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Mutagenic and Carcinogenic Hazards of Settled House Dust I: Polycyclic Aromatic Hydrocarbon Content and Excess Lifetime Cancer Risk from Preschool Exposure

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Settled house dust (SHD) may be a significant source of children's indoor exposure to hazardous substances including polycyclic aromatic hydrocarbons (PAHs). In this study, organic extracts of sieved vacuum cleaner dust from 51 homes were examined for the presence of 13 PAHs via GC/MS. PAHs were found in all samples with levels of total PAHs ranging between 1.5 and 325 μg g^{-1}. The PAH concentrations in the SHD were correlated with information contained in corresponding household questionnaires. Analyses showed levels of PAHs to be negatively associated with noncombustion activities such as vacuum cleaning frequency. A risk assessment was conducted to evaluate the excess lifetime cancer risks posed to preschool aged children who ingested PAHs in SHD. The assessment revealed that exposure to PAHs at levels found in 90% of the homes ($<$40 μg g^{-1}) would result in excess cancer risks that are considered acceptable (i.e., 1–100 $\times$ 10^{-6}). However, exposure to higher levels of PAHs found in five homes yielded risks that could be higher than 1 $\times$ 10^{-4}.

Introduction

Previous studies have noted that concentrations of chemical contaminants may be higher in indoor air than in outdoor air (*1*). Indoors, many contaminants adsorb to particulate matter, which is initially suspended in air and later settles out as dust. Research suggests that settled house dust (SHD) may be a significant source for indoor exposures to many pollutants (*2*). Previous studies have revealed the presence of numerous chemical contaminants in SHD including pesticides, smoke residues, PCBs, flame retardants, plasticizers, heavy metals, and asbestos (*3–8*). With Canadians spending as much as 70% of their time at home and up to 90% of their time indoors (*9*), the health risks posed by exposure to contaminants indoors are of significant concern.

Polycyclic aromatic hydrocarbons (PAHs) have also been detected in SHD (for a review, see Maertens et al. (*10*)), and many of these compounds are known mutagens or animal carcinogens (*11*). There exists a high potential for human exposure to PAHs because of the ubiquity of their sources in both indoor and outdoor environments. As products of incomplete combustion, indoor sources of PAHs include cooking (*12, 13*), heating (*14*), smoking (*15*), wood burning (*16*), candle burning (*17*), and incense burning (*12, 18*). Outdoor sources include vehicle exhaust (*19*), forest fires, volcanoes, and industrial processes such as aluminum smelting and coke production (*20*).

Exposure to PAHs in SHD are of particular concern for children who tend to crawl on the floor and place objects in their mouths that have been in direct contact with dusty floors (*21*). A small number of studies have assessed children's exposure to PAHs in SHD as compared to other matrices (*22–24*). These assessments show that dietary ingestion of PAHs in food is often the primary exposure pathway for children. However, they also show that nondietary ingestion of carcinogenic PAHs in dust and soil is significant and is a more important exposure route than inhalation of PAHs in air. Exposure assessments indicate that toddlers playing on the floor and exhibiting hand-to-mouth behavior can ingest more than 2.5 times more PAHs than adults (*25*). Furthermore, since a child's body weight is only about one-fifth that of an average adult, a child's intake of PAHs in dust, in milligrams per kilogram of body weight per day, is likely to be far greater than that for an adult. In addition, early developmental stages of organ, immune, and nervous systems in children are thought to contribute to an enhanced contaminant sensitivity (*26*). Consequently, the adverse health risks for children exposed to PAHs in SHD are believed to be considerably greater than those for adults.

In Canada, PAHs are priority substances for assessment under the Canadian Environmental Protection Act (CEPA), and a number have been declared toxic under this Act (*20*). However, to our knowledge, there are no published studies that have evaluated PAH concentrations in SHD from Canadian homes. The objectives of this study, which includes a companion publication, are (i) to quantify levels of PAHs in SHD collected from homes in Ottawa, Canada, (ii) analyze relationships between these levels and various attributes of the households (e.g., home location, presence of smokers, percent carpet covering), and (iii) estimate the carcinogenic risks associated with preschool children's nondietary ingestion of PAHs in SHD.

Experimental Section

Study Design and Dust Sample Collection. Vacuum cleaner bags were collected between November 2002 and March 2003 from 75 homes located in Ottawa, the capital city of Canada. A two-stage stratified sampling process was used to randomly select homes that were representative of both urban and suburban locations within the city. A description of the sampling design is provided elsewhere (*27*). The bags were removed from the vacuum cleaner, placed in zip-seal plastic bags (Fisher Scientific, Ottawa, Canada), and transported to the laboratory where they were stored at −20 °C.

All participants answered a detailed questionnaire that was designed to collect information on the house and any activities that might affect chemical loading. The majority of household occupants classified their home as being located in a quiet residential area (61%). Fewer homes were located in a main residential area (29%), and an even lower number were located in a main commercial (6%) or rural area (4%). Most of the households were characterized as nonsmoking households (85%). Only 15% contained occupants who smoked, and the median number of cigarettes smoked per day was eight. The primary heating source in most of the homes was natural gas (83%); oil and electric heat were less

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TABLE 1. Monitored Ions and Fragments, Method Detection Limits (MDL), Recovery Efficiencies, and Relative Percent Difference in Duplicates (RPD) for the Quantification of 13 PAHs in SHD Extracts by GC/MS

PAH	mass to charge ratio of monitored ions and fragments	IDL (ng μL^{-1})	recovery efficiency (%)	corrected MDL ($\mu g\ g^{-1}$)[a]	av RPD[b]
acenaphthylene	152, 151, 76	0.0017	57.1	0.011	5.5
fluorene	166, 164, 82	0.0025	65.8	0.014	6.7
phenanthrene	178, 176, 89	0.0022	70.1	0.011	6.9
anthracene	178, 176, 89	0.0019	62.7	0.011	9.5
pyrene	202, 101, 100	0.0025	74.7	0.012	3.7
benz[*a*]anthracene	228, 114, 101	0.0041	72.4	0.021	5.1
chrysene	228, 114, 101	0.0052	75.7	0.025	3.8
benzo[*b*]fluoranthene	252, 126, 113	0.0039	72.1	0.019	4.5
benzo[*k*]fluoranthene	252, 126, 113	0.0072	74.3	0.035	5.5
benzo[*a*]pyrene	252, 126, 113	0.0080	57.1	0.051	4.5
indeno[1,2,3-*c,d*]pyrene	276, 138, 137	0.0074	68.1	0.040	3.4
dibenz[*a,h*]anthracene	276, 138, 137	0.0105	70.6	0.054	4.0
benzo[*g,h,i*]perylene	278, 139, 138	0.0063	69.7	0.033	3.6

[a] Corrected MDL = IDL (ng μL^{-1}) × 1000 (final volume, μL)/1000 (ng μg^{-1})/0.275 (sample mass, g)/recovery efficiency. [b] RPD = absolute difference between the duplicate divided by their average value times 100%.

common (11% and 4%, respectively), and one home did not describe the heating source.

Sample Preparation. Prior to sieving, the vacuum cleaner bags were thawed overnight in a fume hood. The dust was removed from the bags using large forceps and placed into a USA Standard Testing Sieve, ASTM E-11 specification, with a 150 μm opening. The dust was shaken through the sieve using an AS200 Digit Analytical Sieve Shaker (Retsch GmbH & Co. KG, Haan, Germany). The shaker was run at 80% (amplitude = 20 mm) for 10 min. Any visible hairs were removed from the collection pan using tweezers, a paintbrush, or both. The sieved dust (<150 μm) was then resieved on the shaker for an additional 3 min. The sieved dust was transferred to a glass jar, and the weight of the sieved dust was recorded. The jars were sealed with Teflon tape and stored at −20 °C until analysis. Of the 75 dust samples that were collected, 51 samples contained sufficient dust for chemical analyses.

Extraction and Sample Cleanup Procedures. Approximately 0.3 g of each of the 51 dust samples were extracted with dichloromethane (DCM) and hexane (1:1) using an ASE 200 Accelerated Solvent Extractor (ASE) (Dionex, Oakville, ON, Canada). The ASE settings were 175 °C and 1500 PSI, with a preheat time of 7 min, a heat time of 5 min, and a static extract time of 10 min. The extracts were collected in vials containing 5 g of anhydrous sodium sulfate.

Extracts were filtered through 0.45 μm Whatman Teflon syringe filters, reduced to 0.5 mL under nitrogen at 30 °C, and brought up to 2 mL in DCM. Gel permeation chromatography (GPC), using a Waters Autopurification system with tandem Waters Envirogel GPC columns (19 × 300 mm and 19 × 150 mm, styrene/divinylbenzene) (Waters, Mississauga, ON, Canada) was used to remove high molecular weight compounds. The GPC was performed and calibrated according to EPA Method 3640a (*28*).

Gas Chromatography–Mass Spectrometry. A solvent exchange to hexane was performed, and the dust extracts were analyzed for 13 PAHs including acenaphthylene, fluorene, phenanthrene, anthracene, pyrene, benz[*a*]anthracene, benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, chrysene, indeno[1,2,3-*c,d*]pyrene, dibenz[*a,h*]anthracene, and benzo[*g,h,i*]perylene via gas chromatography/mass spectrometry (GC/MS). Analyses were conducted using a Hewlett-Packard 5890 gas chromatograph (GC) fitted with an HP-5MS capillary column (30 m × 0.25 mm × 0.25 μm film thickness) (J&W Scientific, Folsom, CA) and equipped with a HP5972 mass selective (MS) detector (Agilent Technologies, Palo Alto, CA). The initial GC temperature was held at 50 °C for 2 min, then ramped 8 °C min^{-1} to 300 °C, and held at 300 °C for 12 min. The injection port temperature was 270 °C, and the detector temperature was 280 °C. One microliter volumes were injected in splitless mode. The purge time was 2 min. The MS was operated in the selected ion monitoring mode. The masses monitored included the molecular ions and their associated characteristic fragment ions. A calibration standard consisting of a standard solution of 13 PAHs (EPA 525 PAH Mix A, Supelco, PA) and two deuterated PAHs (acenaphthene $d_{10}$and benzo[*a*]pyrene d_{12}) were run with each batch of samples. Prior to GC/MS analyses, each sample was spiked with two deuterated standards (acenaphthene-d_{10} and benzo[*a*]pyrene-d_{12}) that permit the normalization of GC/MS peak area. Identification of the PAHs was based on their retention time relative to the calibration standard solution. Quantification of the PAHs, based on the internal standards, was conducted using MSD Productivity ChemStation (*29*).

QA/QC. A recovery efficiency study was conducted (N = 4) using 15 μg of the EPA 525 PAH Mix A, and recovery efficiencies ranged between 57 and 76%. All PAH concentration values were corrected for the recoveries. Empty cells were included as blanks with each batch of samples that was processed. No detectable amounts of PAHs were found in any of the blanks. Six randomly selected dust samples (10% of the samples) were subjected to duplicate analysis. The average relative percent difference (RPD) for the PAHs ranged from 3.4% to 9.5%. The method detection limit (MDL) in this study was defined as the instrument detection limit (IDL), which was calculated from repeats of low concentration standards, divided by the recoveries (MDL = IDL/recovery). The MDLs, recovery efficiencies and average RPD in duplicates are summarized in Table 1.

Cancer Risk Assessment of PAHs in Settled House Dust. The following equation was used to estimate the excess lifetime cancer risks associated with nondietary ingestion of PAHs in SHD during preschool years (*30*)

$$\text{lifetime cancer risk} = \sum_{i=1}^{n}\left(\frac{(C_i \times \text{PEF}_i) \times \text{IR} \times \text{EF} \times \text{SF} \times \text{AF}}{\text{BW} \times 1000}\right)$$

for B2 PAHs 1 through n, where C = concentration ($\mu g\ g^{-1}$) of each carcinogenic PAH in the SHD samples. The PAHs included in this assessment were benzo[*a*]anthracene (BaA), benzo[*a*]pyrene (BaP), benzo[*b*]fluoranthene (BbF), benzo[*k*]fluoranthene (BkF), chrysene (CHRY), dibenz[*a,h*]anthracene (DBahA), and indeno[1,2,3-*c,d*]pyrene (I123cdP).

TABLE 2. Minimum, Maximum, and Mean Values of 13 PAHs Measured in the Extracts of Settled House Dust Collected from Homes in Ottawa, ON[a]

PAH	MDL[b] (μg g^{-1})	no. of samples below MDL	minimum (μg g^{-1})	maximum (μg g^{-1})	median (μg g^{-1})	arithmetic mean (μg g^{-1})	SEM[c]	geometric mean (μg g^{-1})
acenaphthylene	0.011	30	0.005	0.171	0.005	0.039	0.007	0.015
fluorene	0.013	7	0.007	1.37	0.093	0.170	0.032	0.084
phenanthrene	0.012	0	0.149	21.0	1.48	2.78	0.558	1.53
anthracene	0.011	2	0.006	6.62	0.196	0.485	0.136	0.222
pyrene	0.012	0	0.207	46.0	1.46	4.36	1.15	1.91
benz[*a*]anthracene	0.021	0	0.105	32.1	0.696	2.38	0.707	0.956
chrysene	0.025	0	0.150	35.1	1.19	3.29	0.858	1.46
benzo[*b*]fluoranthene	0.019	0	0.160	54.0	1.66	4.87	1.31	2.01
benzo[*k*]fluoranthene	0.034	0	0.049	19.0	0.532	1.60	0.442	0.674
benzo[*a*]pyrene	0.051	0	0.040	38.8	0.803	2.91	0.899	0.963
indeno[1,2,3-*c,d*]pyrene	0.039	0	0.100	33.5	0.911	3.07	0.819	1.29
dibenz[*a,h*]anthracene	0.054	0	0.022	6.27	0.185	0.549	0.148	0.250
benzo[*g,h,i*]perylene	0.034	0	0.118	31.4	0.793	2.79	0.764	1.13
total PAHs[d]			1.50	325	9.53	29.3	7.78	12.9
B2 PAHs[e]			0.656	219	6.06	18.7	5.17	7.68

[a] Values are corrected for recovery efficiencies. [b] Method detection limit. Samples below the MDL were assigned a value of one half of the MDL. [c] Standard error of the arithmetic mean. [d] Sum of the 13 targeted PAHs. [e] Sum of the PAHs classified as probable human carcinogens by the U.S. EPA (*31*).

All of these PAHs are categorized as probable human carcinogens (B2) on the basis of U.S. EPA classifications (*31*). PEF = Potency equivalency factor. These factors are applied to the individual PAH concentrations to express the potency of each PAH in terms of benzo[*a*]pyrene. The PEFs were BaA = 0.1, BbF = 0.1, BkF = 0.1, CHRY = 0.001, I123cdP = 0.1, DBahA = 5. All PEFs were taken from Collins et al. (*32*), except for DbahA, which was taken from Nisbet and Lagoy (*33*). IR = Daily ingestion rate of dust (g day^{-1}). Three ingestion rates were considered: 0.01, 0.05, and 0.1 g day^{-1}. Investigators estimate that children ingest between 0.05 and 0.1 g of dust per day depending on the season and the amount of time spent indoors (*34*). Both 0.05 and 0.1 g day^{-1} are considered to be conservative estimates, erring on the side of greater exposure. On the basis of studies with tracer elements, other researchers have suggested that children likely ingest closer to 0.04 g day^{-1} of soil and dust combined (*35*). Dust is estimated to account for only a quarter of this value (*35, 36*). Consequently, a lower ingestion rate of 0.01 g day^{-1} was also used. EF = exposure factor. The average proportion of a seventy year lifetime that preschoolchildren are exposed to dust via nondietary ingestion. Seven hours per day was considered an average exposure rate based on the fact that preschool-aged children spend approximately 19–20 h day^{-1} indoors (*37, 38*) and sleep for approximately 12–13 h day^{-1} (*38, 39*). It was assumed that preschool-aged children would be exposed from birth up to the fifth birthday. BW = average body weight (kg). A standard value of 13 kg was used (*40*). SF = slope factor ((mg kg^{-1} day^{-1})$^{-1}$). This is the estimate of the probability of a response occurring per unit intake of the PAH over a lifetime. For these analyses, an oral slope factor for benzo[*a*]pyrene of 7.3 (mg kg^{-1} d^{-1})$^{-1}$was used (*31*). Slope factors represent the upper-bound estimate of risk per unit dose for an average population (*41*). AF = adjustment factor. This factor accounts for exposures taking place during early life stages when children are more susceptible to the effects of chemical toxins (*26*). For exposure to carcinogens with a mutagenic mode of action, the U.S. EPA recommends an adjustment factor of 10 for children less than 2 years of age and an adjustment factor of 3 for children between 2 and 15 years of age (*42*). Therefore, a composite adjustment factor of 5.8 was used for this risk assessment where exposures occur from birth to 5 years of age.

Data Analyses. All descriptive statistics (e.g., minimum, maximum, mean), ordinary least-squares linear regression, and Pearson correlations were performed using the SAS System, version 8.2, for Windows (*43*). PAH concentration data, as well as the data for two variables contained in the homeowner survey (vacuum frequency and number of people living in the house), were log transformed to equalize the variance across the range of observations. The Shapiro−Wilk statistic and inspection of normal probability plots were used to assess normality of residuals. Significant outliers were identified by calculation of the studentized deleted residual (*44*). In those cases where PAH values were below the detection limit, a value of one-half the detection limit was substituted into the data set for statistical analyses.

Results and Discussion

Levels of Polycyclic Aromatic Hydrocarbons (PAHs). The dust extracts were evaluated for the presence of 13 targeted PAHs (Table 2). The individual concentrations of the 13 PAHs spanned between 2 and 3 orders of magnitude both for a single PAH and between different PAHs. Acenaphthylene was detected in the lowest concentrations, while benzo[*b*]fluoranthene was detected in the highest. With a molecular weight of 152.2 and a vapor pressure of 0.378 Pa (*45*), acenaphthylene is more readily volatilized and generally found only at low levels in dust samples (*23, 46–48*). In contrast, benzo[*b*]fluoranthene is heavier and less volatile and has previously been detected in the highest concentrations in dust samples (*47, 49*). Although PAHs with a higher number of rings generally have lower volatilities, there were no consistent trends in concentration of the PAHs based on the number of rings.

The sum of the 13 PAHs, referred to hereafter as total PAHs, ranged between 1.5 and 325 μg g^{-1}, with a geometric mean of 12.9 μg g^{-1}. These values are similar to the findings of a previous review in which the total PAHs for samples collected from urban, rural, and suburban homes ranged between 0.4–544 μg g^{-1} with a geometric mean of 4.5 μg g^{-1} (*10*). The distribution of the total PAHs in the samples was positively skewed (skewness = 4.00).

The sum of the seven PAHs classified as probable human carcinogens by the U.S. EPA (2003), referred to hereafter as the B2 PAHs, accounted for approximately 60% of the total PAHs. Similar results were observed in other studies where the concentrations of the B2 PAHs, were shown to be approximately one-half of the total PAH concentrations (*24, 50*).

The house with the lowest total PAH concentration (1.5 μg g^{-1}) was a newly constructed home whose owners moved

in shortly before the sampling took place. The house with the highest total PAH level (325 μg g^{-1}) was an 18-year-old residence whose only distinguishing feature was that it was 90% carpeted. Lewis et al. (1994) noted that contaminants have the potential to accumulate in carpet dust (*21*), and therefore, a high percentage of carpeting in this home may have resulted in high PAH levels.

The German Federal Environmental Agency's Commission for Indoor Air Quality has established the only current guideline for PAHs in house dust. It states that exposure to concentrations above 10 μg of benzo[*a*]pyrene per gram of household dust should be minimized to prevent adverse health effects (*51*). In the present study, three samples (11% of total) contained concentrations of benzo[*a*]pyrene that were above 10 μg g^{-1} (ppm), with maximum values reaching 39 μg g^{-1}. Although there are currently no Canadian guidelines for PAH contaminants in SHD, guidelines for PAHs in residential soils, a comparable particulate matrix, do exist. The 2003 Canadian Environmental Quality Guideline for benzo[*a*]pyrene in residential soil is 0.7 μg g^{-1} (ppm) (*52*). More than half of the SHD samples examined in this study contained concentrations above this value. The Environmental Quality Guideline for benzo[*b*]fluoranthene in residential soils is 1 μg g^{-1}. The geometric mean value for benzo[*b*]fluoranthene in SHD was 2 μg g^{-1}, and the maximum value was 54 μg g^{-1}. Similarly, other PAHs were also observed to exceed Canadian Environmental Quality Guidelines for residential soils.

Empirical Relationships Between Dust PAH Content and Household Attributes. Empirical relationships between dust PAH content and the attributes of the homes from which the dust was collected (see Table 1, Supporting Information) were investigated. Previous studies have shown that PAH concentrations in the indoor environment are related to PAH source activities such as smoking (*15, 24*), cooking (*13*), fireplace and woodstove use (*53, 54*), and urban location (*10*). However, in the present analyses, no significant relationships were observed between dust PAH concentrations and the PAH sources identified in the homeowner survey. This was unexpected because an earlier review by our group, based on 132 observations from 18 publications (*10*), showed that both cigarette smoking and an urban home location were weakly but significantly related to PAH contamination of dust. The absence of empirical relationships in the present study may be the result of a relatively small sample size.

Relationships were also evaluated between PAH concentrations and other survey variables (i.e., not combustion related). Weak, but significant, negative relationships ($r = -0.29$ to -0.32, $p < 0.05$) were found between the concentrations of PAHs with four rings or more and the frequency of vacuuming. This finding suggests that the cleaning habits of the inhabitants in some way reduces the PAH concentration of the dust. It is possible that homeowners who vacuum more frequently do so because their homes are noticeably dustier. Dustier homes may contain more diluted PAH concentrations. Alternatively, vacuum frequency may be an indicator of general cleanliness, and "cleaner" residents may engage in activities that reduce the likelihood of PAH contamination in the indoor area (e.g., removal of shoes, more ventilation during PAH generating events).

Cancer Risk Assessment of PAHs in Settled House Dust. To assess the potential consequences associated with exposure to carcinogenic PAHs, a risk assessment was conducted using the concentration data for PAHs classified as probable human carcinogens (B2) by the U.S. EPA (*31*). Specifically, the risk assessment evaluated the excess lifetime cancer risk resulting from nondietary ingestion of carcinogenic PAHs in SHD during preschool years.

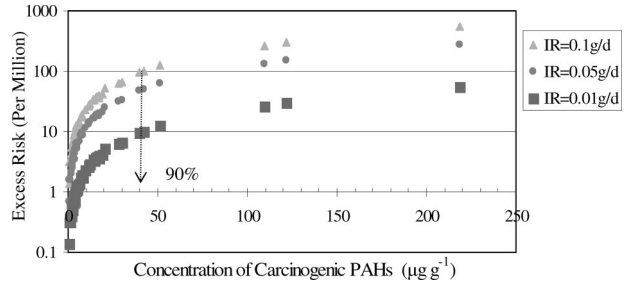

FIGURE 1. Excess cancer risks resulting from nondietary ingestion of B2 PAHs in SHD during preschool years. Three ingestion rates (IR) are considered. Arrow denotes the 90th percentile of the B2 PAH concentrations.

TABLE 3. Comparisons of Levels of Excess Cancer Risk Resulting from the Nondietary Ingestion of PAHs in Settled House Dust

	ingestion rate = 0.05 g day^{-1}		ingestion rate = 0.1 g day^{-1}	
PAH concentration	Roberts et al.[a]	this study	roberts et al.[a]	this study
0.97 μg g^{-1}	7.8×10^{-6}	1.6×10^{-6}	1.6×10^{-5}	3.2×10^{-6}
4.2 μg g^{-1}	3.4×10^{-5}	5.1×10^{-6}	6.8×10^{-5}	1.0×10^{-5}
21 μg g^{-1}	1.7×10^{-4}	2.6×10^{-5}	3.4×10^{-4}	5.1×10^{-5}

[a] Values taken from ref *2*.

Figure 1 shows the levels of excess cancer risk plotted against the concentration of B2 PAHs found in each house dust sample. Risk curves are shown for low, medium, and high dust ingestion scenarios. The results indicate that exposure to carcinogenic PAHs at levels found in 90% of the sampled households (i.e., <40 μg g^{-1}) results in excess cancer risks that are generally between 1×10^{-6} and 1×10^{-4}. Five of the house dust samples contained levels of carcinogenic PAHs that were higher than 40 μg g^{-1}. Examination of the household survey results for these homes did not reveal any distinguishing characteristics that could account for the higher PAH levels.

To interpret the outcome of the risk assessment, the results can be evaluated against "acceptable risk" levels. One cancer case per million people is commonly used as a baseline level of acceptable risk. However, depending on exposure scenarios, agencies assessing risk frequently build upon this level and adopt ranges of acceptable risk. When the risk assessment outcomes of the present study are compared to the Canadian maximum acceptable level of risk (i.e., 1×10^{-5}) (*55*), the interpretation varies substantially according to ingestion rate. If the lowest ingestion rate is considered, only 5 homes (10%) contain PAH levels that result in unacceptable risk. If the middle ingestion rate is considered, 21 homes (41%) contain levels of carcinogenic PAHs (>8 μg g^{-1}) resulting in unacceptable risk. Similarly, at the highest ingestion rate, 34 homes (67%) contain levels of carcinogenic PAHs (>4.2 μg g^{-1}) resulting in unacceptable risk.

These results can be compared to a previously conducted assessment by Roberts et al. which also evaluated the lifetime cancer risks associated with the ingestion of PAHs in SHD (*2*). The exact number, type, and proportion of individual PAHs that made up the total PAH concentration in the Roberts et al. study are unknown; however, general comparisons can still be made with houses in the present study that had similar levels of total carcinogenic PAHs. The results in Table 3 show that the risk values calculated in the Roberts et al. study are consistently higher (approximately six times higher) than in the present study. The higher values may be partially

TABLE 4. Comparison of Gastric Cancer Incidence and Mortality in Canada with Calculated Risk Estimates

statistic	per 100 000
new cases of stomach cancer in Canada (2001 raw values)[a]	12.8
age-adjusted incidence of stomach cancer in Canada (2001)[a]	16.8
age-adjusted mortality from stomach cancer in Canada (2002)[a]	11.0
excess risk at 50th percentile PAH concentration for 0.01 g day^{-1}ingestion	0.1
excess risk at 50th percentile PAH concentration for 0.05 g day^{-1}ingestion	0.7
excess risk at 50th percentile PAH concentration for 0.10 g day^{-1}ingestion	1.3
excess risk at maximum PAH concentration for 0.01 g day^{-1}ingestion	5.5
excess risk at maximum PAH concentration for 0.05 g day^{-1}ingestion	27.4
excess risk at maximum PAH concentration for 0.10 g day^{-1}ingestion	54.9

[a] Cancer statistics from Canadian Cancer Society/ National Cancer Institute of Canada (*57*). Values are totals that include both sexes. Values for total new cases is incidence per 100 000 adults 20 years or older. Population statistics from Statistics Canada (*58*).

accounted for by the use of a higher slope factor (i.e., 11.3 instead of 7.3).

The BaP slope factor employed for the risk assessment calculations in this study represents the risk of gastric cancer following dietary ingestion (*56*). Therefore, it is also useful to compare the excess lifetime risk values calculated in the present study with incidence and mortality rates of gastric cancer in Canada (Table 4).

The data show that exposures to SHD in houses where the PAH concentration is at the 50th percentile will result in essentially a negligible risk. However, the risk associated with the highest ingestion rate and the most contaminated sample is more than 3-fold greater than the age-adjusted incidence. Therefore, although excess risk of this magnitude would be relatively infrequent, concern is certainly justified.

As with all risk assessments, the calculation of risk involves a number of assumptions and uncertainties that have the potential to influence the outcome of the assessment. Future estimates of risk would benefit from more accurate, age-specific estimates of dust ingestion rates, including in relation to dust loading. In addition, since human exposure to compounds such as PAHs occurs in mixtures, additional information on the cumulative (geno)toxicity of compounds in real mixtures would provide a more accurate indication of actual risk.

This work, which characterized the PAH contamination of SHD, is part of a larger effort investigating the contamination, mutagenic activity and carcinogenic risk of SHD collected from homes in Ottawa, Canada. Specifically, this portion of the study assessed the levels of 13 priority PAHs, investigated empirical relationships between household attributes and PAH levels, and calculated the excess cancer risk posed by the detected carcinogenic PAHs to preschool children. Not surprisingly, the distribution of total PAH concentration was heavily skewed, and most (e.g., 90%) SHD samples contained less than 40 μg g^{-1}. Moreover, the geometric mean PAH concentration (12.9 μg g^{-1}) was in the same range as those described in other house dust studies (*10*). Empirical analyses failed to detect relationships between PAH concentration and combustion activity, and the source(s) of the detected PAHs remain unclear.

It should be noted that this study only investigated PAH contamination and excess risk posed by seven carcinogenic PAHs, and toxicity data is lacking on PAHs with higher molecular weights (i.e., larger than BaP). It is likely that SHD, a highly complex environmental matrix, contains dozens, perhaps even hundreds, of chemical contaminants that can contribute to the risk of adverse health effects. It would prove interesting to use a nontargeted approach to investigate the toxicological activity of chemical fractions derived from SHD. Moreover, a strategy such as effect-directed fractionation (*59*) could be employed to track and eventually identify toxic substances in highly complex environmental matrices such as SHD. In our companion paper (*60*), we use a nontargeted, bioassay-based approach to investigate the mutagenic activity of SHD extracts.

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Supporting Information Available

Table containing additional details regarding the topics included in the Health Canada Indoor Air Study of November 2002−March 2003. Survey questions were abbreviated and include only those relevant to settled house dust. This material is available free of charge via the Internet at http://pubs.acs.org.

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