4.0, 4.0)	-
	Built by NGO	-	-	-	5632.6 (4737.0, 7303.5)	28.0 (25.0, 35.0)	163.0 (142.0, 211.0)
	Built by NGO	-	-	-	751.5 (455.1, 1967.4)	7.0 (6.0, 12.0)	19.0 (16.0, 26.0)
	Built by NGO	-	-	-	15.4 (10.8, 23.1)	26.0 (23.0, 32.0)	38.0 (34.0, 40.0)
	Improved by household	323.4 (179.4, 729.2)	4.0 (2.0, 6.0)	4.5 (4.0, 8.0)	861.6 (735.4, 1285.9)	4.0 (4.0, 5.0)	4.0 (3.0, 5.0)
Control	Traditional	3927.8 (2999.9, 5811.2)	16.0 (10.0, 21.0)	-	172.4 (123.2, 271.8)	3.0 (2.0, 5.0)	-
	Traditional	4071.8 (3338.0, 5326.1)	19.0 (14.0, 27.0)	69.0 (62.0, 73.0)	13193.2 (12738.1, 14529.9)	49.0 (47.0, 52.0)	82.0 (73.0, 93.0)
	Traditional	-	-	-	36.2 (33.1, 39.3)	0.0	0.0
	Traditional	1864.2 (194.8, 2153.0)	14.0 (11.0, 21.0)	7.0 (6.0, 11.0)	2816.7 (1647.0, 4338.2)	26.0 (19.0, 42.0)	32.0 (25.0, 40.0)
	Traditional	975.6 (599.1, 2299.2)	3.0 (1.0, 5.0)	12.0 (10.0, 15.0)	35.4 (27.0, 44.7)	0.0	9.0 (8.0, 10.0)

[a]Functionality level (FL) I refers to a stove in good conditions, level II refers to a stove in need of minor repairs (re-plastering) or major repairs (e.g. chimney valve replacement).

[b]NGO: three main NGOs had improved stoves; JUNTOS-National cash transfer program. Part of the requirements is that families must build an improved stove with a chimney; SEMBRANDO & ADIAR are NGOs that work in nearby communities.
[c]Sidepak $PM_{2.5}$ measurements adjusted with a correction factor of 0.77 according to measurements by Jiang et al (2011)
- denotes unavailable data

Figure 7.1.
An OPTIMA stove user wearing vest which encases a Sidepak, a Pac III and a personal sampling pump (A). Stoves built by an NGO, self-improved by household and a traditional three stone open fire are shown in B, C and D respectively.

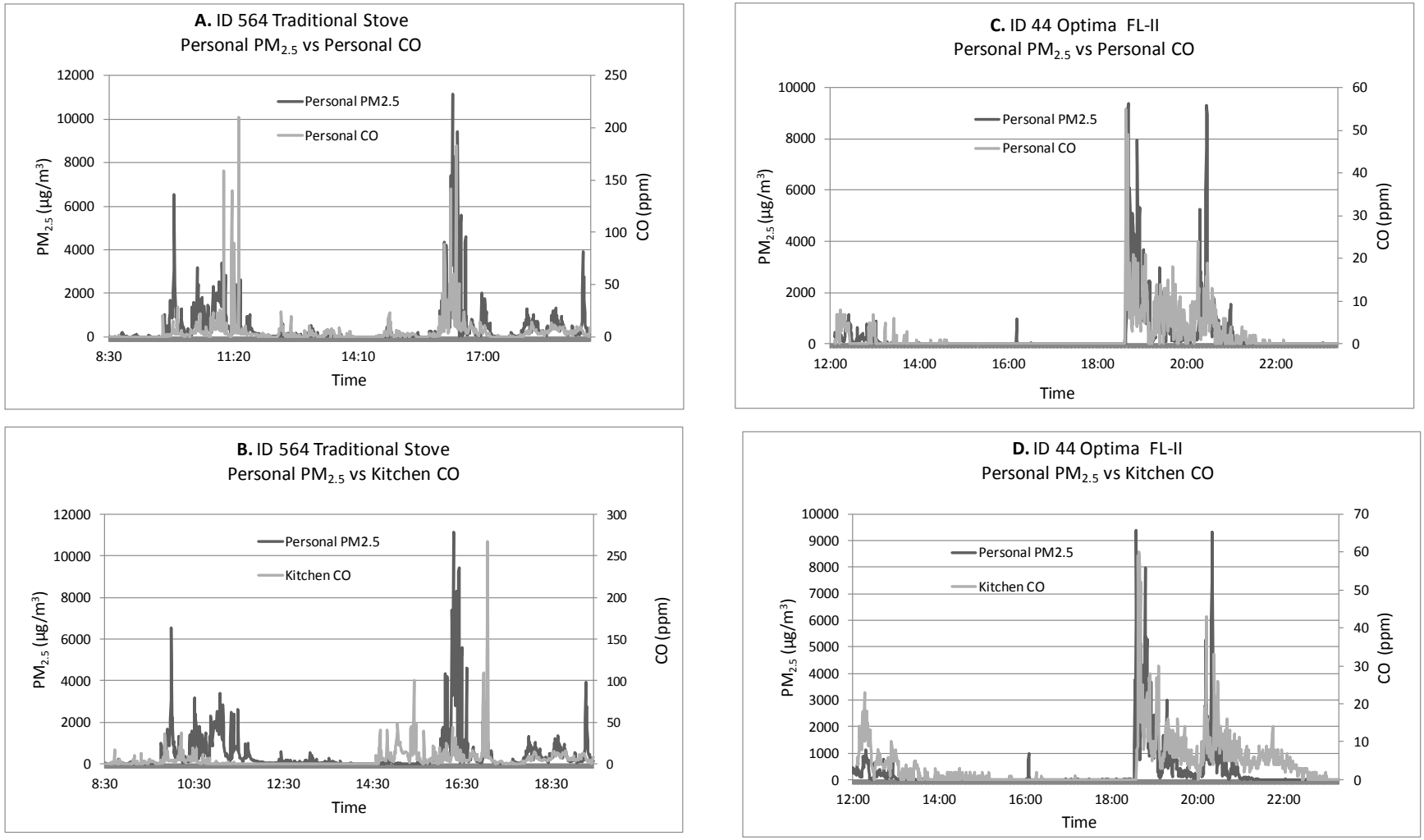


Figure 7.2.
Temporal profiles for personal $PM_{2.5}$ vs. personal CO and personal $PM_{2.5}$ vs. kitchen CO for a traditional stove user (A and B) and an OPTIMA FL-II stove user (C and D).

CHAPTER 8

SUMMARY AND CONCLUSION

Summary on radionuclides in the environment

Forest fires have the potential to release certain constituents, including radioactive material into the atmosphere. The release of these radionuclides raises health concerns depending on the nature of the radionuclides (Peterson et al. 2007). The main concern for exposures that could occur from this airborne radioactivity is for firefighting personnel, nearby communities and the ecology. Studies on radionuclide exposure assessment need to be conducted to fill in the literature and scientific gaps that exist with the question of the fate and bioavailability of radionuclides during forest fires.

Radioactivity in smoke particulates from prescribed burns

In this study, we compare airborne radionuclide concentrations at the Savannah River Site (SRS) and a sample of forests in the Southeastern United States during prescribed burns. The spatial trends of airborne radionuclide concentrations from prescribed burn areas at SRS were also characterized. Results indicate that prescribed burns at SRS lead to measurable levels of ^{40}K, gross beta, plutonium, thorium and uranium isotopes compared to offsite burns and nonburn days, yet there were no apparent spatial trends in relation to SRS facilities.

Although radiation exposure from measureable levels of airborne radionuclides during SRS prescribed burns appears insignificant, there are potential health impacts associated with exposure to forest biomass combustion products. Future studies need to further characterize radionuclide emissions, together with exposures during fires at facilities where nuclear materials exist, to understand and quantify any inhalation hazards to nearby workers and the community.

Summary on household air pollution

Nearly half of the world's population burns biomass, mostly as fuel for cooking (Martin et al. 2011; Naeher et al. 2007; Smith 1987) resulting in household air pollution (HAP). Women and young children bear the brunt of high HAP exposures due to long hours spent in close proximity to cooking fires (Ezzati and Kammen 2002). Household stoves typically used for cooking and heating in the developing world do not burn fuel cleanly leading to incomplete combustion in the domestic environment (Perez-Padilla et al. 2010; Smith 1987). A vast majority of HAP constituents are health-damaging in nature (Bølling et al. 2009; Jalava et al. 2010; Naeher et al. 2007).

HAP is responsible for almost 50% of the burden of diseases resulting from poor household environments (Rehfuess et al. 2006). The use of biomass fuel in the household environment is linked to respiratory and nonrespiratory illnesses and is considered a major public health threat globally (Kim et al. 2011a). Biomass smoke exposure is associated with the risk of acute lower respiratory infections (ALRI) (WHO 2011). ALRI is the chief cause of death in children in developing countries (Martin et al. 2011). In particular, the most fatal cases are in the form of pneumonia among children less than five years old (Bryce et al. 2005). There is the need studies to better characterize HAP and associated health effects.

In order to fill in some of the identified gaps regarding HAP exposure and associated health effects, we conducted a cross sectional study within the framework of a community randomized control trial. The primary objectives of the study were to characterize HAP exposures and quantify urinary levels of biomarkers of oxidative stress and lipid peroxidation among control and intervention stove users.

Carbon monoxide exposures and kitchen concentrations

The objective of this study was to compare CO exposures and concentrations among study promoted intervention stove users and control stove users in San Marcos Province, Cajamarca region, Peru. Inexpensive passive CO diffusion tubes were deployed over a 48-hour sampling period to measure kitchen CO concentrations and personal mother and child CO exposures in 197 control and 182 intervention households. Geometric means (95%CI) for child, mother and kitchen measurements were 1.1 (0.9, 1.2) ppm, 1.4 (1.3, 1.6) ppm and 7.3 (6.4, 8.3) ppm in control households, and 1.0 (0.9, 1.1) ppm, 1.4 (1.3, 1.6) ppm and 7.3 (6.4, 8.2) ppm among intervention households respectively.

Personal CO exposures and kitchen levels measured with passive diffusion tubes did not differ significantly between intervention and control households. Personal mother CO exposures were correlated with children's exposures. These results point to the fact that where data are unavailable, mothers' exposures can be used to predict children's exposures especially in high pollution settings. Results suggest that proper and exclusive chimney stove use, maintenance of stoves as well as changes to fuel types may be necessary in reducing CO and more generally, HAP exposures.

Concentrations of urinary 8-OHdG and 8-isoprostane between intervention and control stove users

Aside experimental studies where the effect of woodsmoke on oxidative stress is well studied, oxidative stress as an adverse health end point has not been widely studied in humans and rarely in women in the developing world. Given the continuous exposure experienced by these women over a lifetime, studies are needed to better understand the effects of woodsmoke at important stages in their lives, namely with the introduction of improved stoves and during child bearing years. As such, we aimed to investigate whether woodsmoke exposure induces oxidative stress by examining the relationship between woodsmoke exposure and biomarkers of DNA and lipid oxidation. The first morning voids after 48hr HAP exposure assessment were taken from 45 control stove users and 39 intervention stove users and analyzed for 8-OHdG and 8-isoprostane.

Creatinine corrected urinary oxidative stress biomarkers ranged from 11.2 to 2270.0 ug/g creatinine (median: 132.6 ug/g creatinine) for 8-OHdG and from 0.1 to 4.5 ug/g creatinine (median: 0.8 ug/g creatinine) for 8-isoprostane among all study subjects (n=84). There were no statistically significant differences in urinary oxidative stress biomarkers between the two arms of the intervention. Mean (95% CI) urinary 8-OHdG and 8-isoprostane levels among intervention stove users were 132.9 (97.7, 180.8) ug/g creatinine and 0.8 (0.6, 1.1) ug/g creatinine respectively. Likewise, among control stove users, these levels were 139.3 (108.7, 178.4) ug/g creatinine and 0.7 (0.6, 0.9) ug/g creatinine respectively.

After controlling for the effects of traffic in the community and eating food exposed to fire, cooking time was positively correlated with urinary 8-OHdG (correlation coefficient (r)=0.29, p=0.01, n=80). Subjects' real time personal CO exposures were negatively correlated with 8-OHdG, particularly the maximum 30 second CO exposure during the sampling period

(r=-0.32, p=0.001, n=73). After controlling for the effect of distance of road to subjects' homes, 1-hyroxyphenanthrene and 2-hyroxyphenanthrene were both positively correlated with 8-isoprostane (r for both hydroxy-PAH biomarkers=0.21, p=0.05 and 0.06 respectively, n=84). 48hr time integrated personal $PM_{2.5}$ was negatively, but marginally correlated with urinary 8-isoprostane after controlling for the effect of distance of homes to the road (r=-0.21, p=0.09, n=69). This cross sectional study conducted within the framework of a community based randomized control trial shows relatively high levels of urinary 8-OHdG compared to data in the literature for 8-OHdG excretion. Results suggest that there is a sustained systemic oxidative stress status among these women.

Real time exposures to particulate matter and carbon monoxide

In this study we aimed to use $PM_{2.5}$ and CO measurements from households in San Marcos to characterize exposure to cookstove generated woodsmoke in real time among control and intervention households. Real time $PM_{2.5}$ and personal and kitchen CO measurements were taken in control (n=10) and intervention households (n=9). Measurements were categorized into lunch (9am – 1pm) and dinner (3pm – 7pm) periods, where applicable, to adjust for a wide range of sampling periods (2.8–13.1hrs). There were no statistically significant differences in exposure measurements between control and intervention stove users. Mean (95% CI) personal $PM_{2.5}$ exposures were 557.9 (0, 1404.7) $\mu g/m^3$ and 396.8 (0, 840.1) $\mu g/m^3$ in intervention and control homes respectively. Likewise personal CO exposures were 4.9 (0, 12.5) ppm and 2.8 (1.0, 5.0) ppm, and kitchen CO concentrations were 12.3 (0, 30.0) ppm and 10.8 (2.0, 15.2) ppm in intervention and control homes respectively.

During the 4hr time periods, mean personal $PM_{2.5}$ exposures were correlated with personal CO exposures during lunch (r=0.67 p=0.024 n=11) and dinner (r=0.72 p=0.0011 n=17) in all study households. Personal $PM_{2.5}$ exposures and kitchen CO concentrations were also correlated during lunch (r=0.76 p=0.018 n=9) and dinner (r=0.60 p=0.018 n=15). In all study households, mean personal $PM_{2.5}$ exposures were marginally correlated with personal CO exposures (r=0.41 p=0.08 n=19) and significantly correlated with kitchen CO levels (r=0.56 p=0.03 n=16). Results suggest that CO may be a useful indicator of PM during 4hr time scales measured in real time and particularly during high PM exposures. With the use of real time instruments, the temporal patterns of $PM_{2.5}$ and CO can be observed and subjects can be educated on when these peaks occur in order to adopt strategies to avoid exposure to these high levels of HAP generated during cooking.

Assessment of Exposure to PAHs among study subjects

In this study we compare the levels of urinary hydroxy-PAH metabolites among control and intervention stove users. Preliminary fresh weight results showed significantly lower levels of urinary 2-napthol (35%) and 3-hydroxyfluorene (23%) in persons using improved stoves compared to open-fire stove users. Urinary OH-PAH levels are comparable or higher than the 95^{th} percentile of the levels in the general US population. The effects of the use of these chimney stoves over long periods of time remain unclear. Even with the improved stoves, exposure to PAHs in this population remains high compared to the referenced US population.

CO and $PM_{2.5}$ exposures and kitchen concentrations

The objective of this study was to compare CO and $PM_{2.5}$ exposures and kitchen concentrations in control and intervention households in San Marcos. We determined 48hr indoor air concentration levels of CO and $PM_{2.5}$ in 93 kitchen environments and personal

exposure, after OPTIMA stoves had been installed for an average of seven months. Household kitchen concentration of $PM_{2.5}$ and CO did not differ significantly between OPTIMA stoves and control stoves. However a post-hoc stratification of improved stoves by level of performance revealed mean $PM_{2.5}$ and CO levels of the fully functional (or FL-I) stoves were 28% lower (n=20, $PM_{2.5,}$ 136μg/m^3 95%CI 54-217) and 45% lower (n=25, CO, 3.2ppm, 95%CI 1.5-4.9) in the kitchen environment compared to the control stoves (n=34, $PM_{2.5,}$ 189μg/m^3, 95%CI 116–261; n=44, CO, 5.8ppm, 95%CI 3.3-8-2ppm). Likewise, personal exposures for OPTIMA-improved stoves were 43% and 167% lower for $PM_{2.5}$ (n=23) and CO (n=25). At the time of sampling, 66% (29/43) of the Optima stoves were properly maintained. Stove maintenance and functionality level are factors worthy of consideration for future evaluations of stove interventions.

Conclusion

Carbon monoxide and $PM_{2.5}$ measurements in this study did not demonstrate statistically significant differences across the various stove types in both arms of the intervention. The lack of differences between control and intervention households seems contrary to results reported in other chimney stove intervention studies (Chengappa et al. 2007; Cynthia et al. 2008; Dutta et al. 2007; Fitzgerald et al. 2012a; Masera et al. 2007; Smith et al. 2010). While some of the intervention studies mentioned above assessed HAP exposures before and soon after stove installation, other stoves were monitored frequently and stoves were routinely fixed. In this cross sectional study, we present data on HAP measurements of chimney stoves referred to as OPTIMA stoves that had been in use, on average, for several months. Some of these stoves had not been maintained, and may have been improperly used. Our results have potential

implications for intervention studies in the developing world aiming to answer the question of stove performance months after installation and use.

A number of reasons may have led to high HAP levels in intervention households in the San Marcos study. Adequate stove design, manner of stove use (i.e. whether it is used continuously, properly and exclusively), as well as maintenance over time are key factors in HAP mitigation. Design and construction of efficient cookstoves is also key to reducing and sustaining low exposure levels (Smith et al. 2011). It has been documented that intervention stoves can improve health when properly used (Diaz et al. 2008; Romieu et al. 2009; Smith et al. 2011). It is important then to determine the stove's performance at the time of installation (Fitzgerald et al. 2012a) and also months and years after installation, as the intervention stove may possibly introduce greater HAP if improperly maintained and used. Also the importance of functionality levels within stove type is important in HAP exposure assessment. Clark et al. (2010) suggest the utility of stove functionality levels to be more representative of HAP exposures and indoor levels. They note the importance of assessing the condition of the stoves rather than a mere comparison between traditional and improve stove type (Clark et al 2010). Our results indicate that after an average of seven months of use, OPTIMA stoves (whether they were in need of repairs or not) did result in significantly lower personal CO exposures and kitchen levels when compared to control stoves. Hence stove maintenance and functionality are both essential in understanding HAP exposures (Albalak et al. 2001).

Aside experimental studies where the effect of woodsmoke on oxidative stress is well studied, oxidative stress as an adverse health end point has not been widely studied in humans and rarely in women in the developing world. The study on oxidative stress presented herein is the first to investigate the effect of woodsmoke exposure on systemic oxidative stress among

women exposed to high levels of cookstove related woodsmoke in rural Peru. Given the continuous exposure experienced by these women over a lifetime, studies are needed to better understand the effects of woodsmoke at important stages in their lives, namely with the introduction of improved stoves and during child bearing years. Adequate characterization of exposure to residential biomass combustion is crucial in vulnerable populations such as in rural communities in developing countries where biomass fuels are used on a daily basis for cooking and heating (Balakrishnan et al. 2011; Ezzati and Kammen 2002; INEI 2007).

As the Global Alliance for Clean Cookstoves (GACC) aims to reduce HAP and the adverse health effects associated with it, these studies add to the limited body of literature on the HAP exposure characterization and associated health effects. The GACC, led by the United Nations Foundation, has the goal of 100 million households adopting clean and efficient cookstoves by the year 2020 (GACC 2011). Certain issues must be addressed before these clean and efficient stoves are adopted globally. It is essential to determine the amount of HAP reduction necessary to improve health (Pope III et al. 2011; Smith et al. 2011; Smith and Peel 2010), as well as develop new and rigorous means to evaluate the health benefits of worldwide stove implementation programs (Martin et al. 2011). In the advent of national cookstove programs in Peru and other countries, evaluation of new stove models is also an important step to understanding the impact of cookstove related woodsmoke exposure (Fitzgerald et al. 2012a).

Future Research

Radionuclides released from SRS prescribed burn areas appear to be a small percentage of the total radiation dose received from numerous other radiation sources (McBride et al. 1978; Volkerding 2004). Although radiation exposure from measureable levels of airborne radionuclides during SRS prescribed burns appears insignificant, there are potential health

impacts associated with exposure to forest biomass combustion products (Naeher et al. 2007). There is currently a proposal by the Naeher lab to conduct baseline measurements of the inhalation personal exposures of wildland firefighters to radionuclides during prescribed burns at SRS. Unlike in the previous study, personal inhalation exposures of the firefighters to radioactivity will be measured. This will enable the estimation of actual radiation dose received by firefighting personnel.

There is a dearth of knowledge on HAP exposures in the developing world human woodsmoke exposures and the physiological response due to these exposures. In the advent of national cookstove programs in Peru and other countries, it is essential that stove programs be thoroughly evaluated and solutions with lasting benefits be invested in and developed.

There is the need to determine which interventions, whether stove or fuel type, are capable of reducing HAP in a given geographic location. Simultaneously, the association of HAP exposure and other adverse health outcomes needs investigation, as these outcomes may require lower or perhaps even higher reductions in HAP exposures. Additionally, stove designs and models need to be improved upon. This global solution for cooking needs to be in partnership with engineers, researchers and of course the users themselves. Current HAP exposure assessment tools such as biomarkers, personal and stationary monitors as well as questionnaires need to be improved upon to better quantify HAP. Finally, since there are current stove programs underway worldwide, there needs to be evaluations to understand the health benefits or the lack thereof before global stove dissemination occurs for any particular stove type.

REFERENCES

Adetona, O., D. B. Hall, and L. P. Naeher. 2011. "Lung function changes in wildland firefighters working at prescribed burns." *Inhalation Toxicology* 23(13): 835-41.

Aggarwal, R. K. and S. S. Chandel. 2004. "Review of Improved Cookstoves Programme in Western Himalayan State of India." *Biomass and Bioenergy* 27(2): 131-44.

Alam, S., S. J. Chowdhury, A. Begum, and M. Rahman. 2006. "Effect of improved earthen stoves: improving health for rural communities in Bangladesh." *Energy for Sustainable Development* 10(3): 46-53.

Albalak, R., N. Bruce, J. P. McCracken, K. R. Smith, and T. De Gallardo. 2001. "Indoor respirable particulate matter concentrations from an open fire, improved cookstove, and LPG/open fire combination in a rural Guatemalan community." *Environmental science & technology* 35(13): 2650-55.

Allen, R. W., C. Carlsten, B. Karlen, S. Leckie, S. van Eeden, S. Vedal, I. Wong, and M. Brauer. 2011. "An air filter intervention study of endothelial function among healthy adults in a woodsmoke-impacted community." *American journal of respiratory and critical care medicine* 183(9): 1222-30.

Andreae, M. O. and P. Merlet. 2001. "Emission of trace gases and aerosols from biomass burning." *Global biogeochemical cycles* 15(4): 955-66.

Armendáriz-Arnez, C., R. D. Edwards, M. Johnson, I. A. Rosas, F. Espinosa, and O. R. Masera. 2010. "Indoor particle size distributions in homes with open fires and improved Patsari cook stoves." *Atmospheric Environment* 44(24): 2881-86.

Arnett, S. D., D. M. Osbourn, K. D. Moore, S. S. Vandaveer, and C. E. Lunte. 2005. "Determination of 8-oxoguanine and 8-hydroxy-2′-deoxyguanosine in the rat cerebral cortex using microdialysis sampling and capillary electrophoresis with electrochemical detection." *Journal of Chromatography B* 827(1): 16-25.

Avakian, M. D., B. Dellinger, H. Fiedler, B. Gullet, C. Koshland, S. Marklund, G. Oberdörster, S. Safe, A. Sarofim, and K. R. Smith. 2002. "The origin, fate, and health effects of combustion by-products: a research framework." *Environmental health perspectives* 110(11): 1155.

Balakrishnan, K., P. Ramaswamy, S. Sambandam, G. Thangavel, S. Ghosh, P. Johnson, K. Mukhopadhyay, V. Venugopal, and V. Thanasekaraan. 2011. "Air pollution from household solid fuel combustion in India: an overview of exposure and health related information to inform health research priorities." *Global health action* 4.

Balakrishnan, K., P. Ramaswamy, and S. Sankar. 2004. "Biomass Smoke and Health Risks–The Situation in Developing Countries." *Air Pollution*: 219-39.

Ballard-Tremeer, G. and H. Jawurek. 1996. "Comparison of five rural, wood-burning cooking devices: efficiencies and emissions." *Biomass and Bioenergy* 11(5): 419-30.

Banerjee, A., N. K. Mondal, D. Das, and M. R. Ray. 2011. "Neutrophilic inflammatory response and oxidative stress in premenopausal women chronically exposed to indoor air pollution from biomass burning." *Inflammation*: 1-13.

Barregard, L., G. Sällsten, L. Andersson, A. C. Almstrand, P. Gustafson, M. Andersson, and A. C. Olin. 2008. "Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress." *Occupational and Environmental Medicine* 65(5): 319.

Barregard, L., G. Sällsten, P. Gustafson, L. Andersson, L. Johansson, S. Basu, and L. Stigendal. 2006. "Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation." *Inhalation Toxicology* 18(11): 845-53.

Bates, E., N. Bruce, A. Doig, and S. Gitonga. 2002. "Participatory approaches for alleviating indoor air pollution in rural Kenyan kitchens." *Boiling point*: 12-15.

Bølling, A. K., J. Pagels, K. E. Yttri, L. Barregard, G. Sallsten, P. E. Schwarze, and C. Boman. 2009. "Health effects of residential wood smoke particles: the importance of combustion conditions and physicochemical particle properties." *Particle and fibre toxicology* 6(1): 29.

Bruce, N., J. McCracken, R. Albalak, M. Schei, K. R. Smith, V. Lopez, and C. West. 2004. "Impact of improved stoves, house construction and child location on levels of indoor air pollution exposure in young Guatemalan children." *Journal of Exposure Science and Environmental Epidemiology* 14: S26-S33.

Bruce, N. G. 2010. "Symposium: The RESPIRE randomized control trial: preventing child ALRI by reducing household air pollution and the implications for pneumonia prevention globally. Presented at the Health Effects Institute 2010 annual conference, April 27th 2010."

Bryce, J., C. Boschi-Pinto, K. Shibuya, and R. E. Black. 2005. "WHO estimates of the causes of death in children." *The Lancet* 365(9465): 1147-52.

Chengappa, C., R. Edwards, R. Bajpai, K. N. Shields, and K. R. Smith. 2007. "Impact of improved cookstoves on indoor air quality in the Bundelkhand region in India." *Energy for Sustainable Development* 11(2): 33-44.

Chuang, C. Y., C. C. Lee, Y. K. Chang, and F. C. Sung. 2003. "Oxidative DNA damage estimated by urinary 8-hydroxydeoxyguanosine: influence of taxi driving, smoking and areca chewing." *Chemosphere* 52(7): 1163-71.

Clark, M. L., J. L. Peel, J. B. Burch, T. L. Nelson, M. M. Robinson, S. Conway, A. M. Bachand, and S. J. Reynolds. 2009. "Impact of improved cookstoves on indoor air pollution and adverse

health effects among Honduran women." *International Journal of Environmental Health Research* 19(5): 357-68.

Clark, M. L., S. J. Reynolds, J. B. Burch, S. Conway, A. M. Bachand, and J. L. Peel. 2010. "Indoor air pollution, cookstove quality, and housing characteristics in two Honduran communities." *Environmental research* 110(1): 12-18.

Cynthia, A. A., R. D. Edwards, M. Johnson, M. Zuk, L. Rojas, R. D. Jiménez, H. Riojas-Rodriguez, and O. Masera. 2008. "Reduction in personal exposures to particulate matter and carbon monoxide as a result of the installation of a Patsari improved cook stove in Michoacan Mexico." *Indoor air* 18(2): 93-105.

Danielsen, P. H., E. V. Bräuner, L. Barregard, G. Sällsten, M. Wallin, R. Olinski, R. Rozalski, P. Møller, and S. Loft. 2008. "Oxidatively damaged DNA and its repair after experimental exposure to wood smoke in healthy humans." *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis* 642(1-2): 37-42.

Danielsen, P. H., S. Loft, N. R. Jacobsen, K. A. Jensen, H. Autrup, J. L. Ravanat, H. Wallin, and P. Møller. 2010. "Oxidative stress, inflammation, and DNA damage in rats after intratracheal instillation or oral exposure to ambient air and wood smoke particulate matter." *Toxicological Sciences* 118(2): 574-85.

Danielsen, P. H., S. Loft, A. Kocbach, P. E. Schwarze, and P. Møller. 2009. "Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines." *Mutation Research/Genetic Toxicology and Environmental Mutagenesis* 674(1-2): 116-22.

Danielsen, P. H., P. Møller, K. A. Jensen, A. K. Sharma, H. Wallin, R. Bossi, H. Autrup, L. Mølhave, J. L. Ravanat, and J. J. Briedé. 2011. "Oxidative stress, DNA damage, and inflammation induced by ambient air and wood smoke particulate matter in human A549 and THP-1 cell lines." *Chemical Research in Toxicology*.

Dennis, R. J., D. Maldonado, S. Norman, E. Baena, and G. Martinez. 1996. "Woodsmoke exposure and risk for obstructive airways disease among women." *Chest* 109(1): 115-19.

Diaz, E., N. Bruce, D. Pope, A. Diaz, K. Smith, and T. Smith-Sivertsen. 2008. "Self-rated health among Mayan women participating in a randomised intervention trial reducing indoor air pollution in Guatemala." *BMC International Health and Human Rights* 8(1): 7.

Dionisio, K., S. Howie, K. Fornace, O. Chimah, R. Adegbola, and M. Ezzati. 2008. "Measuring the exposure of infants and children to indoor air pollution from biomass fuels in The Gambia." *Indoor air* 18(4): 317-27.

Dionisio, K. L., S. R. C. Howie, F. Dominici, K. M. Fornace, J. D. Spengler, S. Donkor, O. Chimah, C. Oluwalana, R. C. Ideh, and B. Ebruke. 2011. "The exposure of infants and children

to carbon monoxide from biomass fuels in The Gambia: a measurement and modeling study." *Journal of Exposure Science and Environmental Epidemiology*.

Dionisio, K. L., S. R. C. Howie, F. Dominici, K. M. Fornace, J. D. Spengler, S. Donkor, O. Chimah, C. Oluwalana, R. C. Ideh, B. Ebruke, R. A. Adegbola, and M. Ezzati. 2012. "The exposure of infants and children to carbon monoxide from biomass fuels in The Gambia: a measurement and modeling study." *J Expos Sci Environ Epidemiol* 22(2): 173-81.

Dräger-Tubes & Chip-Measurement-System Handbook (PDF). 16th edition Soil, W., and Air Investigations as well as Technical Gas Analysis Carbon monoxide 50/a-D, 6733191.

Drost, E., K. Skwarski, J. Sauleda, N. Soler, J. Roca, A. Agusti, and W. MacNee. 2005. "Oxidative stress and airway inflammation in severe exacerbations of COPD." *Thorax* 60(4): 293.

Dutta, K., K. N. Shields, R. Edwards, and K. R. Smith. 2007. "Impact of improved biomass cookstoves on indoor air quality near Pune, India." *Energy for Sustainable Development* 11(2): 19-32.

Edwards, R. D., Y. Liu, G. He, Z. Yin, J. Sinton, J. Peabody, and K. Smith. 2007. "Household CO and PM measured as part of a review of China's National Improved Stove Program." *Indoor air* 17(3): 189-203.

Ekici, A., M. Ekici, E. Kurtipek, A. Akin, M. Arslan, T. Kara, Z. Apaydin, and S. Demir. 2005. "Obstructive airway diseases in women exposed to biomass smoke." *Environmental research* 99(1): 93-98.

El Tayeb Muneer, S. and E. W. Mukhtar Mohamed. 2003. "Adoption of biomass improved cookstoves in a patriarchal society: an example from Sudan." *Science of the Total Environment* 307(1): 259-66.

England, T., E. Beatty, A. Rehman, J. Nourooz-Zadeh, P. Pereira, J. O'Reilly, H. Wiseman, C. Geissler, and B. Halliwell. 2000. "The steady-state levels of oxidative DNA damage and of lipid peroxidation (F2-isoprostanes) are not correlated in healthy human subjects." *Free Radical Research* 32(4): 355-62.

Ezzati, M. and D. M. Kammen. 2002. "The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps, and data needs." *Environmental health perspectives* 110(11): 1057.

Fan, R., D. Wang, C. Mao, S. Ou, Z. Lian, S. Huang, Q. Lin, R. Ding, and J. She. 2011. "Preliminary study of children's exposure to PAHs and its association with 8-hydroxy-2'-deoxyguanosine in Guangzhou, China." *Environment international*.

Fitzgerald, C., M. Aguilar-Villalobos, A. R. Eppler, S. C. Dorner, S. L. Rathbun, and L. P. Naeher. 2012a. "Testing the effectiveness of two improved cookstove interventions in the Santiago de Chuco Province of Peru." *Science of the Total Environment* 420(0): 54-64.

Fitzgerald, C., M. Aguilar-Villalobos, A. R. Eppler, S. C. Dorner, S. L. Rathbun, and L. P. Naeher. 2012b. "Testing the effectiveness of two improved cookstove interventions in the Santiago de Chuco Province of Peru." *Science of the Total Environment*.

Fullerton, D. G., N. Bruce, and S. B. Gordon. 2008. "Indoor air pollution from biomass fuel smoke is a major health concern in the developing world." *Transactions of the Royal Society of Tropical Medicine and Hygiene* 102(9): 843-51.

GACC. 2011. "The Global Alliance for Clean Cookstoves: Overview [Document on the Internet] [updated 2011; cited 2011 Oct 23]. Available from http://cleancookstoves.org/."

Ghio, A. J., J. M. Soukup, M. Case, L. A. Dailey, J. Richards, J. Berntsen, R. B. Devlin, S. Stone, and A. Rappold. 2011. "Exposure to wood smoke particles produces inflammation in healthy volunteers." *Occupational and Environmental Medicine*.

Ghio, A. J., J. M. Soukup, M. Case, L. A. Dailey, J. Richards, J. Berntsen, R. B. Devlin, S. Stone, and A. Rappold. 2012. "Exposure to wood smoke particles produces inflammation in healthy volunteers." *Occupational and Environmental Medicine* 69(3): 170-75.

Gniwotta, C., J. D. Morrow, L. J. Roberts II, and H. Kühn. 1997. "Prostaglandin F2-like compounds, F2-isoprostanes, are present in increased amounts in human atherosclerotic lesions." *Arteriosclerosis, thrombosis, and vascular biology* 17(11): 3236-41.

Gopalan, H. N. B., S. Saksena, I. Tata Energy Research, and P. United Nations Environment. 1999. *Domestic environment and health of women and children*. New Delhi: Tata Energy Research Institute [and] United Nations Environment Programme, New Delhi, PNUMA, 253 p.

Hartinger, S., Commodore, AA, Hattendorf, J, Lanata, CF, Gil, AI, Verastegui, H, Aguilar-Villalobos, M, Mäusezahl, D, Naeher, LP. in review. "Chimney stoves in an intervention study do not improve household air pollution when compared with traditional open fire stoves in rural Peru." *Indoor air*.

Hartinger, S., C. Lanata, J. Hattendorf, A. Gil, H. Verastegui, T. Ochoa, and D. Mäusezahl. 2011. "A community randomised controlled trial evaluating a home-based environmental intervention package of improved stoves, solar water disinfection and kitchen sinks in rural Peru: Rationale, trial design and baseline findings." *Contemporary Clinical Trials* 32(6): 864-73.

Hartinger, S., Lanata, CF, Gil, AI, Hattendorf, J, Verastegui, H, Mäusezahl, D. . 2012. "Combining interventions: improved stove, kitchen sinks and solar disinfection of drinking water and kitchen cloths to improve hygiene in rural Peru." *Field Actions Science Reports [Online]*,

Special Issue 6 | 2012, Online since 31 May 2012, Connection on 24 September 2012. URL : http://factsreports.revues.org/1627.

Hartinger SM, L. C., Gil, AI, Hattendorf J, Verastegui H, Mäusezahl, D "Combining interventions: improved stove, kitchen sinks and solar disinfection of drinking water and kitchen cloths to improve hygiene in rural Peru." *Field Actions Science Reports [Online], Special Issue 6 | 2012, Online since 31 May 2012, Connection on 24 September 2012. URL : http://factsreports.revues.org/1627*.

Henkle, J., C. Mandzuk, E. Emery, L. Schrowe, and J. Sevilla-Martir. 2010. "Global Health and International Medicine: Honduras Stove Project." *Hispanic Health Care International* 8(1): 36-46.

Hosgood, H. D., H. Wei, A. Sapkota, I. Choudhury, N. Bruce, K. R. Smith, N. Rothman, and Q. Lan. 2011. "Household coal use and lung cancer: systematic review and meta-analysis of case–control studies, with an emphasis on geographic variation." *International Journal of Epidemiology* 40(3): 719-28.

IARC. 2010. "Household use of solid fuels and high-temperature frying." *IARC Monogr Eval Carcinog Risks Hum.* 95: 1-430.

INEI. 2007. "Instituto Nacional de Estadística e Informática[Document on the Internet] [updated 2007; cited 2012 May 10]. Available from http://www.inei.gob.pe."

Jalava, P. I., R. O. Salonen, K. Nuutinen, A. S. Pennanen, M. S. Happo, J. Tissari, A. Frey, R. Hillamo, J. Jokiniemi, and M. R. Hirvonen. 2010. "Effect of combustion condition on cytotoxic and inflammatory activity of residential wood combustion particles." *Atmospheric Environment* 44(13): 1691-98.

Khushk, W., Z. Fatmi, F. White, and M. Kadir. 2005. "Health and social impacts of improved stoves on rural women: a pilot intervention in Sindh, Pakistan." *Indoor air* 15(5): 311-16.

Kim, J. Y., S. Mukherjee, L. C. Ngo, and D. C. Christiani. 2004. "Urinary 8-hydroxy-2'-deoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to fine particulates." *Environmental health perspectives* 112(6): 666.

Kim, K. H., S. A. Jahan, and E. Kabir. 2011a. "A review of diseases associated with household air pollution due to the use of biomass fuels." *Journal of hazardous materials*.

Kim, K. H., S. A. Jahan, and E. Kabir. 2011b. "A review of diseases associated with household air pollution due to the use of biomass fuels." *Journal of hazardous materials* 192(2): 425-31.

Kimura, S., H. Yamauchi, Y. Hibino, M. Iwamoto, K. Sera, and K. Ogino. 2006. "Evaluation of urinary 8-hydroxydeoxyguanine in healthy Japanese people." *Basic & Clinical Pharmacology & Toxicology* 98(5): 496-502.

Kinnula, V. L., H. Ilumets, M. Myllärniemi, A. Sovijärvi, and P. Rytilä. 2007. "8-Isoprostane as a marker of oxidative stress in nonsymptomatic cigarette smokers and COPD." *European Respiratory Journal* 29(1): 51-55.

Kipen, H., D. Rich, W. Huang, T. Zhu, G. Wang, M. Hu, S. Lu, P. Ohman-Strickland, P. Zhu, and Y. Wang. 2010. "Measurement of inflammation and oxidative stress following drastic changes in air pollution during the Beijing Olympics: a panel study approach." *Annals of the New York Academy of Sciences* 1203(1): 160-67.

Kocbach, A., J. I. Herseth, M. Låg, M. Refsnes, and P. E. Schwarze. 2008a. "Particles from wood smoke and traffic induce differential pro-inflammatory response patterns in co-cultures." *Toxicology and applied pharmacology* 232(2): 317-26.

Kocbach, A., E. Namork, and P. E. Schwarze. 2008b. "Pro-inflammatory potential of wood smoke and traffic-derived particles in a monocytic cell line." *Toxicology* 247(2-3): 123-32.

Kurmi, O. P., S. Semple, P. Simkhada, W. C. S. Smith, and J. G. Ayres. 2010. "COPD and chronic bronchitis risk of indoor air pollution from solid fuel: a systematic review and meta-analysis." *Thorax* 65(3): 221-28.

'Lai, C., S. Liou, H. Lin, T. Shih, P. Tsai, J. Chen, T. Yang, J. Jaakkola, and P. Strickland. 2005. "Exposure to traffic exhausts and oxidative DNA damage." *Occupational and Environmental Medicine* 62(4): 216-22.

Lai, C. H., S. H. Liou, J. J. K. Jaakkola, H. B. Huang, T. Y. Su, and P. T. Strickland. 2012. "Exposure to Polycyclic Aromatic Hydrocarbon Associated with Traffic Exhaust Increases Lipid Peroxidation and Reduces Antioxidant Capacity."

Lee, M.-W., M.-L. Chen, S.-C. C. Lung, C.-J. Tsai, X.-J. Yin, and I. F. Mao. 2010. "Exposure assessment of PM2.5 and urinary 8-OHdG for diesel exhaust emission inspector." *Science of the Total Environment* 408(3): 505-10.

Lewtas, J. 2007. "Air pollution combustion emissions: characterization of causative agents and mechanisms associated with cancer, reproductive, and cardiovascular effects." *Mutation Research/Reviews in Mutation Research* 636(1-3): 95-133.

Li, C., S. Kang, P. Chen, Q. Zhang, J. Guo, J. Mi, P. Basang, Q. Luosang, and K. R. Smith. 2012a. "Personal PM< sub> 2.5</sub> and indoor CO in nomadic tents using open and chimney biomass stoves on the Tibetan Plateau." *Atmospheric Environment*.

Li, N., T. Xia, and A. E. Nel. 2008. "The role of oxidative stress in ambient particulate matter-induced lung diseases and its implications in the toxicity of engineered nanoparticles." *Free Radical Biology and Medicine* 44(9): 1689-99.

Li, Z., A. Commodore, S. Hartinger, E. Pittman, D. Trinidad, R. Ostovar, K. Hubbard, A. Sjodin, C. Lanata, A. Gil, D. Mäusezahl, and L. Naeher. 2012b. "O-046: Assessment of Exposure to PAHs in Cook Stove Intervention Projects in Peru by Urinary Biomonitoring." *Epidemiology* 23(5S): 10.1097/01.ede.0000416704.62764.3d.

Li, Z., L. C. Romanoff, A. Debra, N. Hussain, R. S. Jones, E. N. Porter, D. G. Patterson Jr, and A. Sjödin. 2006. "Measurement of urinary monohydroxy polycyclic aromatic hydrocarbons using automated liquid-liquid extraction and gas chromatography/isotope dilution high-resolution mass spectrometry." *Analytical chemistry* 78(16): 5744-51.

Li, Z., A. Sjödin, L. C. Romanoff, K. Horton, C. L. Fitzgerald, A. Eppler, M. Aguilar-Villalobos, and L. P. Naeher. 2011. "Evaluation of exposure reduction to indoor air pollution in stove intervention projects in Peru by urinary biomonitoring of polycyclic aromatic hydrocarbon metabolites." *Environment international* 37(7): 1157-63.

Limmeechokchai, B. and S. Chawana. 2007. "Sustainable energy development strategies in the rural Thailand: The case of the improved cooking stove and the small biogas digester." *Renewable and Sustainable Energy Reviews* 11(5): 818-37.

Ling, S. H. and S. F. Van Eeden. 2009. "Particulate matter air pollution exposure: role in the development and exacerbation of chronic obstructive pulmonary disease." *International journal of chronic obstructive pulmonary disease* 4: 233.

Lisouza, F. A., O. P. Owuor, and J. O. Lalah. 2011. "Variation in indoor levels of polycyclic aromatic hydrocarbons from burning various biomass types in the traditional grass-roofed households in Western Kenya." *Environmental Pollution* 159(7): 1810-15.

Liu, S., Y. Zhou, X. Wang, D. Wang, J. Lu, J. Zheng, N. Zhong, and P. Ran. 2007. "Biomass fuels are the probable risk factor for chronic obstructive pulmonary disease in rural South China." *Thorax* 62(10): 889-97.

Lodovici, M., C. Casalini, R. Cariaggi, L. Michelucci, and P. Dolara. 2000. "Levels of 8-hydroxydeoxyguanosine as a marker of DNA damage in human leukocytes." *Free Radical Biology and Medicine* 28(1): 13-17.

Loft, S., K. Vistisen, M. Ewertz, A. Tjønneland, K. Overvad, and H. E. Poulsen. 1992. "Oxidative DNA damage estimated by 8-hydroxydeoxyguanosine excretion in humans: influence of smoking, gender and body mass index." *Carcinogenesis* 13(12): 2241.

Lokras, S. 2012. "Development and Dissemination of Fuel-Efficient Biomass Burning Devices." *Journal of the Indian Institute of Science* 92(1): 99-110.

Lu, C. Y., Y. C. Ma, J. M. Lin, C. Y. Chuang, and F. C. Sung. 2007. "Oxidative DNA damage estimated by urinary 8-hydroxydeoxyguanosine and indoor air pollution among non-smoking office employees." *Environmental research* 103(3): 331-37.

Martin, W. J., R. I. Glass, J. M. Balbus, and F. S. Collins. 2011. "A Major Environmental Cause of Death." *Science* 334(6053): 180-81.

Masera, O., R. Edwards, C. A. Arnez, V. Berrueta, M. Johnson, L. R. Bracho, H. Riojas-Rodríguez, and K. R. Smith. 2007. "Impact of Patsari improved cookstoves on indoor air quality in Michoacán, Mexico." *Energy for Sustainable Development* 11(2): 45-56.

McBride, J. P., R. E. Moore, J. P. Witherspoon, and R. E. Blanco. 1978. "Radiological Impact of Airborne Effluents of Coal and Nuclear Plants." *Science* 202(4372): 1045-50.

McCracken, J., K. R. Smith, P. Stone, A. Díaz, B. Arana, and J. Schwartz. 2011. "Intervention to Lower Household Wood Smoke Exposure in Guatemala Reduces ST-Segment Depression on Electrocardiograms." *Environmental health perspectives* 119(11): 1562.

McCracken, J. P., J. Schwartz, N. Bruce, M. Mittleman, L. M. Ryan, and K. R. Smith. 2009. "Combining individual-and group-level exposure information: child carbon monoxide in the Guatemala woodstove randomized control trial." *Epidemiology* 20(1): 127.

McCracken, J. P. and K. R. Smith. 1998. "Emissions and efficiency of improved woodburning cookstoves in Highland Gatemala." *Environment international* 24(7): 739-47.

McCracken, J. P., K. R. Smith, A. Díaz, M. A. Mittleman, and J. Schwartz. 2007. "Chimney stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women." *Environmental health perspectives* 115(7): 996.

Montuschi, P., M. Corradi, G. CIABATTONI, J. Nightingale, S. A. Kharitonov, and P. J. Barnes. 1999. "Increased 8-isoprostane, a marker of oxidative stress, in exhaled condensate of asthma patients." *American journal of respiratory and critical care medicine* 160(1): 216-20.

Naeher, L., K. Smith, B. Leaderer, L. Neufeld, and D. Mage. 2001. "Carbon monoxide as a tracer for assessing exposures to particulate matter in wood and gas cookstove households of highland Guatemala." *Environmental science & technology* 35(3): 575-81.

Naeher, L. P., M. Brauer, M. Lipsett, J. T. Zelikoff, C. D. Simpson, J. Q. Koenig, and K. R. Smith. 2007. "Woodsmoke Health Effects: A Review." *Inhalation Toxicology* 19(1): 67-106.

Nazmul Alam, S. and S. J. Chowdhury. 2010. "Improved earthen stoves in coastal areas in Bangladesh: Economic, ecological and socio-cultural evaluation." *Biomass and Bioenergy* 34(12): 1954-60.

Northcross, A., Z. Chowdhury, J. McCracken, E. Canuz, and K. R. Smith. 2010. "Estimating personal PM2. 5 exposures using CO measurements in Guatemalan households cooking with wood fuel." *J. Environ. Monit.* 12(4): 873-78.

Nuernberg, A. M., P. D. Boyce, J. M. Cavallari, S. C. Fang, E. A. Eisen, and D. C. Christiani. 2008. "Urinary 8-isoprostane and 8-OHdG concentrations in boilermakers with welding exposure." *Journal of occupational and environmental medicine* 50(2): 182.

Oanh, N. T. K., L. B. Reutergårdh, and N. T. Dung. 1999. "Emission of polycyclic aromatic hydrocarbons and particulate matter from domestic combustion of selected fuels." *Environmental science & technology* 33(16): 2703-09.

Orozco-Levi, M., J. Garcia-Aymerich, J. Villar, A. Ramirez-Sarmiento, J. Anto, and J. Gea. 2006. "Wood smoke exposure and risk of chronic obstructive pulmonary disease." *European Respiratory Journal* 27(3): 542-46.

Palmer, K., R. McNeill-Love, J. Poole, D. Coggon, A. Frew, C. Linaker, and J. Shute. 2006. "Inflammatory responses to the occupational inhalation of metal fume." *European Respiratory Journal* 27(2): 366-73.

Patel, P. R., R. J. Bevan, N. Mistry, and J. Lunec. 2007. "Evidence of oligonucleotides containing 8-hydroxy-2′-deoxyguanosine in human urine." *Free Radical Biology and Medicine* 42(4): 552-58.

Pennise, D., S. Brant, S. M. Agbeve, W. Quaye, F. Mengesha, W. Tadele, and T. Wofchuck. 2009. "Indoor air quality impacts of an improved wood stove in Ghana and an ethanol stove in Ethiopia." *Energy for Sustainable Development* 13(2): 71-76.

Perez-Padilla, R., A. Schilmann, and H. Riojas-Rodriguez. 2010. "Respiratory health effects of indoor air pollution [Review article]." *The International Journal of Tuberculosis and Lung Disease* 14(9): 1079-86.

Peterson, J., M. MacDonell, L. Haroun, F. Monette, R. D. Hildebrand, and A. Taboas. 2007. "Radiological and chemical fact sheets to support health risk analyses for contaminated areas." *Human Health Fact Sheet, Argonne*: 38-39.

Pilger, A., D. Germadnik, K. Riedel, I. Meger-Kossien, G. Scherer, and H. W. Rüdiger. 2001. "Longitudinal study of urinary 8-hydroxy-2'-deoxyguanosine excretion in healthy adults." *Free Radical Research* 35(3): 273-80.

Pilger, A., S. Ivancsits, D. Germadnik, and H. Rudiger. 2002. "Urinary excretion of 8-hydroxy-2'-deoxyguanosine measured by high-performance liquid chromatography with electrochemical detection." *Journal of Chromatography B* 778(1-2): 393-401.

Pope III, C. A., R. T. Burnett, M. C. Turner, A. Cohen, D. Krewski, M. Jerrett, S. M. Gapstur, and M. J. Thun. 2011. "Lung Cancer and Cardiovascular Disease Mortality Associated with Ambient Air Pollution and Cigarette Smoke: Shape of the Exposure–Response Relationships." *Environmental health perspectives* 119(11): 1616.

Regalado, J., R. Pérez-Padilla, R. Sansores, J. I. P. Ramirez, M. Brauer, P. Paré, and S. Vedal. 2006. "The effect of biomass burning on respiratory symptoms and lung function in rural Mexican women." *American journal of respiratory and critical care medicine* 174(8): 901-05.

Rehfuess, E., S. Mehta, and A. Prüss-Üstün. 2006. "Assessing household solid fuel use: multiple implications for the Millennium Development Goals." *Environmental health perspectives* 114(3): 373.

Roberts II, L. J. and J. D. Morrow. 2000. "Measurement of F2-isoprostanes as an index of oxidative stress in vivo." *Free Radical Biology and Medicine* 28(4): 505-13.

Romieu, I., H. Riojas-Rodriguez, A. T. Marron-Mares, A. Schilmann, R. Perez-Padilla, and O. Masera. 2009. "Improved biomass stove intervention in rural Mexico: impact on the respiratory health of women." *American journal of respiratory and critical care medicine*: 200810-1556OCv1.

Saha, A., P. Kulkarni, A. Shah, M. Patel, and H. Saiyed. 2005. "Ocular morbidity and fuel use: an experience from India." *Occupational and Environmental Medicine* 62(1): 66.

Sakano, N., D. H. Wang, N. Takahashi, B. Wang, R. Sauriasari, S. Kanbara, Y. Sato, T. Takigawa, J. Takaki, and K. Ogino. 2009. "Oxidative stress biomarkers and lifestyles in Japanese healthy people." *Journal of clinical biochemistry and nutrition* 44(2): 185.

Saksena, S., R. Prasad, R. Pal, and V. Joshi. 1992. "Patterns of daily exposure to TSP and CO in the Garhwal Himalaya." *Atmospheric Environment. Part A. General Topics* 26(11): 2125-34.

Smith, K. R. 1987. *Biofuels, air pollution, and health: a global review*: Plenum press.

Smith, K. R., J. P. McCracken, L. Thompson, R. Edwards, K. N. Shields, E. Canuz, and N. Bruce. 2010. "Personal child and mother carbon monoxide exposures and kitchen levels: Methods and results from a randomized trial of woodfired chimney cookstoves in Guatemala (RESPIRE)." *J Expos Sci Environ Epidemiol* 20(5): 406-16.

Smith, K. R., J. P. McCracken, M. W. Weber, A. Hubbard, A. Jenny, L. M. Thompson, J. Balmes, A. Diaz, B. Arana, and N. Bruce. 2011. "Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial." *The Lancet* 378(9804): 1717-26.

Smith, K. R. and S. Mehta. 2003. "The burden of disease from indoor air pollution in developing countries: comparison of estimates." *International Journal of Hygiene and Environmental Health* 206(4-5): 279-89.

Smith, K. R. and J. L. Peel. 2010. "Mind the gap." *Environmental health perspectives* 118(12): 1643.

Smith, K. R., J. M. Samet, I. Romieu, and N. Bruce. 2000. "Indoor air pollution in developing countries and acute lower respiratory infections in children." *Thorax* 55(6): 518-32.

Swiston, J. R., W. Davidson, S. Attridge, G. T. Li, M. Brauer, and S. F. van Eeden. 2008. "Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters." *European Respiratory Journal* 32(1): 129-38.

Tamura, S., H. Tsukahara, M. Ueno, M. Maeda, H. Kawakami, K. Sekine, and M. Mayumi. 2006. "Evaluation of a urinary multi-parameter biomarker set for oxidative stress in children, adolescents and young adults." *Free Radical Research* 40(11): 1198-205.

Thompson, L. M., N. Bruce, B. Eskenazi, A. Diaz, D. Pope, and K. R. Smith. 2011. "Impact of Reduced Maternal Exposures to Wood Smoke from an Introduced Chimney Stove on Newborn Birth Weight in Rural Guatemala." *Environmental health perspectives* 119(10): 1489.

Tuohy, K. M., D. J. S. Hinton, S. J. Davies, M. J. C. Crabbe, G. R. Gibson, and J. M. Ames. 2006. "Metabolism of Maillard reaction products by the human gut microbiota–implications for health." *Molecular nutrition & food research* 50(9): 847-57.

Van Eeden, S., J. Leipsic, S. F. P. Man, and D. D. Sin. 2012. "The relationship between lung inflammation and cardiovascular disease." *American journal of respiratory and critical care medicine* 186(1): 11-16.

van Eeden, S. F. and D. D. Sin. 2008. "Chronic obstructive pulmonary disease: a chronic systemic inflammatory disease." *Respiration* 75(2): 224-38.

Venkataraman, C., A. Sagar, G. Habib, N. Lam, and K. Smith. 2010. "The Indian national initiative for advanced biomass cookstoves: the benefits of clean combustion." *Energy for Sustainable Development* 14(2): 63-72.

Volkerding, J. M. 2004. "Comparison of the radiological dose from the Cerro Grande fire to a natural wildfire." *Environment International* 29(7): 987-93.

Wang, Z., D. Neuburg, C. Li, L. Su, J. Y. Kim, J. C. Chen, and D. C. Christiani. 2005. "Global gene expression profiling in whole-blood samples from individuals exposed to metal fumes." *Environmental health perspectives* 113(2): 233.

WHO. 2011. "Indoor air pollution and health; fact sheet 292.".

Wu, L. L., C.-C. Chiou, P.-Y. Chang, and J. T. Wu. 2004. "Urinary 8-OHdG: a marker of oxidative stress to DNA and a risk factor for cancer, atherosclerosis and diabetics." *Clinica Chimica Acta* 339(1-2): 1-9.

Y von Schirnding, N. B., K Smith, G Ballard-Tremeer, M Ezzati, K Lvovsky. 2002. *Addressing the Impact of Household Energy and Indoor Air Pollution on the Health of Poor: Implications*

for Policy Action and Intervention Measures—paper prepared for the Commission on Macroeconomics and HealthWHO, Geneva (2002): World Health Organization.

Zhang, J. J. and K. R. Smith. 2007. "Household air pollution from coal and biomass fuels in China: measurements, health impacts, and interventions." *Environmental health perspectives* 115(6): 848.

APPENDICES

APPENDIX A

ASSESSMENT OF EXPOSURE TO PAHS IN COOKSTOVE INTERVENTION PROJECTS IN PERU BY URINARY BIOMONITORING[1]

[1] Li, Z., Commodore, A., Hartinger, S., Pittman, E., Trinidad, D., Ostovar, R., Hubbard, K., Sjodin, A., Lanata, C., Gil, A., Mäusezahl, D., Naeher, L. 2012. *Epidemiology* 23: 10.1097/01.ede.0000416704.62764.3d. Reprinted here with permission of the publisher.

ABSTRACT

Background: Burning biomass fuels in indoor open fire stoves is common in developing countries, which releases harmful combustion products such as particulate matter ($PM_{2.5}$) and polycyclic aromatic hydrocarbons (PAHs).

Objectives: We aimed to investigate if replacing open fire stoves with improved stoves equipped with a chimney would reduce exposure to PAHs.

Methods: We conducted two studies in Peru. The first, an intervention study, investigated concentration of urinary PAH biomarkers and $PM_{2.5}$ and CO in air samples in 57 female participants/homes before and three weeks after installing new stoves. The second study is part of a community randomized controlled trial. First-morning urine specimens were collected from female participants in 122 homes used open-fire stoves and 216 homes with chimney-equipped stoves.

Results: Three weeks after using the new stoves in the intervention study, concentration of $PM_{2.5}$ and CO in air were significantly reduced by 43-69%, as did all 10 urinary PAH biomarkers (creatinine-adjusted, 17-43% reduction). In the second study, preliminary fresh weight results showed significantly lower levels of urinary 2-napthol (35%) and 3-hydroxyfluorene (23%) in persons using improved stoves compared to open-fire stove users. Urinary OH-PAH levels in both studies were comparable or higher than the 95^{th} percentile of the levels in the general US population.

Conclusion: Indoor open-fire stoves can be improved by installing stoves with chimney, which lead to reduced exposure to PAH in a short term. However, the long term effect is unclear. Even with the improved stoves, exposure to PAHs in this population remains high compared to reference population.

APPENDIX B

CHIMNEY STOVES IN AN INTERVENTION STUDY DO NOT IMPROVE HOUSEHOLD AIR POLLUTION WHEN COMPARED WITH TRADITIONAL OPEN FIRE STOVES IN RURAL PERU[1]

[1] Hartinger, S., Commodore, AA, Hattendorf, J, Lanata, CF, Gil, AI, Verastegui, H, Aguilar-Villalobos, M, Mäusezahl, D, Naeher, LP. Submitted to *Indoor Air,* 05/17/2012.

ABSTRACT

Background: Nearly half of the world's population depends on biomass fuels to meet domestic energy needs, producing high levels of pollutants responsible for substantial morbidity and mortality globally.

Objectives: We compare carbon monoxide (CO) and particulate matter ($PM_{2.5}$) exposures and kitchen concentrations in households with study promoted intervention (OPTIMA) stoves and control stoves in San Marcos Province, Cajamarca region, Peru.

Methods: The study was embedded in a community randomized trial. We determined 48hr indoor air concentration levels of CO and $PM_{2.5}$ in 93 kitchen environments and personal exposure, after OPTIMA-improved stoves had been installed for an average of seven months

Results: Household kitchen concentration of $PM_{2.5}$ and CO did not differ significantly between OPTIMA-improved stoves and control stoves. However a post-hoc stratification of improved stoves by level of performance revealed mean $PM_{2.5}$ and CO levels of the fully functional (or FL-I) stoves were 28% lower (n=20, $PM_{2.5,}$ 136μg/m^3 95%CI 54-217) and 45% lower (n=25, CO, 3.2ppm, 95%CI 1.5-4.9) in the kitchen environment compared to the control stoves (n=34, $PM_{2.5}$, 189μg/m^3, 95%CI 116–261; n=44, CO, 5.8ppm, 95%CI 3.3-8-2). Likewise, personal exposures for OPTIMA-improved stoves were 43% and 167% lower for $PM_{2.5}$ (n=23) and CO (n=25). At the time of sampling, 66% (29/43) of the Optima-improved stoves were properly maintained.

Conclusion: No difference in $PM_{2.5}$ and CO concentrations was observed between OPTIMA-improved and control stoves. Stove maintenance and functionality level are factors worthy of consideration for future evaluations of stove interventions.

INTRODUCTION

Approximately half of the world's population continues to depend on biomass fuels in order to meet their basic energy needs for cooking, boiling water, lighting and heating (Rehfuess et al. 2006, Martin et al. 2011). Burning biomass fuels in un-vented stoves and closed rooms produces high levels of pollutants (Fullerton et al. 2008; Smith et al. 2000) beyond the USEPA National Ambient Air Quality Standards (EPA, 2005). According to the WHO, household air pollution is responsible for about 1.6 million premature deaths per year due to incomplete biomass fuel (BMF) combustion (Smith et al. 2004), representing nearly 3% of the overall disease burden in developing countries. This large burden affects mainly women and small children (Rehfuess et al. 2006; Díaz et al. 2007) due to their continuous indoor exposure to health damaging pollutants, including several carcinogenic compounds, hazardous gases (CO and NOx) and fine particles while cooking (Naeher et al. 2007).

These pollutants increase the risk of acute lower respiratory infections, chronic obstructive pulmonary disease and may cause lung cancer (from coal stoves), asthma, low birth weight and other adverse birth outcomes (Po et al. 2011; Siddiqui et al. 2008; Tielsch et al. 2009), neurodevelopment impairments (Dix-Cooper et al. 2011), cardio-vascular and other inflammatory condition (Baumgartner et al. 2011; Clark et al. 2011; McCracken et al. 2011) , eye diseases, such as cataract and blindness (Smith and Mehta 2003; Saha et al. 2005) and headaches (Díaz et al. 2007).

In Peru almost 93% of the rural population relies on biomass fuels for cooking and heating (INEI, 2007). Exposure-response analysis shows the relationship between combustion particles and respiratory illnesses and the need to reach low levels of household air pollution from biomass fuel use to successfully reduce adverse health

effects including pneumonia (Smith and Peel. 2010; Smith et al. 2011). One of the most cost-effective household air pollution (HAP) control measures is the use of improved chimney stoves (Naeher, 2009), given they are adequately designed, installed, maintained and continuously used. A recent randomized controlled trial found significant reductions in severe pneumonia cases for children under 18 months after receiving a woodstove with chimney (Smith et al. 2011).

The Global Alliance for Clean Cookstoves (GACC) initiative launched on September 2010 (GACC, 2011) has provided a platform where different entities can converge into a common goal which is to deploy 100 million clean and efficient cookstoves by 2020. The GACC is supported by private, public and non-profit partners which aim to overcome the market barriers and achieve the establish goal. In Peru, two years prior to this initiative, several organisations aimed to install/deploy 500,000 certified biomass improved chimney stoves by 2011 (Bodereau, 2011); by the end of 2011 around 300,000 improved stoves were built. However, in many cases the success of these household indoor air pollution mitigation programmes, like the Peru national stove program, is often measured by the number of installed stoves rather than adoption, continuous utilization and maintenance by the users over time (Armendáriz-Arnez et al. 2010, Bodereau, 2011).

As part of a community cluster randomised controlled field trial carried out in the Cajamarca region of Peru, we installed 250 improved chimney stoves (called OPTIMA-improved stove), to determine their impact on reducing acute lower respiratory infections (ALRI) in children between the ages of six and 36 months (Hartinger et al. 2011).

The current study describes household air pollution levels of $PM_{2.5}$ and CO in 93 of the 250 kitchen environments and personal exposures of mothers at a median of seven months after the OPTIMA-improved stoves were installed. The effectiveness of the OPTIMA-improved stoves of improving air quality is compared to air pollution levels in control household using traditional stoves.

METHODS

Setting

The study was carried out in the northern highlands of Peru (Province of San Marcos, Cajamarca Region), between the months of June and August 2009 (dry season). The altitude ranges between 2200 and 3900 meters above sea level, with temperatures fluctuating between 7 to 25ºC and relative humidity between 59 to 73% as measured during the study period.

The population comprised mostly of farmers, typically living in small houses made out of earthen floors and adobe walls, with three or more people sleeping together in the same room. The majority of the population relied on firewood for cooking and heating. The wood was usually gathered from nearby shrubs, parcels of land or bought from the town or from local landowners. The cost of one load of wood (approx. 20kg) was about US$ 2.5 in local currency and usually lasted three to four days for cooking. Traditional stoves or open fires are usually located inside the house in an unventilated kitchen area (Hartinger et al. 2011). There were no relevant sources of outdoor or of indoor pollution (other than from open fire cooking) in study homes, and in the community.

Study Design and Enrolment

We conducted a cross-sectional HAP exposure assessment within the framework of a community-randomised controlled trial (c-RCT, parent study) of 51 communities in the San Marcos Province (Hartinger et al. 2011, Hartinger et al. 2012). The aim of the parent study was to evaluate an integrated home-based environmental intervention package (IHIP) against childhood diarrhoea and respiratory infections. The interventions comprised of an improved chimney stove – called OPTIMA- and a kitchen sink, complemented by the promotion of a solar disinfection method as a home-based water treatment (HWT), hand washing and kitchen hygiene. In an effort to increase desire to use the stove and foster sustained user compliance for future users and recipients of the interventions during the trial we conducted a pilot study in seven communities outside the study area, where we tested several potential designs, and consulted on cooking habits and preferences to provide a user-friendly stove design meeting their household and cooking needs. The families thus commented on operation and maintenance issues, size of the mouth of the stove, number of furnaces and heat emission need per furnace (Hartinger et al. 2012).

All OPTIMA-improved stoves were installed between October 2008 and January 2009 and evaluated for this study 6 and 8 months later (median 7.4 IQR = 6.6-8.1 month). All households from the parent study were eligible to participate, if they complied with the following criteria: (1) the stoves had to be located in a in-house kitchen environment (at least three full walls and a roof over the kitchen), (2) the households had to be within a half-hour walking distance from a road in order to transport the equipment and (3) the mother or caretaker had to agree to wear the equipment to measure air quality and comply with the project instructions for the duration of the study (48hr) and agree to sign the informed consent forms.

In the current study, households were conveniently selected from participating households of the parent study. Since we had a limited number of air quality measurement devices, we stopped the enrolment in each of the 51 communities after two households consented to participate. We enrolled a total of 93 households: 43 households had an OPTIMA-improved stove installed, 48 belonged to the control group of households using diverse cooking stoves (open fires N=35, self-improved stoves N=7, supplied by NGO N=6) and two household belonged to a neighbouring community were the NGO Sembrando had implemented an improved stove programme. We selected the two NGO household for comparison reasons and sampled them using the same selection criteria as described above.

Given that the control arm of the parent study included a diversity of stove types, the control households we selected for the current study also reflect this heterogeneity. This heterogeneity comprised the following stove types: 'open fire', 'self-improved by household' and 'supplied by NGO'. The 'open fires' included the "Tulpia" stove, the most common traditional three-stone fire stove type in this area. The 'self-improved by household' type includes all households which constructed a stove without support or advice from any organisation or institution. The "supplied by NGO" type included stoves provided by the national programme JUNTOS or independent NGOs such as ADIAR. These stoves were originally enrolled into the control arm of the RCT as control stoves which were improved by an NGO by the time enrolment for this study took place.

After the HAP exposure assessment (CO and $PM_{2.5}$ measurements), we decided to classify post-hoc all OPTIMA-improved stoves. All OPTIMA-improved stoves were re-visited 9 months (median 9.3 IQR= 9.0-9.7 month) after installation and repaired as needed by the original stove builders. The stoves were then stratified

into two functionality levels: FL-I that were at the time of the assessment in good running conditions (plastered stove and no visible leaks when in use) and FL-II stoves were in need of repairs (re-plastering, filling small cracks, cleaning the chimney, chimney valve replacement, etc). Among all OPTIMA-improved stoves, 159/250 (64%) were classified as FL-I, and 91/250 (36%) as FL-II. Among household participating in this study, 28/43 (66%) were classified as FL-I, and 15/43 (35%) as FL-II.

Household air pollution measurements

Personal exposure sampling

Personal air sampling equipment was placed in vests worn in the breathing zones of mother/caretakers for 48hr. These vests held real time CO monitors and 48hr time integrated $PM_{2.5}$ samplers. The sampling inlets were placed on the chest halfway between the throat and the diaphragm. Subjects were instructed to keep the vests on at all times except when sleeping or washing clothes, in which case the equipment was placed next to them. They were instructed to place vests on a nightstand next to their bed during the night.

To measure real-time CO exposure, each vest held a Draeger Pac III datalogger and a CO-specific sensor (Draeger Safety Inc., Pittsburgh, PA), set to record concentration levels at 30-second intervals. Forty-eight-hour time-integrated $PM_{2.5}$ samples were collected using particle-size-selective Triplex Cyclones (BGI Inc., Waltham, MA, Model SCC 1.062) and SKC universal sampling pumps (SKC Inc, Eighty Four, PA, Aircheck® XR5000), set to pull air at 1.5L per minute. Pre-flows and post flows were taken for each pump and all equipment was calibrated and cleaned per manufacturer protocol.

After 48 hours, the vests were retrieved; runtime starting and completion times were recorded at the household for each piece of equipment, and air sampling calculated thereof. Filters for each sampling day were placed in individual cassettes and stored in Ziploc bags in a -20°C freezer at the study site.

Kitchen environment air pollutant sampling

A stationary sampling box was placed indoors and at approximate breathing height (1.5m) adjacent to were the mother/caretaker stands for cooking. Each box contained a sampling pump (SKC Inc, Eighty Four, PA, Aircheck® 2000), a 12V battery, a filter/cyclone sampling train attached to Tygon® tubing, and a Pac III CO monitor (Draeger Safety Inc., Pittsburgh, PA). The same protocol as for the personal filters was used. After 48 hours, the equipment and sampling box were retrieved; runtimes were recorded at the household for each piece of equipment, and sampled filters were transported in a cooler from the household and stored in the lab freezer at -20°C.

Community air pollutant sampling

A central outdoor location was selected in San Marcos town to serve as a fixed sampling site, providing background levels of both CO (real time) and $PM_{2.5}$ (48hr time integrated) concentrations. A sampling scheme similar to that used in the study homes was set up outside a window at this stationary outdoor site. To measure real-time CO, a Langan CO monitor (Langan Products Inc., Elmwood Park, NJ, model T15n) was used. Forty-eight-hour time-integrated $PM_{2.5}$ was measured using a SKC Air Check pump with a BGI Triplex Cyclone and Teflon-coated glass fibre filter.

Laboratory, field and open blanks

Two laboratory filter blanks were collected at the time of the pre- and post weighing. During each sampling week, field blanks were collected to adjust for background noise in the equipment, and the open blanks collected to account for noise in the filter media. There were a total of 44 field and open blanks (mean ± SE): 28 field blanks (0.013 ± 0.002mg) and 16 open blanks (0.004 ± 0.001mg). There was approximately one field blank for each sampling day and an open blank for every other sampling day. Final particulate mass values for study samples have been adjusted for field filter blank values by subtracting the average of the field blank (13μg) from the post weights. The difference of the pre and adjusted post weights, together with the average volume of air sampled over the 48hr period were used to calculate mass concentrations. All mass concentrations are presented in $\mu g/m^3$.

Analysis of pumps and filters

To better describe daily variability in our exposure measurements (Smith et al. 2004), the homes were sampled for a 48hr period. The $PM_{2.5}$ measurements were only considered valid if the equipment ran for at least 2160 minutes. Filters were collected, stored at the site lab and transported on cold packs to the University of Georgia for gravimetric analysis. The filters were desiccated in climate-controlled conditions (21 ± 0.1°C; 40.9 ± 1.5% relative humidity) for 48hr prior weighing. Following the EPA's Quality Assurance Guidance Document (2005) each filter was weighed twice before and after sampling using a Cahn C-35 microbalance with a sensitivity of ±1μg. $PM_{2.5}$ concentrations (weight/ cubic meter air sampled) were derived by dividing the average mass of each filter weight by the intake volume of sampled air.

Compliance and observational data

We measured compliance and maternal cooking behaviour using questionnaires, conducting participatory observations and assessing compliance during monthly training visits as part of the c-RCT parent study. Questionnaires were administered on the second day of the indoor air sampling scheme. They were used to assess personal exposure to air pollution, behavioural habits (household chores, child care), mobility (including activities in- and around the home, attending the fields and commuting) cooking, cleaning, personal and household characteristics. We measured the kitchen volume and took window and door measurements (in cm).

Participatory observational data was collected as part of the c-RCT parent study in 236 (108 intervention and 128 controls) out of the 503 participating households. Such observational data were available for 18 out of 43 households with OPTIMA-improved stoves and 25 of the 48 control households in the present study. The mother/caretakers' behaviour was observed during the preparation of a lunch meal (9am-1pm) and recorded. Field workers remained at the household between three to four hours. This information provided input on the mother cooking practices and usage of the stove. Additionally, we measured compliance in all OPTIMA-improved stove homes (N=43), routinely monitored actual usage, maintenance and problems with the stoves, with the aim of determining daily use and the mothers perception of the maintenance level of their stoves.

Statistical Analysis

Data were analyzed using STATA 10.0. Personal and kitchen PM2.5 and CO means, standard deviations, confidence intervals and medians were calculated by stove type. Skewed data was log-transformed where appropriate; and, although we present levels of pollution and exposure using means (and medians), non-parametric

tests were used for comparisons. Scheffe's multiple comparison tests were used to calculate significant levels between study groups. Results were considered to be statistically significant at $p<0.05$.

Spearman correlation coefficients were calculated for air quality measurements, between kitchen $PM_{2.5}$ and CO measurements and between kitchen and personal $PM_{2.5}$ and CO measurements. Linear regression models were created to determine potential covariates that could explain the variation in air quality measurements in the kitchen environment and personal exposure. The variables with P values less than 0.25 in the bivariate model were included in the multivariable model.

Ethics

The study was approved by the Nutrition Research Institute (IIN) ethical review board, the institutional review boards at the University of Georgia and Emory University and the ethical review board at the Cayetano Heredia University. Written informed consent for this study was obtained from each study participant. The demographic and socio-economic data had previously been collected in the parent study (ClinicalTrials.gov Identifier: NCT00731497) which had received clearance from the independent ethics committees of IIN and the ethical review board of University of Basel, Switzerland (Ethikkommission Beider Basel, EKBB). The participant information provided and the informed consent obtained for the current study included the information that previously collected data would be used and asked for the respective permission.

RESULTS

We enrolled a total of 93 households. Forty three households had an OPTIMA-improved stove installed, 48 belonged to control stove households and two belonged to a neighbouring community with Sembrando stoves. The total "N" for the analysis of each group varies due to measurement errors and equipment failure. In total we exclude 27 $PM_{2.5}$ kitchen measurements (14 controls and 13 intervention), 14 personal $PM_{2.5}$ measurements, (6 intervention and 8 control), 8 CO kitchen measurements (4 intervention and 4 control), and 7 CO personal measurements (4 intervention and 3 control).

The study groups were comparable with respect to their socio-demographic and kitchen characteristics (table B.1): 86% of the kitchens had four walls, and 43% had no windows in the kitchen area. Both groups used Eucalyptus sp. as the main source of firewood for cooking (table B.1). Community air pollution sampling showed that the average background outdoor- $PM_{2.5}$ level during the study period was 13µg/m^3 for $PM_{2.5}$ and 0.6ppm for CO.

Arithmetic mean and median kitchen- and personal exposure to air pollutants are presented in figure B.1 and table B.2. Overall, $PM_{2.5}$ mean values for OPTIMA-improved stoves (148µg/m^3 95%CI 88-208, N=30) in the kitchen environment were 22% lower compared to control stoves (189µg/m^3 95%CI 116-261, N=34), however the differences were not statistically significant. Similar values were observed for CO in the kitchen environment, with an overall difference of 19% (4.7ppm 95%CI 2.8-6.6ppm, N=39 vs 5.8ppm 95%CI 3.3-8.2ppm, N=44), which was not statistically significant.

At the personal level we did not observe a statistically significant difference in CO levels between users cooking with an OPTIMA-stove and in the control stove (35 open fires, 7 self-improved stoves, 6 supplied by NGO). However, $PM_{2.5}$ at personal levels were 20% lower among OPTIMA-stove users (table B.2) compared to the control group, but this difference was also not statistically significant.

Larger differences in pollution concentrations were observed within the OPTIMA-improved stove functionality levels (figure B.2 and table B.2). FL-I stoves had a 28% reduction of $PM_{2.5}$ (136µg/m^3 95%CI 54–216, N=20) and a 45% reduction of CO (3.2ppm 95%CI 1.5–4.9, N=25) in the kitchen environment measurements compared to control stoves, however significance was not reached (table B.2). Similarly, reductions for personal exposure to PM and CO were 43% and 17% respectively, with no statistical significance observed compared to control stoves.

$PM_{2.5}$ and CO concentrations were moderately correlated in simultaneous measurements in the kitchen environments (Spearman's rank correlation coefficient $r_s = 0.63$, $n = 61$, $p<0.0001$). A significant correlation between $PM_{2.5}$ and CO was also found when we stratified the data by study group (OPTIMA-improved stove: $r_s = 0.70$, $n=27$, $p<0.0001$; Control: $r_s = 0.65$, $n= 32$, $p<0.0001$). Likewise, statistically significant correlations were found between kitchen and personal $PM_{2.5}$ ($PM_{2.5}$: $r_s = 0.52$, $n=59$, $p<0.0001$) and kitchen and personal CO concentrations (CO: $r_s = 0.64$, $p<0.0001$, $n=84$).

A bivariate analysis (table B.3) showed that Acacia, a type of firewood (coefficient 1.0 95%CI 0.1; 1.9), was a significant determinant for predicting $PM_{2.5}$ concentrations in the kitchen environment. However, we did not observe any other predictor values for kitchen CO concentrations or for personal exposure levels of CO or $PM_{2.5}$.

The multivariable analysis did not reveal any significant predictors for any of the personal or kitchen measurements of CO and $PM_{2.5}$. The R values were low, indicating that the predicting factors could only explain a low proportion of the overall variance.

Findings from the participatory observational surveys (n=236) revealed a reported 90% (212/236) daily use of the OPTIMA-improved stove and an observed reduction in the lunch cooking times (50min versus 66min; $p<0.0001$) compared to those using other cooking stoves. Additionally, 96% of the mothers using the OPTIMA-improved stove (n=43) reported performing other activities while cooking, such as washing clothes, feeding the animals, cleaning, tending their children or visiting a neighbour. Finally, mothers' from the control households perceived stove-related smoke exposure more strongly as a nuisance than using the OPTIMA-improved stove (table B.4).

DISCUSSION

We investigated the effectiveness of a beneficiary-designed improved stove in reducing exposure to household air pollution within the framework of a community-randomised trial. About seven months after initial introduction of the OPTIMA-improved stoves, $PM_{2.5}$ and CO concentrations were measured as household air pollution and compared to control stove households which were comprised of three-stone open fire stoves, self-improved by household stoves or supplied by NGOs

Overall $PM_{2.5}$ and CO arithmetic mean values for the kitchen environment and personal exposure were lower in the improved stoves group, but the difference lacked statistical significance. Air quality improvements of the improved stove only became apparent after stratification by functionality levels. Fully functional improved stoves showed a 28% reduction of $PM_{2.5}$ and a 45% reduction of CO values in kitchen

environment measurements. Similarly, personal 48hr PM2.5 and CO exposures were reduced among OPTIMA-improved stove users to 43% and 17% respectively, however these results were not statistically significant. Additionally, faster cooking times and the possibility of performing other activities while cooking were much welcomed benefits derived from the improved stove confirming our findings from the exploratory pilot phase developing the parent trial (Hartinger et al. 2012).

Previous studies have yielded inconsistent evidence. In two randomized controlled field trials Smith and colleagues found in Guatemala up to 90% lower CO concentrations in the intervention group (Smith et al. 2011) whereas Burwen and Levine found no noteworthy reduction in rural Ghana (Burwen and Levine. 2012). In two uncontrolled before and after studies, reductions between one third and two third were observed (Dutta et al. 2007, Masera et al. 2007, Fitzgerald et al. 2012)

We captured ambient air CO and $PM_{2.5}$ levels as a background against which to compare changes in indoor levels in control and intervention households. The purpose for community air pollution measures was to report the general ambient air levels in San Marcos in order to observe any changes throughout the study period which may have impacted our results. No such trends during the study period were observed.

In order to understand the variation $PM_{2.5}$ and CO concentrations of our improved stove, we classified them post-hoc into FL-I and FL-II and observed a trend of increasing pollutant concentrations with declining stove performance due to structural damages from use. These included observed cracks or leaks of the general structure of the stove and around the potholders, the broken parts of the internal combustion chamber, or the chimney structure as well as the malfunction of the chimney valve. In categorizing the improved stoves the $PM_{2.5}$ and CO exposures at

kitchen and personal levels could be better predicted compared to using a less sensitive dichotomous categorisation of stove type (OPTIMA vs Control). This indicates the importance of presenting stove performance in terms of reduction of $PM_{2.5}$ and CO in relation to current stove conditions or levels of operation and maintenance needs.

Although the use of local materials and monthly training on the importance of repairs facilitated the self-maintenance of the stoves, OPTIMA-improved stoves were partly well-kept with post-hoc repairs revealing that 36% (91/250), of the stoves were not properly maintained. Further assessment of our compliance data revealed a gap between the mother's perception of appropriate maintenance and the actual repairs needed for the stove. The use of stove type to assign or determine exposure may be flawed given the varying HAP concentrations among households in our study which employed the same stove type. Clark et al. (2010) suggest the utility of stove levels may be more representative of HAP exposures and indoor levels. They note the importance of assessing the condition of the stoves rather than a mere comparison between traditional and improve stove type (Clark et al. 2010).

Improved stove adherence could also prove to be a challenge. Our reported high daily use was due to the perceived convenience gains (shorter cooking times, reduced wood consumption and limited supervision) and matched traditional cooking practices (Hartinger et al. 2012). In Central Mexico the Patsari wood cook stove reported a 50% adherence after 10 months (Romieu et al. 2009; Ruiz-Mercado et al. 2011). We expect adherence to OPTIMA-improved stove use to be higher given that after a median of 7.4 months (IQR: 6.6-8.1) OPTIMA-improved stove usage ranged at 90% although we cannot exclude dual use of open fire stoves during the study period.

In Bangladesh, of 105 biofuel-using households that had considered improved stoves, nine (8.5%) decided to use them, while the rest did not adopt improved stoves due to the large initial investment, inconvenience of the stoves or other reasons.(Dasgupta et al. 2006). Our results suggest that stove repair and maintenance are important in the success of any HAP mitigation program. Moreover, the metric of success needs to include the number of stoves that are adequately designed, as well as continually and exclusively used (Naeher, 2009, Smith et al. 2011, Clark et al. 2010, Dutta et al. 2007).

The type of wood used for cooking was associated with $PM_{2.5}$ concentrations in the kitchen. This finding reinforces the fact that, in the quest to reduce HAP, the use of clean energy (for example gas and electricity) needs to be conjunction with the design of cookstoves. Mothers using improved stoves reported spending less time cooking a lunch meal while performing unrelated cooking activities, inside and outside the kitchen environment. Subjects performing other tasks in or around the kitchen may experience exposures which outweigh potential exposure risk reductions due to shorter cooking times (Künzli, 2011).

Our study experienced some equipment failure of the $PM_{2.5}$ pumps which were occasionally not recording measurements for the full 48hr battery lifetime due to insufficient charging of batteries caused by power fluctuations at the field site. Further, the study had no means to validate the correct and uninterrupted wearing of the mother's equipment vest during the 48hr collection periods. Nonetheless, consistent with another study, we found moderate correlations between personal and kitchen $PM_{2.5}$ and CO measurements (Bruce et al. 2004). Finally, and since this study commenced after installing the OPTIMA-improved stoves, no data of baseline emissions of pollutants were available for before and after comparisons.

In line with others, this study showed that mother's identified well the nuisance of perceptible smoke and its immediate sequels like eye irritations (Romieu et al. 2009). Mothers perceived smoke reduction from the OPTIMA-improved stove which ranged along a 45% reduction of $PM_{2.5}$ particles of the personal exposure for well-maintained stoves after being in daily use for an average of seven months and a 17% reduction on CO. However, future impact evaluations of household air pollutions interventions should consider assessing both, outdoor and indoor determinants for air pollution risk exposures, since improved chimney stoves eliminate household air pollutants into the community environment, which may cause significant human exposure outdoors particularly in densely populated areas (Künzli, 2011).

CONCLUSION

Overall, the reductions of indoor air $PM_{2.5}$ and CO concentrations from the OPTIMA-improved stove were lower than expected. At the overall mean concentrations measured in both intervention groups ($PM_{2.5:}$ 148µg/m^3 and CO: 4.7ppm), the reduction in HAP is not expected to result in significant health improvements (Smith et al. 2011). In their analysis of outdoor air pollution, tobacco smoke, and active smoking studies Smith and Pillarisetti (2012) demonstrate that at about 150 ug/m3 $PM_{2.5}$ average annual exposure for example, the CVD risk slowly increases to the level experienced by active smokers.

In our study, kitchens with intervention stoves had overall mean PM2.5 concentrations of 148µg/m3 while control kitchens had a mean of 189µg/m3. Hence the risk is essentially the same at these two mean levels although the mean concentration measured in intervention kitchens is lower compared to control kitchens. Given the large global population which experiences exposures between second-hand smoke and active tobacco smoke, lower HAP levels must be achieved and sustained to yield greater public health benefits (Smith et al. 2011; Smith and Peel 2010).

REFERENCES

Armendáriz-Arnez, C. Edwards R.D., Johnson M, Rosas I.A., Espinosa F, Masera O.R. (2010) Indoor particle size distributions in homes with open fires and improved Patsari cook stoves, Atmospheric Environment, 44, 2881-2886.

Bodereau, PN (2011) Peruvian Highlands Fume-Free, Science, 344, 157.

Baumgartner, J.C. (2011) The effects of biomass smoke exposure on blood pressure among adult women and children in the United States and China: THE UNIVERSITY OF WISCONSIN-MADISON.

Bruce, N. McCracken J, Albalak R, Schei M.A., Smith K.R, 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, S26-33.

Burwen, J, Levine, DI. (2012) A rapid assessment randomized-controlled trial of improved cookstoves in rural Ghana. Energy for Sustainable Development, 16, 328-338.

Chengappa C, Edwards R, Bajpai R, Shields K.N., Smith K.R. (2007) Impact of improved cookstoves on indoor air quality in the Bundelkhand region in India. Energy for Sustainable Development, 11, 33-44.

Clark M.L., Reynolds S.J., Burch J.B, Conway S, Bachand A.M., Peel J.L. (2010) Indoor air pollution, cookstove quality, and housing characteristics in two Honduran communities. Environ. Res, 110, 12-18.

Cynthia A.A., Edwards R.D., Johnson M, Zuk M, Rojas L, Jiménez R.D., et al. (2008) Reduction in personal exposures to particulate matter and carbon monoxide as a result of the installation of a Patsari improved cook stove in Michoacan Mexico. Indoor Air, 18, 93-105.

Dasgupta S, Huq M, Khaliquzzaman M, Pandey K, Wheeler D. (2006) Indoor air quality for poor families: new evidence from Bangladesh. Indoor Air, 16, 426-444.

Dasgupta S, Wheeler D, Huq M, Khaliquzzaman M. (2009) Improving indoor air quality for poor families: a controlled experiment in Bangladesh. Indoor Air 19(1): 22-32.

Díaz E, Smith-Sivertsen T, Pope D, Lie R.T., Díaz A, McCracken J, et al. (2007) Eye discomfort, headache and back pain among Mayan Guatemalan women taking part in a randomized stove intervention trial. J Epidemiol Community Health, 61, 74-79.

Dix-Cooper, L. et al., 2011. Neurodevelopmental performance among school age children in rural Guatemala is associated with prenatal and postnatal exposure to carbon monoxide, a marker for exposure to woodsmoke. NeuroToxicology. 33, 246-54.

Dutta K, Shields K.N., Edwards R, Smith K.R. (2007) Impact of improved biomass cookstoves on indoor air quality near Pune, India. Energy for Sustainable

Development, 11, 19-32.

EPA (Environmental Protection Agency) (2005) Quality Assurance Guidance Document, Quality Management Plan for the National Air Toxic Trends Stations. EPA 454/R-02-006, Research Triangle Park, NC, Office of Air Quality Planning and Standards, United States Protection Agency.

Ezzati, M. and Kammen, D. (2001). Indoor air pollution from biomass combustion and acute respiratory infections in Kenya: an exposure-response study. Lancet, 358, 619-624.

Fitzgerald, C; Aguilar-Villalobos, M; Eppler, A.R: Dorner, S.C; Rathbun, S.L; and Naeher, L.P (2012) Testing the effectiveness of two improved cookstove interventions in the Santiago de Chuco Province of Peru. Sci. Total Environ, 420, 54 – 64.

Fullerton, D.G., Bruce, N. and Gordon, S.B. (2008) Indoor air pollution from biomass fuel smoke is a major health concern in the developing world. Trans. R. Soc. Trop. Med. Hyg, 102, 843-851.

GACC. The Global Alliance for Clean Cookstoves: Overview (2011). http://cleancookstoves.org/2011

Gil, A., Lanata C.L., Hartinger, S.M., Mäusezahl D, Padilla B, Ochoa T.J., Lozada M, Pineda I, Verastegui H. Faecal contamination of food, water, hands and kitchen utensils at household level in rural areas of Peru. J. Environ. Health. In-press.

Hartinger S.M, Lanata C.F., Hattendorf J, Gil A.I, Verastegui H, Ochoa T, and Mäusezahl D. (2011) A community randomised controlled trial evaluating a home-based environmental intervention package of improved stoves, solar water disinfection and kitchen sinks in rural Peru: Rationale, trial design and baseline findings. Contemp Clin Trials. 32, 864-873.

Hartinger S.M., Lanata C.F., Gil, A.I., Hattendorf J, Verastegui H, Mäusezahl, D. (2012) Combining interventions: improved stove, kitchen sinks and solar disinfection of drinking water and kitchen cloths to improve hygiene in rural Peru. FACTS Reports. Special Issue 6

INEI (Instituto Nacional de Estadística e Informática) (2007) http://www.inei.gob.pe.
Künzli, N. (2011). Commentary: Abating climate change and lung cancer! Int J Epidemiol, 40, 729-730.

Martin W.J., Glass R.I., Balbus J.M., Collins F.S. (2011) A Major Environmental Cause of Death. Science, 334, 180 -181.

Masera, O. Edwards R, Arnez C.A., Berrueta V, Johnson M, Bracho L.R., et al. (2007) Impact of Patsari improved cookstoves on indoor air quality in Michoacán, Mexico. Energy for Sustainable Development, 11, 45-56.

McCracken, J.P., Smith K.R., Díaz A, Mittleman M.A., Schwartz J. (2007) Chimney

stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women. Environ. Health Perspect, 115, 996-1001.

McCracken J, Smith KR, Stone P, Díaz A, Arana B, Schwartz J. (2011) Intervention to Lower Household Wood Smoke Exposure in Guatemala Reduces ST-Segment Depression on Electrocardiograms. Environ. Health Perspect, 119(11): 1562.

Naeher, L.P., Leaderer, B.P., and Smith, K.R. (2000). Particulate matter and carbon monoxide in highland Guatemala: indoor and outdoor levels from traditional and improved wood stoves and gas stoves. Indoor Air, 10, 200-205.

Naeher, L.P., Smith, K.R., Leader B.P., Mage D, Grajeda R. (2000) Indoor and outdoor PM2.5 and CO in high- and low-density Guatemalan villages. J Expo Anal Environ Epidemiol, 10, 544-551.

Naeher, L.P., Smith K.R., Leaderer B.P., Neufeld L, Mage D.T., (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, 575-581.

Naeher, L.P. (2009). Editorial: Biomass-fueled intervention stoves in the developing world: potential and challenges. Am. J Resp. Crit. Care, 180, 586-587.

Naeher, L.P., Brauer M, Lipsett M, Zelikoff J.T., Simpson C.D., Koenig J.Q., et al. (2007) Woodsmoke health effects: a review. Inhal Toxicol, 19, 67-106.

Po, J.Y.T., Fitzgerald, J.M., and Carlsten, C. (2011). Respiratory disease associated with solid biomass fuel exposure in rural women and children: systematic review and meta-analysis. Thorax, 66, 232-239.

Rehfuess, E., Mehta, S. and Prüss-Ustün, A. (2006) Assessing household solid fuel use: multiple implications for the Millennium Development Goals. Environ. Health Perspect, 114, 373-378.

Romieu, I., Riojas-Rodríguez H., Marrón-Mares A.T., Schilmann A., Perez-Padilla R., Masera O. (2009) Improved biomass stove intervention in rural Mexico: impact on the respiratory health of women. Am J Respir. Crit. Care Med, 180, 649-656.

Ruiz-Mercado, I., Mesera O., Zamora, H., Smith K.R., (2011) Adoption and sustained use of improved cookstoves. Energy Policy, 39, 7557-7566.

Saha, A., Kulkarni P.K., Shah A., Patel M., Saiyed H.N. (2005) Ocular morbidity and fuel use: an experience from India. Occup Environ Med, 62, 66-69.

Siddiqui, A.R., Gold E.B., Yang X., Lee K., Brown K.H., Bhutta Z.A. (2008) Prenatal exposure to wood fuel smoke and low birth weight. Environ Health Perspect, 116, 543-549.

Smith, K.R., Samet, J.M., Romieu, I., Bruce, N. (2000). Indoor air pollution in developing countries and acute lower respiratory infections in children. Thorax, 55(6),

pp.518-532.

Smith, K.R. and Mehta, S. (2003). The burden of disease from indoor air pollution in developing countries: comparison of estimates. International J Hyg Environ Health, 206, 279-289.

Smith, K.R., Mehta, S. and Maeusezahl-Feuz, M., (2004) Indoor air pollution from household use of salid fuels. In: Ezzati M, Lopez A, Roders A et al. (2004) Comparative Quantification of Health Risks, Global and Regional Burden of Disease Attributable to Selected Major Risk Factors, Geneva, World Health Organization, 1435-1494
http://www.who.int/publications/cra/chapters/volume2/1435-1494.pdf

Smith, K.R., Peel, J, L. Mind the Gap. (2010). Environ Health Perspect, 118, 1643–1645

Smith, K. R. and A. Pillarisetti. (2012) A Short History of Woodsmoke and Implications for Chile, Estudios Públicos, 126: 163-79.

Smith, K.R., McCracken J.P., Weber M.W., Hubbard A., Jenny A., Thompson L.M., Balmes J., Diaz A., Arana B., Bruce N. (2011). Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomized controlled trial. Lancet, 378, 1717–26.

Tielsch, J.M., Katz, J., Thulasiraj, R.D., Coles, C.L., Sheeladevi, S., Yanik, E.L., Rahmathullah, L. (2009) Exposure to indoor biomass fuel and tobacco smoke and risk of adverse reproductive outcomes, mortality, respiratory morbidity and growth among newborn infants in south India. Int. J. Epidemiol, 38,1351-1363.

USEPA (United States Environmental Protection Agency) (2005) National Ambient Air Quality Standards (NAAQS). http://www.epa.gov/air/criteria.html.

Table B.1: Basic socio-demographic and kitchen characteristics from the study participants of the San Marcos province. Data are means (SD) or numbers (%).

	Optima-Improved Stove (N=43)	**Control stoves (N=48)**
Socio demographic characteristics		
Number of family members [¶]	4.7 (1.2)	4.7 (1.3)
Housewife as main activity of mother	39 (91%)	45 (94 %)
Farming as main activity of the family head	34 (79%)	40 (83 %)
Family members that smoke cigarettes	4 (9%)	10 (21 %)
Kitchen characteristics		
Kitchen volume (m^3) [†]	29 (18.6)	37.2 (25.7)
Type of wood used for cooking [§]		
Eucalipto (*Eucalyptus sp*.)	18 (42 %)	21(45 %)
Acacia *(Acacia macrantha)*	8 (19 %)	9 (19 %)
Chamana (type of wood)	3 (7 %)	6 (13 %)
Other	14 (33%)	11 (23%)
Kitchen windows [§]		
Completely closed - No windows	20 (47 %)	20 (43 %)
One window	20 (47 %)	20 (43 %)
More than one window or door opening	3 (7 %)	7 (15 %)
Number of kitchen walls [§]		
Four walls	40 (93 %)	40 (85 %)

[¶] N = 42 for traditional stove arm.
[†] N = 42 for Optima stove arm.
[§] N = 47 for traditional stove arm

Table B.2: Air quality measured in the kitchen and at personal level in relation to stove type and functionality levels in rural Peru

Sampling Location	Measurement	Stove Type	N	Mean	95% CI	Median	% difference	p-values
Kitchen environment	PM 2.5 ($\mu g/m^3$)	**Control Stoves ***	**34**	**189**	**116 – 261**	**116**	**reference**	
		Open Fire	24	211	116 - 305	139		
		Self-improved by household	6	117	3.7 – 230	93		
		NGO	4	166	0 – 559	50		
		OPTIMA Improved Stove (*)	**30**	**148**	**88 – 208**	**102**	**22%**	**0.337**
		FL-I	20	136	54 – 217	77	28%	0.465
		FL-II	10	173	72 – 273	123	8%	1
	CO (ppm)	**Control Stoves**	**44**	**5.8**	**3.3 - 8-2**	**2.4**	**reference**	
		Open Fire	32	5.2	2.8 – 7.5	2.4		
		Self-improved by household	7	7.2	0 - 17.8	3.1		
		NGO	5	7.5	0 - 23.1	2		
		OPTIMA Improved Stove	**39**	**4.7**	**2.8 - 6.6**	**2.9**	**19%**	**0.955**
		FL-I	25	3.2	1.5 – 4.9	2.1	45%	0.951
		FL-II	14	7.5	3.2 - 11.7	5.5	-28%	0.352
Personal Exposure	PM 2.5 ($\mu g/m^3$)	**Control Stoves**	**40**	**129**	**82 – 176**	**94**	**reference**	
		Open Fire	28	145	90 -200	116		
		Self-improved by household	7	135	0 – 320	59		
		NGO	5	35	0 – 72	40		
		OPTIMA Improved Stove	**37**	**104**	**64 -144**	**55**	**20%**	**0.163**
		FL-I	23	74	38 – 109	40	43%	0.12
		FL-II	14	154	65 - 244	76	-19%	1
	CO (ppm)	**Control Stoves**	**45**	**1.4**	**0.8 - 2.0**	**0.6**	**reference**	
		Open Fire	32	1.5	0.8 - 2.1	0.7		
		Self-improved by household	7	1.8	0.0 – 5.0	0.5		
		NGO	6	0.5	0.1 – 0.8	0.4		
		OPTIMA Improved Stove	**39**	**1.5**	**1 – 2**	**1**	**-6%**	**0.851**
		FL-I	25	1.2	0.7 – 1.7	0.8	17%	1
		FL-II	14	1.9	0.9 -3.2	1.2	-39%	0.401

** Control Stoves: includes all control households (open fires, Self-improved by household and NGO). NGO: Stoves build by non-governmental organization*

(*)OPTIMA-improved stoves: includes all OPTIMA-improved stoves functionality levels (FL-I and FL-II). FL-I: stoves in good running conditions (plastered stove and no visible leaks when in use. FL-II: stove in need of repairs (re-plastering, filling cracks)
Mean refers to arithmetic mean

Table B.3: Bivariate and multivariable regression analysis of covariates for 48hr CO and $PM_{2.5}$ levels of kitchen and personal exposure.

	Carbon monoxide (CO)			Particulate matter ($PM_{2.5}$)		
		Bivariate	Multivariable[a]		Bivariate	Multivariable[b]
Variable	**n**	**Coef (95%CI)**	**Coef (95%CI)**	**n**	**Coef (95%CI)**	**Coef (95%CI)**
Kitchen						
Stove type	83			64		
Control (reference)						
FL-I		-0.3(-0.9; 0.3)	-0.3 (-0.9; 0.2)		-0.7 (-1.4; 0.1)	-0.7 (-1.4; 0.1)
FL-II		0.6 (-0.1; 1.3)	0.6 (-0.1; 1.3)		-0.0 (-1.1; 1.1)	-0.4 (-1.4; 0.6)
Kitchen volume ($100m^3$)	79	-0.2 (-1.4; 1.0)	-	61	-1.6 (-3.2; -0.0)	-1.5 (-3.2; 0.2)
Wood used for cooking	85			65		
Eucalipto (reference)						
Acacia		-0.4 (-1.1; 0.4)			**1.0 (0.1; 1.9)***	0.6 (-0.5; 1.7)
Other wood types		-0.1 (-0.7; 0.4)	-		0.4 (-0.4; 1.1)	0.2 (-0.6; 1.0)
Kitchen windows	82			63		
No windows (reference)						
One or more windows		-0.3 (-0.9; 0.2)	-0.5 (-1.0; 0.1)		-0.2 (-0.9; 0.5)	-
Number of kitchen walls	82			63		
Four walls (reference)						
Less than four walls		-0.0 (-0.8; 0.8)	-		0.3 (-0.8; 1.4)	-
Personal exposure						
Stove type	85			77		
Control (reference)						
FL-I		-0.2 (-0.8; 0.3)	-0.2 (-0.8; 0.3)		-0.6 (-1.2; 0.1)	-0.4 (-1.1; 0.3)
FL-II		0.5 (-0.2; 1.2)	0.6 (-0.1; 1.2)		0.3 (-0.6; 1.1)	0.4 (-0.5; 1.2)
Kitchen volume ($100m^3$)	79	0.3 (-0.9; 1.5)	-	74	-0.4 (-1.9; 1.1)	-
Time spent cooking (hrs)	81	-0.1 (-0.6; 0.4)		75	-0.4 (-1.1; 0.2)	-0.4 (-1.0; 0.3)
Does the mother perform other activities while cooking? (no = reference)	85	0.1 (-0.6; 0.7)		78	-0.1 (-0.9; 0.8)	-
Wood used for cooking	85			78		
Eucalipto (reference)						
Acacia		-0.5 (-1.2; 0.2)	-0.6 (-1.2; 0.08)		-0.1 (-1.0; 0.8)	-
Other wood types		-0.3 (-0.8; 0.3)	-0.4 (-0.9; 0.2)		0.1 (-0.6; 0.8)	-
Kitchen windows	82			76		
No windows (reference)						
One or more windows		0.1 (-0.4; 0.6)	-		0.3 (-0.4; 0.9)	-
Number of kitchen walls	82			74		
Four walls (reference)						
Less than four walls		-0.1 (-0.9; 0.7)	-		0.3 (-0.7; 1.3)	-

[a] Kitchen: n=82, R^2=0.09; Personal: n=82, R^2=0.09

[b] Kitchen: n=61, R^2=0.14; Personal: n=75, R^2=0.06

* Coefficient is statistically significant with confidence interval not including zero.

- refers to variables not included in the multivariate models. Bivariate regression analysis refers to linear models which include the outcome variable and only one predictor variable. Multivariable regression analysis refers to linear models which include the outcome variable and all predictor variables listed in the table.

Table B.4: Mothers' cooking behaviour and smoke exposure perceptions of 93 study participants in rural Peru. Data are means (SD) or numbers (%).

	Optima Improved Stove	**Control Stoves**
	N=43	**N=47**
Mothers' behaviour and perceptions		
Mother performs other activities while cooking[¶]	38 (96 %)	21 (56 %)
Hours the stove was lit[§]	9.1 (4.0)	9.2 (3.8)
Mother's self-report of minutes spent cooking per day	187 (75)	201 (84)
Perceived exposure to smoke from motor vehicles		
Low	29 (67 %)	34 (72 %)
Medium	2 (5 %)	5 (11 %)
High	2 (5 %)	6 (13 %)
DNK*	10 (23%)	2 (4%)
Perceived exposure to smoke from kitchen stoves		
Low	26 (60 %)	11 (24 %)
Medium	7 (16 %)	15 (32 %)
High	5 (12 %)	19 (40 %)
DNK*	5 (12 %)	2 (4 %)

*DNK: Does not know
[¶] N = 38 for Optima stove and 37 for traditional stove arm.
[§] N = 38 for Optima stove arm and 45 for traditional stove

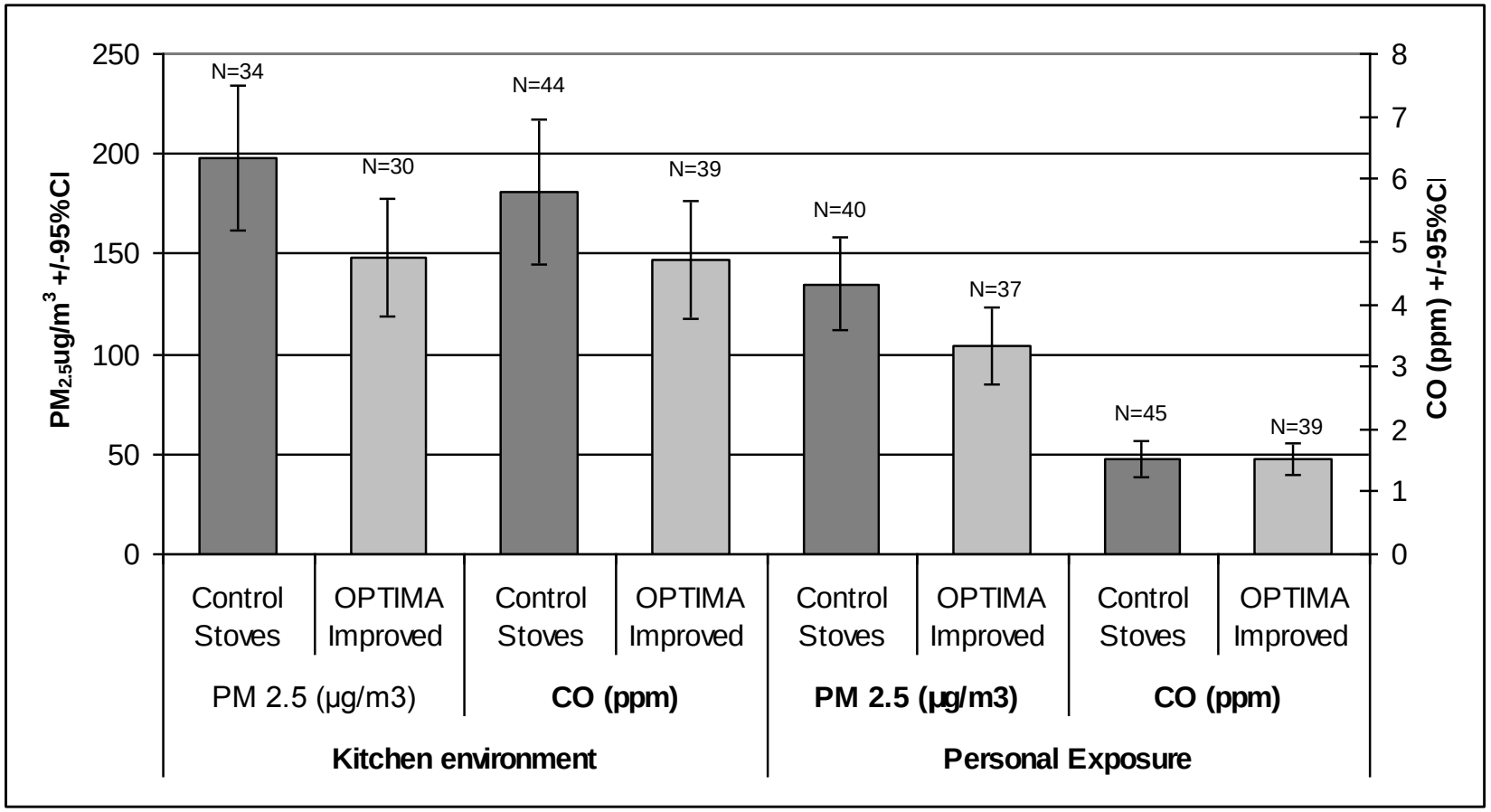


Figure B.1: 48 hr $PM_{2.5}$ and CO mean concentrations between traditional and OPTIMA-improved stove for kitchen environment and personal exposure

Control Stoves: include all control households, (open fires, Self-improved by household and NGO).
OPTIMA Improved: includes all OPTIMA-improved stoves functionality levels (FL-I and FL-II)

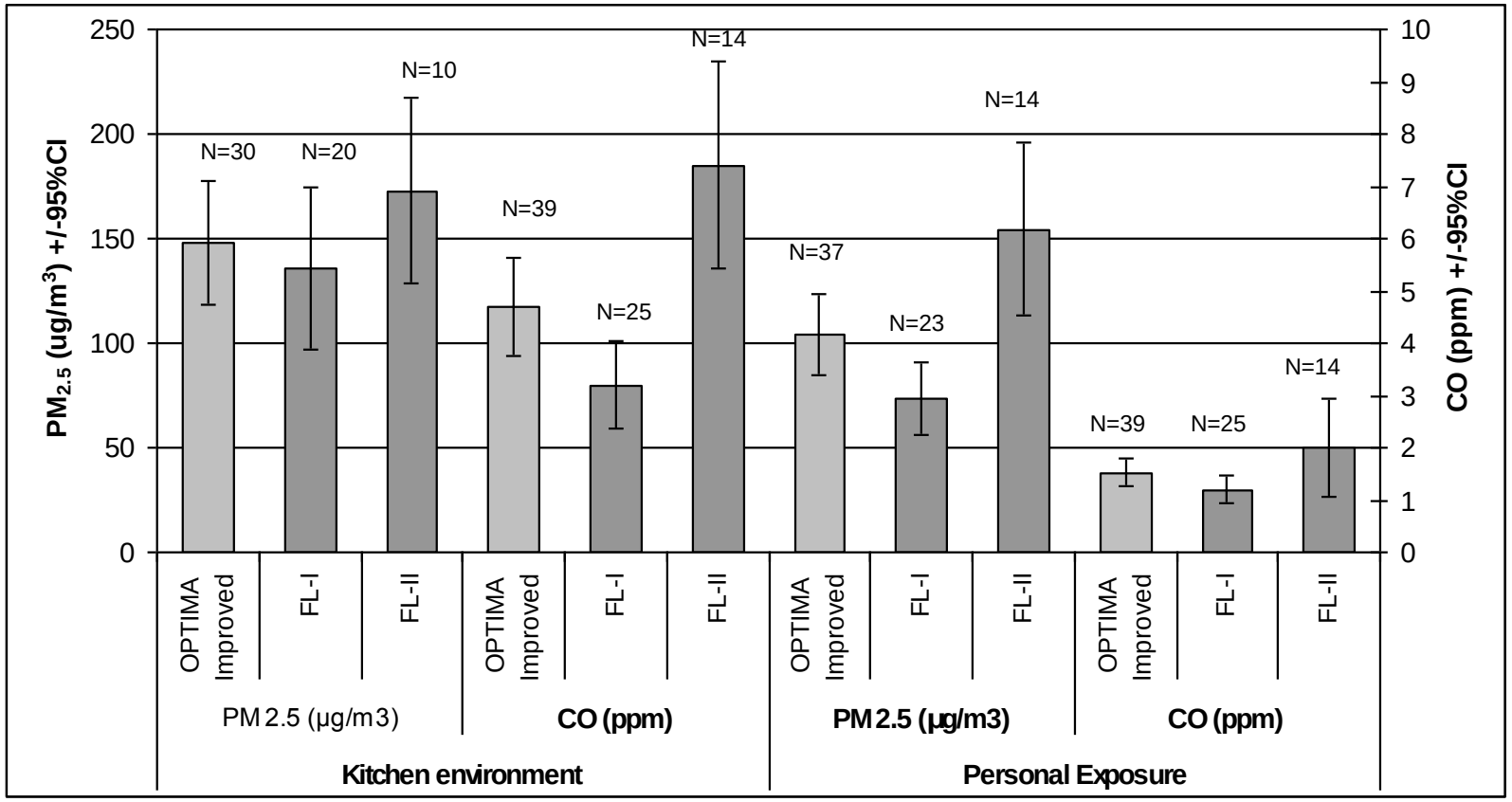


Figure B.2: 48hr PM 2.5 and CO concentration in OPTIMA-improved households separated into functionality levels.

OPTIMA-improved: includes all OPTIMA-improved stoves functionality levels (FL-I and FL-II)
FL-I: stoves in good running conditions (plastered stove and no visible leaks when in use)
FL-II: stove in need of repairs (re-plastering, filling cracks)