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Assessment of vascular function in Mexican women exposed to polycyclic aromatic hydrocarbons from wood smoke

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ABSTRACT

The use of solid fuels for cooking and heating is likely to be the largest source of indoor air pollution on a global scale; these fuels emit substantial amounts of toxic pollutants such as polycyclic aromatic hydrocarbons (PAHs) when used in simple cooking stoves (such as open "three-stone" fires). Moreover, indoor air pollution from biomass fuels is considered an important risk factor for human health. The aim of this study was to evaluate the relationship between exposure to PAHs from wood smoke and vascular dysfunction; in a group of Mexican women that use biomass combustion as their main energy source inside their homes. We used 1-hydroxypyrene (1-OHP) as an exposure biomarker to PAHs and it was assessed using high performance liquid chromatography. The endothelium-dependent vasodilation was assessed through a vascular reactivity compression test performed with a pneumatic cuff under visualization of the brachial artery using high resolution ultrasonography (HRU). Assessment of the carotid intima-media thickness (CIMT) was used as an atherosclerosis biomarker (also assessed using HRU); and clinical parameters such as anthropometry, blood pressure, glucose, triglycerides, total cholesterol, HDL cholesterol, LDL cholesterol, among others were also evaluated. The mean concentration of urinary 1-OHP found in exposed women was 0.46 ± 0.32 μmol/mol Cr (range: 0.086–1.23 μmol/mol Cr). Moreover, vascular dysfunction (diminished endothelium dependent vasodilation) was found in 45% of the women participating in the study. Association between vascular function and 1-OHP levels was found to be significant through a logistic regression analysis ($p = 0.034$; $r^2 = 0.1329$). Furthermore, no association between CIMT and clinical parameters, urinary 1-OHP levels or vascular dysfunction was found. Therefore, with the information obtained in this study, we advocate for the need to implement programs to reduce the risk of exposure to PAHs in communities that use biomass fuels as a main energy source.

1. Introduction

The use of solid fuels for cooking and heating is likely to be the largest source of indoor air pollution on a global scale (WHO, 2014). Moreover, indoor air pollution from biomass fuel combustion is considered an important risk factor for human health, since it is responsible for approximately 1.6 million early deaths around the world (WHO, 2014). Half of the world's population uses biomass fuels including: wood, charcoal, dung, crop residues, among others (WHO, 2014); for example, in Mexico, 27 million people use wood as their principal energy source (Masera et al., 2005; Riojas-Rodriguez et al., 2011). Biomass fuels are usually burned in open fires, and in clay or metal stoves, leading to an incomplete combustion and the subsequent formation of intermediate compounds within the smoke, including carbon monoxide (CO), nitrogen and sulfur oxides (NOx, SOx), aldehydes, polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), dioxins and particulate matter (PM2.5 and PM10) (Albalak et al., 2001).

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PAHs are compounds consisting of carbon and hydrogen with at least two conjugated aromatic rings. PAHs with four or more rings have low-volatility and can be inhaled, mainly when attached to particulate matter (Jongeneelen, 2014). PAHs exposure occurs as a mixture of compounds in which pyrene is almost always found; for this reason, pyrene and its metabolite 1-hydroxypyrene (1-OHP) have been used for the assessment of the presence of these compounds in human populations (Domínguez-Cortinas et al., 2013; Martínez-salinas et al., 2012; Pérez-Maldonado et al., 2014; Pruneda-Álvarez et al., 2012; Ruíz-Vera et al., 2014; Trejo-Acevedo et al., 2012).

Several studies have demonstrated an association between chronic PAHs exposure and toxic effects on human health (Kim et al., 2013; Mumtaz and George, 1996). Increased incidence of lung, skin, bladder and gastrointestinal cancers are associated with occupational exposure to PAHs (Bach et al., 2003; Boffetta et al., 1997; Diggs et al., 2011; Olsson et al., 2010), while non-carcinogenic effects of PAHs primarily involve the pulmonary, gastrointestinal, renal and dermatologic systems (Mumtaz and George, 1996). Furthermore, DNA damage induced by PAHs exposure has been demonstrated in several studies (Jasso-Pineda et al., 2015; Torres-Dosal et al., 2008) and also, other recent studies suggest that PAHs contribute to induce cardiovascular disease (Brucker et al., 2014, 2013; Feng et al., 2014; Xu et al., 2013).

In this regard, vascular dysfunction is considered a pathophysiological link between the cardiovascular risk factors and the development of atherosclerosis and subsequent cardiovascular illnesses (Mudau et al., 2012; Yang et al., 2010). Impairment of the endothelium dependent vasodilation constitutes an early stage, and is the most important characteristic of vascular dysfunction. However, to date information about vascular function in people exposed to PAHs is scarce. In this context, in a previous study performed by Buturak et al. (2011), endothelium dependent vasodilation was assessed in individuals who have used biomass fuels throughout life, the results showed a significant decrease of the clinical parameter when compared to controls, however the authors did not assess the relationship between endothelial dysfunction and PAH exposure through the use of a biological indicator of exposure.

Therefore, the aim of this study was to evaluate the relationship between exposure to PAHs from wood smoke and vascular dysfunction; in a group of Mexican women that use biomass combustion as their main energy source inside their homes.

2. Materials and methods

2.1. Population

During 2013, a cross-sectional study was performed in a group of 40 women (aged 19–81) residing in the rural community of El Leoncito, San Luis Potosi, Mexico. They use wood as their principal fuel to cook in traditional indoor open fires. The community has 300 inhabitants, 151 of which are women, and shows a high degree of poverty (INEGI, 2010). The inclusion criteria were: apparently healthy adult women, non-smokers (only women reported to be unexposed according to survey results were included in the study; for example, we assessed tobacco smoke exposure using the following questions: “is the woman smoking?” and “are the smokers living with the woman?”, the possible responses were “yes” or “no”), cook with biomass; exclusion criteria were: pregnant or lactating, currently under medication and family history of cardiovascular disease. The participants signed an informed consent of voluntary participation, and responded to a survey for socioeconomic, nutrition, health and exposure data. The study was reviewed and approved by the Bioethics Committee of the School of Medicine, at the Autonomous University of San Luis Potosi.

2.2. Blood and urine sample collection

Blood samples were drawn from the cubital vein into 8 mL vacuum tubes without anticoagulant for serum collection. Vacuum tubes were centrifuged at 1200 × *g* for 10 min and serum was transferred to test tubes and stored at −20 °C until analysis. Urine samples were taken in the morning (first morning urine), collected in sealable plastic bottles and stored at −20 °C. Before analysis, samples were thawed at room temperature, homogenized, filtered through a 0.45 μm pore size membrane (Millipore, Bedford, MA, USA), and a 10 mL aliquot of urine was transferred to a test tube. Urine samples were used to determine 1-OHP and creatinine levels, and blood samples were used to perform clinical analyses (glucose, triglycerides, total cholesterol, HDL cholesterol and LDL cholesterol).

2.3. Determination of urinary 1-OHP

Urinary 1-OHP was quantified following the method described by Pruneda-Álvarez et al. (2012). Each aliquot (10 mL) was mixed with sodium acetate buffer (10 mL, 0.2 M, pH 5.0), then 30 μL of β-glucuronidase/arylsulfatase enzyme were added and the sample was incubated at 37 °C for 12 h. The analyte was extracted by solid phase using C-18 cartridges, eluted in methanol containing 1% acetic acid. The eluate was concentrated with nitrogen current to 0.5 mL. The concentrated eluate was then filtered through a polyvinylidene fluoride filter (13 mm, 0.45 μm, MillexDurapore, Millipore, Bedford, MA, USA), and an aliquot was transferred to silanized vials. Then, the analyses were performed by HPLC (HP1100, Agilent Technologies) using a fluorescence detector (G1321A). The pre-column was a Zorbax SB-C18, and the column a Zorbax Eclipse XDB-C18. The column temperature was set to 40 °C, flow was adjusted to 1 mL/min and the injection volume was 20 μL. The mobile phase was 88:12 methanol/water and 1% ascorbic acid. Data were collected and processed using HP ChemStation software. Urinary 1-OHP concentrations were adjusted to urinary creatinine, which was determined using the Jaffe colorimetric method (Taussky, 1954; Spinreact, Spain). Under our conditions, the method detection limits were 0.03 nmol/L. Quality control was set by using control lyophilized standards ClinChek RECIPE (Munich, Germany) I and II (0.64 and 4.14 μg/L 1-OHP), and the recovery was 98%.

2.4. Vascular function assessment

Women were examined by a cardiology expert who was blinded to the study design and participants’ clinical data. Measurement of the endothelium dependent (flow-mediated) vasodilation was performed through high resolution ultrasonography (HRU) of the brachial artery according to the method described by Celermajer et al. (1992). Women were placed in supine posture with the arm in a comfortable position for imaging of the brachial artery. The artery was imaged just above the antecubital fossa in the longitudinal plane, using a portable ultrasound equipment (Sonosite 180 Plus, Washington, USA) coupled with a C11/7-4 transductor (11-mm broadband, 7–4 MHz). A segment with clear anterior and posterior intimal interfaces between the lumen and the vessel wall was selected for continuous 2D grayscale imaging. To create a flow stimulus in the brachial artery, a sphygmomanometric cuff was first placed on the forearm. A baseline rest image was acquired, and blood flow was estimated by time-averaging the pulsed Doppler velocity signal obtained from a mid-artery sample volume. Arterial occlusion was created through cuff inflation at a suprasystolic