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Exposure to polycyclic aromatic hydrocarbons and volatile organic compounds among recently pregnant rural Guatemalan women cooking and heating with solid fuels

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ABSTRACT

Background: Household air pollution is a major contributor to death and disability worldwide. Over 95% of rural Guatemalan households use woodstoves for cooking or heating. Woodsmoke contains carcinogenic or fetotoxic polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs). Increased PAHs and VOCs have been shown to increase levels of oxidative stress.
Objective: We examined PAH and VOC exposures among recently pregnant rural Guatemalan women exposed to woodsmoke and compared exposures to levels seen occupationally or among smokers.
Methods: Urine was collected from 23 women who were 3 months post-partum three times over 72 h: morning (fasting), after lunch, and following dinner or use of wood-fired traditional sauna baths (samples = 68). Creatinine-adjusted urinary concentrations of metabolites of four PAHs and eight VOCs were analyzed by liquid chromatography–mass spectrometry. Creatinine-adjusted urinary biomarkers of oxidative stress, 8-isoprostane and 8-OHdG, were analyzed using enzyme-linked immunosorbent assays (ELISA). Long-term (pregnancy through 3 months prenatal) exposure to particulate matter and airborne PAHs were measured.
Results: Women using wood-fueled chimney stoves are exposed to high levels of particulate matter (median 48 h $PM_{2.5}$ 105.7 $\mu g/m^3$; inter-quartile range (IQR): 77.6–130.4). Urinary PAH and VOC metabolites were significantly associated with woodsmoke exposures: *2-naphthol* (median (IQR) in ng/mg creatinine: 295.9 (74.4–430.9) after sauna versus 23.9 (17.1–49.5) fasting; and *acrolein*: 571.7 (429.3–1040.7) after sauna versus 268.0 (178.3–398.6) fasting. Urinary PAH (total PAH: $\rho = 0.89$, $p < 0.001$) and VOC metabolites of *benzene* ($\rho = 0.80$, $p < 0.001$) and *acrylonitrile* ($\rho = 0.59$, $p < 0.05$) were strongly correlated with long-term exposure to particulate matter. However urinary biomarkers of oxidative stress were not correlated with particulate matter ($\rho = 0.01$ to 0.05, $p > 0.85$) or PAH and VOC biomarkers ($\rho = -0.20$ to 0.38, $p > 0.07$). Urinary metabolite concentrations were significantly greater than those of heavy smokers (mean cigarettes/day = 18) across all PAHs. In 15 (65%) women, maximum *1-hydroxypyrene* concentrations exceeded the occupational exposure limit of coke-oven workers.
Conclusions: The high concentrations of urinary PAH and VOC metabolites among recently pregnant women is alarming given the detrimental fetal and neonatal effects of prenatal PAH exposure. As most women used chimney woodstoves, cleaner fuels are critically needed to reduce smoke exposure.

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1. Introduction

Household air pollution (HAP) from solid fuels is a significant risk factor for death and disability worldwide. In 2013, it was the seventh leading cause of Disability Adjusted Life years (DALYs) and remains one of the leading causes of acute lower respiratory infections, chronic obstructive pulmonary disease, lung cancer, cerebrovascular and ischemic heart disease (Collaborators et al., 2015). The disease burden is highest among the very young (under 5 years old) and women (IHME, 2015), and would be even higher if evidence of the effect of HAP on preterm birth and low birthweight were included in global estimates (Patelarou and Kelly, 2014). Reducing HAP exposures is an important mission of a recent funding opportunity supported by the National Institutes of Health, the Gates Foundation and the Global Alliance for Clean Cookstoves (NIH, 2015).

In Guatemala, 64% of all households and 95% of rural households use wood fuel for cooking (WHO, 2013). HAP ranks as the fifth leading cause of death and is responsible for 4% of all DALYs for children under 5 (IHME, 2015). Annually, over 5000 deaths are attributable to HAP with lower respiratory infections and ischemic heart disease causing the most deaths (McCracken et al., 2015). It will continue to contribute to the epidemiologic transition within Guatemala as the predominant health burden shifts from communicable diseases, such as lower respiratory infections, to non-communicable diseases, such as ischemic heart disease, to which HAP is a major contributing factor.

Polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs), two groups of chemicals created during the incomplete combustion of organic substances, are systemically absorbed. Many are known carcinogens, causes of pulmonary and cardiovascular disease, immune impairment and/or adverse birth outcomes. Multiple PAHs and VOCs, such as benzene, 1,3-butadiene, and acrylamide, are classified as carcinogenic (IARC, 2015). In addition, adult exposure to PAHs and VOCs is associated with cardiovascular disease (Alshaarawy et al., 2016; Haussmann, 2012). Prenatal exposure to ambient levels of PAHs is associated with adverse birth outcomes such as neural tube defects (Ren et al., 2011), small for gestational age and preterm birth (Choi et al., 2008; Padula et al., 2014). Similarly, residential exposures to VOCs have been shown to be associated with small for gestational age in newborns (Sorensen et al., 2010).

A common source of PAHs is dietary intake (WHO and IARC, 2010). In addition, elevated urinary concentrations of PAH and VOC metabolites have been found in the urine of cigarette smokers (Alwis et al., 2012; Benowitz et al., 2015), those exposed to secondhand smoke (St Helen et al., 2014; Suwan-ampai et al., 2009), and from occupational exposures, such as coal processing or aluminum production (Jongeneelen, 2001). Smoke from burning solid fuels typically contains high levels of PAHs (Titcombe and Simcik, 2011), VOCs (Vanker et al., 2015), and airborne fine particulate matter ($PM_{2.5}$) (Li et al., 2011; Titcombe and Simcik, 2011) and exposure to wood smoke from cooking is associated with high urinary levels of PAH metabolites (Pruneda-Alvarez et al., 2012). These reported levels are higher than those found in studies within high-income countries (Alshaarawy et al., 2016) and many are higher than the occupational exposure limit set by Jongeneelen (Jongeneelen, 2001).

The biological mechanisms by which PAH and VOC metabolites exert effects on health outcomes are not well established but they have been shown to induce oxidative stress (Li et al., 2015; Wang et al., 2015). Two urinary markers of oxidative stress are 8-isoprostane, a measure of lipid peroxidation (Milne et al., 2005), and 8-hydroxy-2′-deoxyguanosine (8-OHdG), a measure of DNA oxidation (Evans et al., 2010; Poulsen et al., 2014). Levels of both have been found to be elevated in the urine of those exposed to ambient air pollution (Svecova et al., 2009), household air pollution (Commodore et al., 2013) and welding fumes (Nuernberg et al., 2008).

Previous studies have shown that women in Guatemala are exposed to high concentrations of $PM_{2.5}$ and carbon monoxide (CO), two major constituents produced during the incomplete combustion of solid fuel (Smith et al., 2010; Smith et al., 2011; Thompson et al., 2011a). PAHs and VOCs are also significant by-products of incomplete combustion. Thus, this study aimed to measure the urinary concentrations of PAHs, VOCs and oxidative stress metabolites in recently pregnant Guatemalan women, to compare these concentrations to long-term personal exposures to airborne $PM_{2.5}$ and PAHs, and to compare PAH and VOC urinary metabolite concentrations in this study to levels found with other known high exposures, namely cigarette smoking or the industrial processing of coal products.

2. Methods

2.1. Study population and sampling strategy

This study was nested within a larger cohort study, the NACER (Neurodevelopment and anthropometric growth of infants exposed to household air pollution in rural Guatemala) study, which explored the effect of household woodsmoke on birth outcomes and child development among rural Mam-speaking Mayan or Spanish-speaking ladino women in the Western Highlands of Guatemala between April 2012 and December 2013. Study participants were pregnant women between 18 and 45 years of age who met the following criteria: non-smoking; used wood fuel for cooking; had no plans to migrate in the next 1 1/2 years; and attended the Ministry of Health clinic for prenatal care. Thirty-six pregnant women were recruited consecutively from the clinic if their gestational age was <20 weeks based on ultrasound examination and met the inclusion criteria. Sociodemographic characteristics were collected for all participants.

Airborne kitchen concentrations of particulate matter ($PM_{2.5}$) were measured at three prenatal measurements (< 20 weeks, 24–28 weeks (second trimester) and 32–36 weeks (third trimester) of gestation), two neonatal measurements (<48 h since birth and one month after birth) and at 3 months of infant life (to correspond with health outcomes from the parent study) (Fig. 1A). Personal exposure to airborne PAHs was measured at three times during the course of the study: at 32–36 weeks gestational age and at one and three months after birth (Fig. 1A).

2.2. Urine collection

Twenty-three NACER study participants who were 3 months post-partum participated in the present study. The remaining 13 women were past the 3rd month post-partum and were, therefore, not included in this sub-study. Since use of wood-fired sauna baths have been shown to greatly increase levels of CO exposure, one by-product of incomplete combustion of wood fuel (Lam et al., 2011; Thompson et al., 2011b), we measured urinary metabolite concentrations after multiple types of smoke exposure such as cooking and sauna baths. Urine samples ($n = 68$) were collected three times over a 72 h period: at first morning urine (fasting), after lunch and following dinner or sauna bath use (Fig. 1B). Women were instructed to use the clean-catch method and collected their urine in sterile polypropylene cups which were stored on ice until picked up by study personnel. Samples were processed at the field laboratory and were then shipped on dry ice to the United States and stored at -20 °C until laboratory analysis.