





ORIGINAL RESEARCH ARTICLE



Polycyclic aromatic hydrocarbons from environmental tobacco smoke and wood stoves dominate in settled house dust from Northwestern Ontario First Nations communities

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ABSTRACT

Rates of respiratory tract infections for children living in remote First Nations communities in the Sioux Lookout Zone in Northwestern Ontario are elevated and associated with poor indoor environmental quality including high exposures to endotoxin and serious dampness and mould damage. The studies also revealed a high prevalence of cigarette smoking and most houses have wood stoves, of variable quality. Depending on structure, polycyclic aromatic hydrocarbons (PAH) are carcinogens, immunotoxins and/or inflammatory mediators that are byproducts of the incomplete combustion of organic materials. Indoor sources of PAHs include tobacco smoke, cooking, and burning wood and/or fossil fuels for house heating. Twelve PAHs were measured in the <300 µm fraction of settled house dust by GC-MS in 59 houses. Nine PAHs were detected in all 59 houses, and median concentrations of individual PAHs measured ranged from 66 to 804 ng/g. PAHs associated with environmental tobacco smoke and with wood smoke dominated the PAH profile. Limiting tobacco smoking indoors and upgrading to low emission airtight wood stoves would improve indoor air quality and the respiratory health of children in this remote region of Ontario.

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Introduction

McCuskee et al. (2014) found that 21% of children in remote communities in the Sioux Lookout Zone, Ontario had been admitted to hospital for respiratory infections before two years of age [1]. This is consistent with a pan-Canadian study that reported that on-reserve First Nations populations had higher rates of hospitalisation for respiratory tract infections and asthma than individuals living off-reserve [2]. In partnership with four First Nations Communities in Northwestern Ontario, the Sioux Lookout Zone First Nations Health Authority, and the Nishnawbe Aski Nation, we studied the housing conditions and health in the four communities. A significant fraction of the homes had serious mould damage, most were under-ventilated for their occupancy and had elevated endotoxin concentrations in the settled dust. Almost all the homes had wood stoves and one or more cigarette smokers. All these factors are known to negatively affect respiratory health [3,4].

Polycyclic aromatic hydrocarbons (PAHs) are a group of chemicals naturally found in coal, gasoline and oil, and are formed during the incomplete combustion of oil, wood and other organic substances including tobacco and charred meat [5]. In urban North America, the primary sources of PAH exposures include inhalation through active or passive smoking and wood smoke, as well as the consumption of various foods. Occupational and outdoor air exposures are higher in or near oil refining and other facilities where oil, wood or other organics are burned as well as near major roads [5,6]. In urban Canada, urinary PAH values are primarily explained by smoking and diet [7]. A small study of PAHs in settled house dust in a community in Eastern Ontario found higher values correlated with occupant smoking and the presence of an attached garage [8]. In communities where wood stoves are common, the fraction of air particulates that contain PAHs was higher in homes with older and non-EPA certified stoves or when burning wet wood [9].

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There are limited data on PAH exposures in First Nations communities in Canada. Ratelle et al. (2020) measured urinary PAHs from ~100 First Nations participants from communities in the Northwest Territories (subarctic Canada) [10]. They found the values of the PAHs measured were associated with smoking and consumption of cooked meat. Exposures to certain PAHs measured in urine, total exposure from both inhalation and diet, were weakly associated with impaired lung function in adults [11]. Certain PAHs have been associated with asthma assessed from both inhalation and total exposure [12–14]. Some common PAHs result in inflammation in lung tissues [15]. Aside from respiratory impacts, certain PAHs from cigarette smoke and wood smoke are probable human carcinogens (Group 2A), others are possible human carcinogens (Group 2B), and, benzo[a]pyrene, is carcinogenic to humans (Group 1) [15]. Here, we report PAH concentrations in the settled dust from 59 of the Sioux Lookout Zone homes we previously investigated and discuss their sources.

Methods

This study was conducted in four First Nations communities in Northwestern Ontario, three of which were not accessible by road except during a brief period in winter [3]. They are predominantly Anishinaabe with some Cree communities and are among the remote communities for which the Meno Ya Win Health Centre in Sioux Lookout, Ontario serves as the regional hospital (50.10° N, 91.92° W). Research Ethics Board approvals were obtained from Health Canada, the Children's Hospital of Eastern Ontario, the Ottawa Hospital, and the Sioux Lookout Meno Ya Win Health Centre. Permission was granted and support was provided by each of the four

communities. The Nishnawbe Aski Nation has full ownership of the resulting data, according to the First Nations principles of ownership, control, access, and possession (OCAP®) [3,16].

Settled dust sampling is described in detail by Kovesi et al. (2022) [3]. Briefly, samples were collected from the living room floor with an x-cell 100 dust sock fitted to the hose of an Omega HEPA vacuum cleaner (Midwest Filtration Company, Cincinnati, OH, USA). Samples were transported and stored under air-dry conditions and the dust was sieved to 300 µm and weighed. The dust <300 µm was analyzed for endotoxin, house dust mite allergens, and 1,3-beta-D-glucan [3]. Samples were collected from 101 houses; however, there was sufficient settled dust from 59 houses to perform the PAH analysis. For the 59 houses, wood was the primary heating source for 70% and a secondary source in most of the rest. Cigarette smoking occurred in 93% of the houses with an average of 2.6 smokers per household [3].

The PAHs listed in Table 1 were quantified using a method adapted from Wan et al. (2022) [8]. Briefly, 100 mg amounts of sieved dust from the 59 houses were transferred to KIMAX® clear glass 12 mL glass vials (Fisher Scientific). To each sample, 1 g of anhydrous sodium sulfate (dried overnight, 450°C; Bioshop Canada) and 500 ng of each surrogate standard was added (phenanthrene-d10, benzo[a]anthracene-d12, and benzo[a]pyrene-d12; Chromatographic Specialties). Samples were extracted with 3 mL of ACS grade dichloromethane (CFS Chemicals), subsequently vortexed for 1 min, sonicated for 15 min and centrifuged (3000 rpm, 10 min, 4°C). The organic layers were transferred to 15 mL amber glass vials (Fisher Scientific) and the remaining dust pellets were extracted two additional times as described above. The combined dichloromethane extracts were dried under a gentle

Table 1. Retention times, quantifier and qualifier ions used to measure PAHs, and method limits of detection.

PAH	RT (min)	<i>m/z</i> quantifier	<i>m/z</i> qualifier	MDL (ng/g)
phenanthrene	15.2	178	176	1.1
anthracene	15.4	178	176	1.3
fluoranthene	18.0	202	200	1.3
pyrene	18.6	202	200	0.9
benz[a]anthracene	21.47	228	226	1.4
chrysene	21.57	228	226	1.4
benzo[b]fluoranthene	23.8	252	250	2.3
benzo[k]fluoranthene	23.9	252	250	5.7
benzo[a]pyrene	24.5	252	250	4.1
indeno[123-cd]pyrene	26.6	276	277	2.1
benzo[ghi]perylene	27.0	276	274	2.1
dibenz[ah]anthracene	26.6	278	276	2.4
phenanthrene-d10 (S)	15.2	188		
benz[a]anthracene-d12 (S)	21.4	240		
benzo[a]pyrene-d12 (S)	24.4	264		
fluoranthene-d10 (IS)	17.9	212		

S- surrogate, IS- internal standard.

stream of nitrogen gas. Resulting extracts were suspended in 1 mL of ACS grade hexane (Sigma-Aldrich) and cleaned by SPE (Florisil® Superclean ENVI-Florisil SPE tubes, 6 mL, 1 g; Supelco) cartridges that were conditioned with ACS grade methanol (Fisher Scientific) and hexane. PAHs were eluted from cartridges with 10 mL ACS grade ethyl acetate (Sigma-Aldrich) followed by 4 mL of methanol and dried under a stream of nitrogen gas. Extracts were resuspended in 0.5 mL of toluene (Sigma-Aldrich) and 150 ng of the internal standard (fluoranthene-d10; Chromatographic Specialties) was added before the extracts were filtered through 25 mm 0.2 µm PTFE syringe filters (ChromSpec, Inc) and analysed by GC-MS.

PAHs were quantified with an Agilent 6890 gas chromatograph operated in splitless mode coupled to an Agilent 5973N mass selective detector. Analytes were separated with an HP-5 capillary column (30 m × 0.25 mm i.d × 0.25 µm; Agilent Technologies) and helium carrier gas (1.5 mL/min). The temperature program was held at 75°C for 3 min, increased to 320°C at 10°C/min linearly, and held for 5.5 min. The injector and transfer line temperatures were 300°C and 280°C, respectively. Two µL of each extract was injected and from 150 to 550 *m/z* was scanned with the filament

emission set at 70 eV. Both full scan and single ion monitoring data were collected for all samples. The quantifier and qualifier ions used to measure and confirm the identity of PAHs in the dust samples are shown in Table 1. Quantification of PAHs was achieved using six-point calibration plots (2000 ng/mL to 50 ng/mL) generated from a Polynuclear Aromatic Hydrocarbons Mix (CRM 48,905; Supelco) where the concentration of the internal standard was 300 ng/mL.

Validation of the extraction and analysis method for targeted PAH was achieved using the US National Institute of Standards and Technology Standard Reference Material 2585 (organic contaminants in house dust). PAH concentrations (ng/g) experimentally determined were compared with the certified values and were within 20% for all PAHs considered except anthracene (Table 2). Systat 13 (Systat, Inc. Chicago, IL) was used to perform statistical analyses.

Results

The method used provided comparable results to those values in the NIST Standard Reference Material 2585 (Table 2). Twelve PAHs were quantified in the settled dust samples and nine were detected

Table 2. Comparison of PAH concentrations measured from SRM 2585 as part of this study with certified values. Values in brackets are %RSD.

PAH	this study	SRM 2585*	% difference
phenanthrene	2051 (7)	1920 (1)	6.8
anthracene	118 (7)	96 (5)	23
fluoranthene	4071 (6)	4380 (2)	7.1
pyrene	3004 (6)	3290 (1)	9.7
benz[a]anthracene	1311 (2)	1160 (5)	13
chrysene	1885 (4)	2260 (3)	17
benzo[b]fluoranthene	3177 (11)	2700 (3)	18
benzo[k]fluoranthene	1504 (6)	1330 (5)	13
benzo[a]pyrene	1265 (1)	1140 (1)	11
indeno[123- <i>cd</i>]pyrene	1804 (2)	2080 (5)	13
benzo[ghi]perylene	2031 (5)	2280 (2)	11
dibenz[ah]anthracene	324 (7)	301 (17)	7.6

*NIST. Certificate of Analysis for Standard Reference Material 2585 Organic Contaminants in House Dust. 2014.

Table 3. PAHs measured in the settled house dust from the 59 houses.

PAH	minimum	maximum	median
phenanthrene	326	841	447
anthracene	59	193	88
fluoranthene	269	918	384
pyrene	24	288	66
benz[a]anthracene	195	650	281
chrysene	170	586	264
benzo[b]fluoranthene	364	2019	804
benzo[k]fluoranthene	284	1459	593
benzo[a]pyrene	290	1475	624
indeno[123- <i>cd</i>]pyrene [1]	136	570	228
benzo[ghi]perylene [2]	127	298	237
dibenz[ah]anthracene [3]	53	262	115

1. 25 houses; 2. 14 houses; 3. 44 houses.

from each of the 59 houses examined (Table 3). Median concentrations of individual PAHs measured ranged from 66 to 804 ng/g. Three PAHs, indeno[123-*cd*]pyrene, benzo[*ghi*]perylene, dibenz[*ah*]anthracene, were below the method limit of detection in 58%, 76% and 25% of the homes, respectively (Table 3). The prevalence of the remaining compounds ranged from 10% to 25%.

Hoh et al. compared PAHs in homes with and without smokers [17]. They identified four PAHs in dust significantly associated with smoking including phenanthrene, fluoranthene, pyrene and chrysene. Gustafson et al. conducted a similar experiment except with wood heating and without [18]. These authors found that benzo[*b*]fluoranthene, benzo[*a*]pyrene and indeno[123-*cd*]pyrene were significantly associated with wood heating. The distributions of these seven PAHs are displayed in Figure 1. The centre vertical line in the box plots

marks the median of the sample. The length of each box shows the range within which the central 50% of the values fall, with the box edges at the first and third quartiles.

Discussion

The characteristic PAHs for environmental tobacco smoke and wood smoke indoors were present in the settled house dust from the 59 homes. Median values of individual PAHs in the settled house dust samples ranged from similar to up to >2 orders of magnitude above those reported from the Kingston study in Eastern Ontario [8]. For the Kingston samples, only phenanthrene, chrysene, benzo[*b*]fluoranthene and benzo[*a*]pyrene were present in all homes. There was a high prevalence of a comparable list of PAHs in

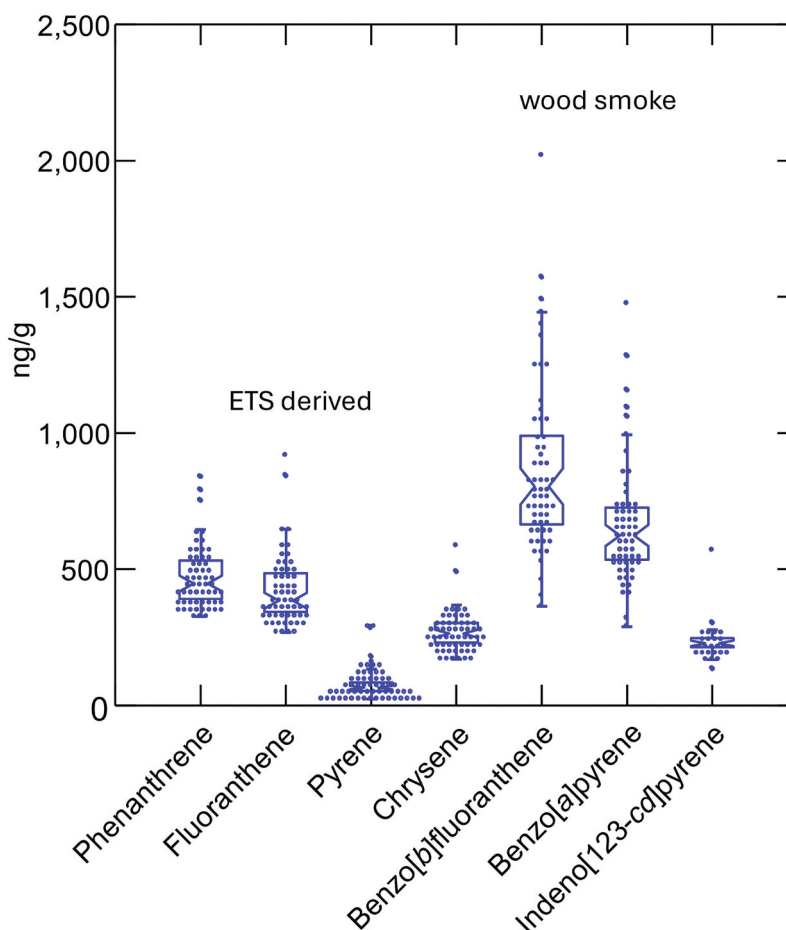


Figure 1. Concentrations of PAHs in settled house dust (ng/g) associated with environmental tobacco smoke (ETS; phenanthrene, fluoranthene, pyrene, chrysene) and wood smoke (benzo[*b*]fluoranthene, benzo[*a*]pyrene, indeno[123-*cd*]pyrene) from 59 houses in the Sioux Lookout Zone.

settled dust collected in Northern California homes sampled between 2001 and 2007. This is a region historically subject to high levels of outdoor air pollution notably from traffic and in a population where ~43% smoked [19]. Median values in these samples are somewhat lower than the present data. PAHs in settled dust samples across China collected in 2021 were present in broadly similar concentrations to those from the Sioux Lookout Zone houses. As was the case in the present samples, the dominant PAHs in dust samples collected in residential housing in China were from smoking and fuels used for heating including biomass [20].

The characteristic PAHs from environmental tobacco smoke (ETS), phenanthrene, fluoranthene, pyrene and chrysene were present in all homes. Chrysene is a possible human carcinogen (Group 2B) and the rest are known to be toxic and inflammatory in lung cells and in other tissues in relevant animal models [15,21–23]. For children, ETS exposure increases the risk of acute lower respiratory (e.g. bronchitis and pneumonia), asthma induction and exacerbation as well as chronic respiratory symptoms [24,25].

The characteristic PAHs from wood smoke, benzo[b]fluoranthene, benzo[a]pyrene, indeno[123-cd]pyrene were present in all homes over a six fold concentration range. Benzo[b]fluoranthene is a possible human carcinogen (Group 2B) and benzo[a]pyrene is a known human carcinogen (Group 1) [15]. As with those derived from second-hand cigarette smoke, the PAHs associated with wood smoke also modulate lung biology. Of these, most is known about the effects of benzo[a]pyrene on lung biology. This PAH is potentially inflammatory in the lungs of relevant animal models and in vitro systems [15,24–26]. The 59 houses considered here had elevated concentrations of another highly inflammatory agent, endotoxin. Endotoxin is known to occur in smoke [4]. Co-exposure to benzo[a]pyrene and endotoxin increases toxicity [27]. Benzo[a]pyrene is a human carcinogen, and benzo[b]fluoranthene, and indeno[123-cd]pyrene are probable human carcinogens. The risk increases if the exposure exceeds the ability of the lung tissue to repair the damage [15].

About 10% of the homes had concentrations of the wood smoke derived PAHs in the settled dust above the top quartile (Figure 1). This suggests that ≥10% of these homes need upgraded stoves as soon as feasible, if not already completed. Funding is required to upgrade all the stoves to low emission airtight stoves in a timely fashion. Replacing wood stoves with EPA certified units reduces PM_{2.5} including the fraction containing PAHs compared to older stoves [28]. Based on studies with healthy adult atopic volunteers, the wood

smoke exposures at concentrations that occur with modern airtight stoves had little effect on their lungs [29]. In addition, more effort may be required to provide information on the impact of smoking of commercial tobacco on child health in this region.

As noted above, it has long been known that exposure to ETS negatively affects the respiratory health of children. The Anishinaabe Sacred Smoke Program has resources on smoking and health that may deserve promotion in this region (<https://tobaccowise.cancer.ca/en/community-success-stories/sacred-smoke-program>). A recent study of housing and health in homes in First Nations communities in British Columbia reported a smoking prevalence of 32% [30].

Conclusion

As part of a larger study on housing and child respiratory health in four remote First Nations in Northwestern Ontario, we report PAH concentrations in a subset of homes. Cigarette smoking was present in all homes and the homes had wood stoves of uncertain ages. Using a validated method, concentrations of 12 PAHs were determined in settled dust, 9 of which were present in 100% of the homes at concentrations higher than in a study PAHs of house dust from eastern Ontario. The results support greater investments in modern airtight stoves in these communities. In addition, more effort may be required to provide information in the region on the impact of cigarette smoking on the respiratory health of children.

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Disclosure statement

No potential conflict of interest was reported by the author(s).

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Author contributions

Kirkland, Rehman, McMullin - Sample preparation and analysis; McMullin - Methodology; McMullin and Miller - Writing original draft, Reviewing and editing - all authors; McMullin, Kovesi, Malloch, Miller - Funding acquisition.

Data access

The data used for the analysis is not available for sharing. When study participants provided informed consent, they did not agree for their data to be shared beyond the research team. This is in the context of policies in Canada on Ownership, Control, Access and Possession of data when conducting research with Indigenous communities (<https://fnigc.ca/ocaptraining/>). Readers may contact the corresponding authors for more information. Readers may also reach out to Dr. Ariel Root <ariel.root@slfnha.com>, Program Manager at the Sioux Lookout First Nations Health Authority, if they would like to request access to the full dataset.

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