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The influence of polycyclic aromatic hydrocarbons on lung function in a representative sample of the Canadian population☆

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ABSTRACT

We investigated the associations between exposure to polycyclic aromatic hydrocarbons (PAHs) and selected respiratory physiologic measures in cycles 2 and 3 of the Canadian Health Measures Survey, a nationally representative population sample. Using generalized linear mixed models, we tested the association between selected PAH metabolites and 1-second forced expiratory volume (FEV_1), forced vital capacity (FVC), and the ratio between the two (FEV_1/FVC) in 3531 people from 6 to 79 years of age. An interquartile change in urinary PAH metabolite was associated with significant decrements in FEV_1 and FVC for eight PAHs, 2-hydroxynapthalene, 1-, and 2-hydroxyphenanthrene, 2-, 3-, and 9-hydroxyfluorene and 3- and 4-hydroxyphenanthrene. Exposure to PAH may negatively affect lung function in the Canadian population.

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) are formed by incomplete combustion of fossil fuels and biomass burning from a variety of sources including wood stoves, cigarettes, and traffic-related air pollution (Khalili et al., 1995; Slezakova et al., 2009). They may be adsorbed onto fine particulate air pollutants or accumulated by plants, while certain types of food processing (e.g. smoking) and cooking (e.g. barbecuing) techniques can increase PAH concentrations in food. Consequently, major routes of human bioaccumulation include ingestion as well as inhalation (Abdel-Shafy and Mansour, 2015; Srogi, 2007). PAHs may be genotoxic, carcinogenic, and induce oxidative stress (Armstrong and Gibbs, 2009; Boström et al., 2002; Gammon and Santella, 2008) PAH exposure has been linked to adverse respiratory health outcomes in children, including bronchitis (Hertz-Picciotto et al., 2007; Jedrychowski et al., 2005), and reductions in the forced expiratory volume, FEV_1 (Choi et al., 2013; Barraza-Villarreal et al., 2014; Jedrychowski et al., 2015; Padula et al., 2015; Zhou et al., 2016). Among adults in occupational settings, elevated PAH exposures have been found to be associated with declines in FEV_1/FVC (Wang et al., 2016).

The present study addresses the gap in knowledge concerning the effect of PAH exposure on lung function, independent of smoking among adults and children in a large, population-based sample. Those exposed to active or passive cigarette smoke were excluded to avoid confounding. Concentrations of urinary PAH metabolites among smokers would be a proxy for exposure to cigarette smoke. Any relation observed between PAH and lung function could be due, not to the PAH per se, but rather the many other toxic combustion products in cigarettes. In our study of non-smokers, sources of PAH exposure are expected be from cooking, motor vehicle exhaust, and foods. These sources are less likely than cigarettes to have significant pulmonary effects. Because air pollution is a known cause of small declines in lung function, we took this into consideration.

We used data from The Canadian Health Measures Survey (CHMS) carried out between 2007 and 2013 (Haines et al., 2016), a nationally representative survey which collects questionnaire data and physical measurements, including blood and urine samples. PAHs were measured in CHMS cycles 2–4 (2009–2011, 2012–2013 and 2014–2015).

2. Methods

2.1. Study population

The CHMS study population included subjects aged between 3

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and 79 years of age who participated either in the cross-sectional CHMS cycle 2 or 3. CHMS cycle 2, from 2009 to 2011, had a total of 6395 participants, while 5071 participated in CHMS cycle 3 from 2012 to 2013. Subjective and objective health data were obtained from the subjects at 18 (cycle 2) and 16 (cycle 3) collection sites across Canada selected based on probability sampling considering the population within the selected sites. Households within each site were stratified by age group, with at least one subject of the desired age living in the household. Participants who self-reported smoking or being exposed to tobacco smoke at home in questionnaires were excluded from this analysis.

2.2. Physiologic measurements

For subjects between 6 and 79 years of age, lung function variables, 1-s forced expiratory volume (FEV_1), and forced vital capacity (FVC) were obtained using a KoKo Spirometer™ (Ferraris CardioRespiratory, Pulmonary Data Services, Inc, Louisville, CO) operated by trained personnel following standardized criteria for test performance (Miller et al., 2005). Up to eight trials were permitted to obtain three acceptable and reproducible test results. Values were expressed as percentage of predicted (100* observed value/predicted value) based on the subject's age, height, and gender using standard prediction equations (Hankinson et al., 1999).

2.3. PAH measurements

PAH metabolites were included in the study if above the limit of detection in more than 80% of samples. Eleven PAHs: 1-Hydroxynaphthalene, 2-Hydroxynaphthalene, 1-hydroxypyrene, 2-hydroxyfluorene, 3-hydroxyfluorene, 9-hydroxyfluorene, 1-hydroxyphenanthrene, 2-hydroxyphenanthrene, 3-hydroxyphenanthrene, 4-hydroxyphenanthrene, and 9-hydroxyphenanthrene were measured in urine samples from respondents between 3 and 79 years of age; results from the 6–79 year old subjects only were included in this study to match the available age range for lung function measurements. Lung function and PAH measurements were available for 4045 participants. A total of 514 smokers were excluded. The number of participants included in the final analysis totaled 3531.

Urine samples were hydrolyzed using a b-glucuronidase enzymatic solution and extracted with an organic solvent at neutral pH, evaporated and derivatised with *N*-methyl-*N*-(trimethylsilyl)-trifluoroacetamide, then analyzed using an Agilent 7890 gas chromatograph coupled to an Agilent 7000 B triple-quad tandem mass spectrometer operating in electron impact ionization mode. Analytes were quantified using microanalytical reference material (Health Canada, 2015). PAH metabolite levels were reported as directly adjusted for the effect of urinary dilution by dividing concentrations (ug/L) by urinary creatinine levels (g/L).

2.4. Statistical analysis

Generalized linear mixed models with measurement site as a random effect were used to link log-transformed creatinine-corrected urinary PAH metabolite concentrations and respiratory physiology. Data management and regression modeling were completed in SAS, EG.5.1 (Cary, NC, USA). Models were adjusted for race (white versus others), age, gender, education, total household income, alcohol consumption, home heating type, and occupation. If inclusion of the variable changed the beta coefficient by more than 10%, it was retained in the model. The analysis was stratified by sex, age (6–15, 16–79 years) and season (Warm: April to September, Cold: October to March). Results were presented as the magnitude of change in a physiologic variable, for an interquartile increase in creatinine-adjusted urinary PAH.

To determine if air pollution, a source of PAH exposure and cause of lung toxicity, could confound an observed association between PAHs and lung function, correlations were measured between PAHs and regional concentrations of ozone, fine particulate matter, and nitrogen dioxide.

Finally, in an effort to tease out the independent effects of individual PAHs, each was adjusted for the sum of the others.

3. Results

3531 participants were eligible for analysis, defined by being a non-smoker with at least one PAH and one lung function measurement. There were approximately equal numbers of males and females, and 583 more subjects in the under 16 age group compared to the older group.

The number of participants with urinary concentrations of PAH metabolites ranged from 3281 for 2-hydroxynaptahelene to 3531 subjects for 2-hydroxyfluorene. Concentrations varied from a low of 0.04 (95%CI, 0.03–0.05) μg/g for 9-hydroxyphenanthrene to a high of 3.92 (95%CI, 3.65–4.21) μg/g 2-hydroxynaphthalene (Table 1). Mean concentrations of all metabolites were similar (within one standard deviation) between the younger and older age groups, males and females, and warm and cold season measurements.

All Pearson correlations between original PAH metabolites were significant at $p < 0.001$, ranging from 0.2 between 1-hydroxypyrene and 1-hydroxynapthalene, and 0.92 between 2-hydroxyfluorene and 3-hydroxyfluorene (Table 2).

Expressed as a percent of predicted based on the external reference equations, lung function was between 98% and 103%, consistent with a general population sample of non-smokers. There was no significant difference between the younger and older age group, males and females, or between warm and cold season measurements (Table 3).

For the population as a whole (Table 4), an interquartile change in urinary PAH metabolite was associated with a significant reduction in FEV_1 expressed as a percent predicted, for eight of the PAH metabolites examined, ranging from −0.56% (95% CI -0.87, −0.25) for 4-hydroxyphenanthrene to −0.90% (95% CI -1.28, −0.53) for 2-hydroxyphenanthrene. Significant reductions in FVC were associated with increases in the same eight PAHs, up to −1.04% (95% CI -1.40, −0.68) for 2-hydroxyphenanthrene. The percent changes in the FEV_1/FVC ratio across all PAHs ranged from −0.07% (95%CI -0.26, 0.13) for 3-Hydroxyfluorene to 0.16% (95% CI -0.04, 0.35) for 3-hydroxyphenanthrene, although none were significantly greater than zero.

Larger effects were reported for the sum of the compound classes. An IQR change in the sum of 2, 3, 9-hydroxyfluorene was associated with a −1.41% (95% CI -2.68, −0.14) reduction in FEV_1, and a −1.28% (95% CI -2.46, −0.10) change in FVC. The sum of 1 and 2-hydroxynapthalene resulted in a greater reduction in FVC than either naphthalenes individually, −0.71% (95% CI -1.30, −0.12). Similarly for an IQR change in the sum of the phenanthrenes, we observed a −2.49% (95% CI -4.56, −0.33) change in FEV_1 and a −1.99% (95% CI -3.85, −0.13) change in FVC. We also performed a sensitivity analysis here: the effects of all but two PAHs were modified significantly when adjusted for the sum of the other PAH metabolites. The FEV_1 fell from −0.78% to −2.27% when 2-hydroxyfluorene was adjusted for the effect of the other PAHs. FEV_1 fell from −0.56 %to −1.66 %when 4-hydroxyphenanthrene was adjusted for the other PAHs. No other significant associations remained after adjustment.