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Household fuel use and biomarkers of inflammation and respiratory illness among rural South African Women

Ankita Misra[a], Matthew P. Longnecker[b], Kathie L. Dionisio[c], Riana M.S. Bornman[d,e,f], Gregory S. Travlos[e], Sukhdev Brar[e], Kristina W. Whitworth[a,g,*]

[a] Department of Epidemiology, Human Genetics and Environmental Sciences, UTHealth School of Public Health in San Antonio, San Antonio, TX, USA
[b] Epidemiology Branch, National Institute for Environmental Health Sciences, National Institutes of Health, DHHS, Research Triangle Park, NC, USA
[c] National Exposure Research Laboratory, Environmental Protection Agency, Research Triangle Park, NC, USA
[d] Department of Urology, University of Pretoria, Pretoria, South Africa
[e] Cellular and Molecular Pathology Branch, National Institute of Environmental Health Sciences, NIH, DHHS, Research Triangle Park, NC, USA
[f] The University of Pretoria Centre for Sustainable Malaria Control, University of Pretoria, Pretoria, South Africa
[g] Southwest Center for Occupational and Environmental Health, UTHealth School of Public Health, Houston, TX, USA

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ABSTRACT

Though literature suggests a positive association between use of biomass fuel for cooking and inflammation, few studies among women in rural South Africa exist. We included 415 women from the South African Study of Women and Babies (SOWB), recruited from 2010 to 2011. We obtained demographics, general medical history and usual source of cooking fuel (wood, electricity) via baseline questionnaire. A nurse obtained height, weight, blood pressure, and blood samples. We measured plasma concentrations of a suite of inflammatory markers (e.g., interleukins, tumor necrosis factor-α, C-reactive protein). We assessed associations between cooking fuel and biomarkers of inflammation and respiratory symptoms/illness using crude and adjusted linear and logistic regression models. We found little evidence of an association between fuel-use and biomarkers of inflammation, pre-hypertension/hypertension, or respiratory illnesses. Though imprecise, we found 41% (95% confidence interval (CI) = 0.72–2.77) higher odds of self-reported wheezing/chest tightness among wood-users compared with electricity-users. Though studies among other populations report positive findings between biomass fuel use and inflammation, it is possible that women in the present study experience lower exposures to household air pollution given the cleaner burning nature of wood compared with other biomass fuels (e.g., coal, dung).

1. Introduction

Much of the world's population uses biomass fuel (i.e., burned plant and animal material such as wood, charcoal, agricultural residue, or animal dung) as a primary source of energy for heating and cooking, particularly in low and middle income countries. It has been estimated that more than 60% of households in Africa rely on solid fuels for cooking (Bonjour et al., 2013; Rehfuess et al., 2006). According to the World Health Organization (WHO), fuels such as kerosene are the cleanest burning while animal dung, twigs and grass are among the most polluting; wood falls in the middle in terms of efficiency and polluting potential (World Health Organization, 2006). The use of simple open stoves or fires to burn biomass fuels coupled with incomplete combustion can result in substantial emissions of pollutants including carbon monoxide, particulate matter, and volatile organic compounds (e.g., benzene, benzo(a)pyrene) (Bruce et al., 2000; Elledge, 2012; Smith et al., 2004). Poor ventilation may also contribute to high concentrations of indoor pollution in households, which rely on biomass fuel for cooking and heating (Naeher et al., 2007; Household air pollution and health, 2016). Women and children may be particularly affected by household air pollution from burning biomass fuels due to the increased time spent in the cooking area by these groups (Smith et al., 2004).

The use of solid fuel accounts for an estimated 4.6% of disability adjusted life-years lost globally (Lim et al., 2013), primarily due to respiratory tract infections in children less than five years of age and chronic obstructive pulmonary disease (COPD) in adult women (Rehfuess et al., 2006). The posited mechanism through which exposure to biomass-related household air pollution affects health is through inflammation and oxidative stress (Naeher et al., 2007). The pro-inflammatory effects of woodsmoke exposure have been demonstrated in vitro (Hawley and Volckens, 2013) and experimental studies

* Correspondence to: UTHealth School of Public Health in San Antonio, 7411 John Smith Drive Suite 1100, San Antonio 78229, TX, USA.
E-mail address: Kristina.W.Whitworth@uth.tmc.edu (K.W. Whitworth).

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have shown short-term controlled inhalation exposure of woodsmoke can induce pulmonary and systemic inflammation (Bonlokke et al., 2014; Ghio et al., 2012; Jensen et al., 2014).

Epidemiologic studies indicate that individuals exposed to biomass smoke have increased risk of respiratory symptoms and illnesses (Kurmi et al., 2013, 2010; Kamal et al., 2016; Regalado et al., 2006), including evidence of respiratory tract infections among individuals exposed to biomass smoke in Kenya (Ezzati and Kammen, 2001) and among South African children living in homes where polluting fuel sources are used for heating and cooking (Barnes et al., 2009). Studies also find more frequent respiratory symptoms or illness among women using traditional stoves compared to women using improved cookstoves (Clark et al., 2009; Romieu et al., 2009). Furthermore, studies in Central America and South Asia suggest cooking with biomass fuels is associated with increased levels of: biomarkers of endothelial inflammation (i.e., intercellular adhesion molecule-1 (ICAM-1)) and vascular cell adhesion molecule-1 (VCAM-1) (Caravedo et al., 2015) and pro-inflammatory biomarkers (i.e., interleukin-6 (IL-6), interleukin-8 (IL-8), tumor necrosis factor-alpha (TNF-α), and C reactive protein (CRP)) (Kamal et al., 2016; Dutta et al., 2012; Banerjee et al., 2012).

Though research from multiple developing countries provides indication of a positive association between cooking with biomass fuels and biomarkers of inflammation, less is known about this purported association among South African women, where just over 40% of the population cooks primarily with wood, generally in a separate kitchen building (Statistics South Africa). The variances in cultural practices around cooking when compared to the Indian and Central American populations could be the cause for the possibly lower levels of exposure in the African women. Therefore, the aim of the present study was to investigate the relation between cooking with wood and biomarkers of inflammation and respiratory symptoms and illnesses among reproductive-aged women living in rural South Africa.

2. Methods

Data for this analysis are from the South African Study of Women and Babies (SOWB), a study of DDT (dichlorodiphenyltrichloroethylene) exposure and clinically-recognized pregnancy loss (Whitworth et al., 2014). From 2010 to 2011, the SOWB study enrolled women from eight villages in the Thulamela Municipality of the Vhembe district in the Limpopo Province of South Africa. Eligibility criteria for SOWB included: aged 20–30 years, not currently using contraception, regular menstrual periods (unless breastfeeding), negative spot pregnancy test, no history of infertility and no medical or other condition which would prevent pregnancy. SOWB was approved by the institutional review boards of the University of Pretoria, South Africa and the National Institute of Environmental Health Sciences (NIEHS).

Consenting, eligible women completed an interviewer-administered questionnaire on demographics, lifestyle, reproductive and medical history, and usual cooking practices. At baseline, women also underwent a short physical exam where anthropometric measurements were obtained in triplicate, including height and weight, from which body mass index (BMI) was calculated. Participants' blood pressure was measured at baseline in triplicate and lastly, the women provided a blood sample. Of the 442 women initially enrolled in the SOWB study, 15 were later found ineligible due to age (n = 3) or not living in one of the eight study villages (n = 12), leaving 427 eligible women. For the present analysis, we excluded one woman who did not have a blood specimen and six women who did not have information on cooking fuel use. Further, five women who reported current smoking were excluded. This left 415 women for the present analysis.

The main exposure metric explored in this study was women's self-reported usual cooking fuel (wood vs. electricity). Additional information on location (indoors versus outdoors) of cooking was also assessed among women who reported using wood for cooking. These women were asked: "Most of the time do you cook inside or outside?" Based on responses to this question, a secondary exposure variable was created with the following categories: electricity users, wood users-mostly outdoors, and wood users-mostly indoors.

The primary dependent variables of interest were biomarkers of inflammation. Plasma samples were aliquoted and stored at − 80 degrees Centigrade until analyzed. The samples were then assayed for: Interleukin-1 β (IL-1β), Interleukin-6 (IL-6), Interleukin 8 (IL-6) and Tumor Necrosis factor-α (TNF-α) using the Human Pro-Inflammatory II 4-Plex Ultra-Sensitive Kit; and for C-Reactive Protein (CRP), Serum amyloid A (SAA), Vascular cell adhesion molecule-1 (VCAM-1) and Intercellular adhesion molecule 1 (ICAM-1) using the Human Vascular Injury Panel II kit, both from MSD (Meso Scale Discovery, Gaithersburg, Maryland, USA) according to manufacturer's protocols. Briefly, a 96-well plate that had been pre-coated with capture antibodies on spatially distinct spots was blocked with blocking solution for one hour at room temperature with constant shaking at 700 rpm. After washing three times with phosphate buffered saline-tween (PBS-T) buffer, samples and standards were added to the appropriate wells and the plate was incubated for two hours at room temperature with constant shaking. The plate was washed again as mentioned above, detection antibody was added to each well, and the plate was again incubated at room temperature with constant shaking (two hours for the Pro-Inflammatory panel plate and one hour for the Human Vascular Injury Panel plate). After washing the plate three times with (PBS-T), 150 µl of 1x Read Buffer was added to each well and the plate was immediately analyzed on the Sector Imager 2400 System (MSD). The instrument measures intensity of emitted light to afford a quantitative measure of Pro-Inflammatory or Vascular Injury Panel analytes in the sample. CRP, SAA, ICAM-1 and VCAM-1 were reported in units of mg/L, and IL-1β, IL-6, IL-8 and TNF-α were reported in units of pg/ml. No measurements were less than the limit of detection and values were log-transformed prior to analysis.

In addition to the inflammatory biomarkers described above, we were also interested in the association between cooking fuel and respiratory symptoms and illnesses, and blood pressure. Respiratory symptoms included breathlessness (i.e., "When you work hard, do you feel you have less breath compared to other people your age"; yes/no) and wheezing/chest tightness (i.e., "During the last year have you had wheezing or tightness of your chest", yes/no). Self-report of tuberculosis, pneumonia, and asthma at age 16 or older were grouped together to represent respiratory illness. For this analysis, we collapsed the mean of the systolic blood pressure (SBP) and diastolic blood pressure (DBP) measurements into a dichotomous variable: normal blood pressure (i.e., SBP < 120 mm Hg and DBP < 80 mm Hg) versus pre-hypertension/hypertension (SBP > = 120 mm Hg or DBP > = 80 mm Hg). We used guidelines reported in the Seventh Report of the Joint National Committee to additionally include women with pre-hypertension (Chobanian et al., 2003) as only a small number of women in this study (n = 15, 3.8%) had clinical hypertension according to the standard cutoff (i.e., SBP ≥ 140 mm Hg or DBP ≥ 80 mm Hg).

We conducted separate linear regression models for each inflammatory marker. Age (years) was included in the regression models as an *a priori* covariate. Other variables we explored in the models included: body mass index (BMI; kg/m^2), years of education (< 11, 11, 12, > 12), gravidity (number of pregnancies; 0, 1, > 2), regular coffee consumption (i.e., "at least once per week for six months or longer"; yes/no), passive smoking (i.e. "in the past 12 months, has anyone smoked at least one cigarette/day for six months or more near you?"; yes/no), consumption of alcohol (ever/never), primary source of drinking water (public tap vs. water piped to yard/home), and any medication taken in the previous 24 h (yes/no). To arrive at a common set of adjustment variables, we included in all models those covariates that were statistically significantly ($p < 0.20$) associated with a minimum of three of the inflammatory biomarkers. Based on this criterion, in addition to age, we included gravidity, caffeine consumption,