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# Polychlorinated biphenyls in domestic dust from Canada, New Zealand, United Kingdom and United States: Implications for human exposure

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ABSTRACT

Ingestion of indoor dust has been highlighted as an important pathway of exposure to brominated flame retardants. Hence, polychlorinated biphenyls (PCBs) were determined in indoor dust from homes in Amarillo/Austin, TX, USA ($n = 20$; median concentration = 200 ng ΣPCB $g^{-1}$); Birmingham, UK ($n = 20$; 48 ng ΣPCB $g^{-1}$); Toronto, Canada ($n = 10$; 260 ng ΣPCB $g^{-1}$); and Wellington, New Zealand ($n = 20$; 46 ng ΣPCB $g^{-1}$). Concentrations in Canadian and US samples were statistically indistinguishable, but exceeded significantly ($p < 0.05$) those in both New Zealand and UK dust. Principal component analysis revealed that while UK samples were enriched comparatively in lower molecular weight congeners; samples from other countries contained proportionally more mid-to-high molecular weight congeners. Concentrations of PCBs determined in air from the same 10 Canadian homes showed concentrations (median = 4.9 ng ΣPCB $m^{-3}$) higher than those reported previously for UK homes (1.8 ng ΣPCB $m^{-3}$). Interpretation of these data alongside that for dietary exposure from other studies suggest that indoor exposures (i.e. air and dust combined) may be a significant contributor to overall exposure for the majority of the population – ranging from 4.3% to 87% in adults and 1.6–73% in toddlers. While inhalation is the principal indoor pathway under a typical dust ingestion scenario, exposure via dust ingestion exceeds that from either inhalation or diet for a small proportion of North American toddlers.

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## 1. Introduction

Polychlorinated biphenyls (PCBs) have found widespread use in a diverse range of applications, with around 1.2–1.3 million t produced worldwide (Harrad et al., 1994; Breivik et al., 2002). Of this, around 640 000 t were produced in the USA, approximately 67 000 and 40 000 t were produced and used, respectively, in the UK, while 40 000 t were imported into Canada (McDonald and Tourangeau, 1986; Harrad et al., 1994; Breivik et al., 2002). There are no reliable estimates of the quantity of PCBs imported into New Zealand, but it is likely to be appreciable given that between 1987 and 1998 an estimated 1300–1600 t were exported from New Zealand for destruction (New Zealand MoE, 1998). Owing to concerns about their adverse effects on humans and wildlife, their production – but not their use – ceased in the UK and throughout most of the industrialised world in the late 1970s. Although the majority of non-occupational exposure to PCBs has been widely considered to occur via the diet (Duarte-Davidson and Jones, 1994), there have been increasing indications that indoor air remains contaminated by PCBs remaining in use in applications such as permanently elastic sealants and acoustic ceiling tiles (Benthe et al., 1992; Balfanz et al., 1993; Herrick et al., 2004; Heinzow et al., 2004; Kohler et al., 2005). As a result, inhalation of indoor air could constitute a significant exposure pathway (Harrad et al., 2006). Furthermore, recent reports that ingestion of indoor dust is a potentially important pathway of exposure to some brominated flame retardants (Jones-Otazo et al., 2005; Harrad et al., 2008), raises similar concerns for PCBs.

This study reports concentrations of tri- through heptachlorinated PCB congeners in indoor dust taken from homes in Austin and Amarillo, TX, USA ($n = 20$); Birmingham, UK ($n = 20$); Toronto, Canada ($n = 10$); and Wellington, New Zealand ($n = 20$). In addition, concentrations of PCBs are reported for 10 air samples (taken using PUF disk passive samplers) in the same 10 Canadian homes from which dust was taken. These data are combined and compared with existing data on dietary and inhalation exposure to PCBs, to provide a preliminary indication of the relative significance of dust ingestion, inhalation and diet to overall human exposure to PCBs for adults and toddlers in Canada, New Zealand, the UK, and the USA.

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Our principal objectives were:

- to augment significantly the worldwide database on concentrations of PCBs in indoor dust,
- to evaluate the significance of dust ingestion as a pathway of exposure to PCBs of adults and toddlers, relative to dietary and inhalation exposure.

## 2. Materials and methods

### 2.1. Sampling methods

Dust samples were collected from homes in each city using a Nilfisk Sprint Plus 1600 W vacuum cleaner or equivalent model available in the country sampled. Sampling was conducted in January–March 2006 (New Zealand), July–August 2006 (UK), September 2006 (Canada), and October 2006 (USA); the homes selected in each city comprised a convenience sample of acquaintances of the authors. Sampling was conducted according to a clearly-defined standard protocol by one of the research team, with the exception of New Zealand, where home-dwellers took samples themselves according to this same protocol. One square meter of carpet was vacuumed for 2 min in each location and in case of bare floors 4 $m^2$ for 4 min. Samples were collected using nylon sample socks (25 μm pore size) that were mounted in the furniture attachment tube of the vacuum cleaner. After sampling, socks were closed with a twist tie, sealed in a plastic bag and stored at −20 °C. Before and after sampling, the furniture attachment was cleaned thoroughly using an isopropanol-impregnated disposable wipe.

### 2.2. Passive air sampling

In the same 10 Canadian homes sampled for dust, passive air samplers (i.e. PUF disks) were employed to provide a time-integrated sample over each 28 d sampling period. These have been used successfully in other studies of POPs in indoor air (Harrad et al., 2006). Samplers (each comprising one shelter each fitted with one PUF disk) were deployed in homes at a height of approximately 1 m above the floor, supported by a customised stainless steel cradle that permitted air flow to all sides of the sampler. Each PUF disk measured 14 cm in diameter and 1.2 cm in thickness, giving a surface area of 360 $cm^2$, and density 0.01685 g $cm^{-3}$. Disks were sheltered by two different size stainless steel housings (18 cm, 1 L bottom housing and 23 cm, 2 L top housing, respectively). Before deployment disks were washed thoroughly with tap and distilled water and then sequentially extracted (Soxhlet) for 16 h with hexane and dichloromethane, to remove target or interfering compounds. Following extraction, disks were desiccated to remove solvent and stored in pre-cleaned foil in air-tight solvent-cleaned glass jars. On deployment, disks were removed from the jars and transferred into the shelters and spiked with known quantities of PCBs 19 and 147 as QA/QC standards to provide a measure of contaminant loss during sampling. At the end of each sampling period, disks were removed from shelters and stored in solvent-cleaned aluminium foil in air-tight glass jars at −20 °C until extraction.

Conversion of contaminant masses per sample into concentrations in air requires knowledge of the air sampling rate of the PUF disk samplers and their deployment time. We used sampling rates reported previously from a specific calibration exercise conducted using an identical sampler configuration in an office (Hazrati and Harrad, 2007). To summarise, homologue group-based sampling rates in the range 0.70–1.27 $m^3$ $d^{-1}$ were employed because sampling rates for individual PCB congeners of the same homologue group were very similar.

### 2.3. Analytical protocols

Analysis of all dust samples was conducted at the University of Birmingham, using the following methodology based on that used previously (Ayris et al., 1997) with minor modifications. Our method quantifies all PCBs containing between three and seven chlorines. Hence in this study, ΣPCB represents the sum of all tri-through heptachlorinated PCBs. In summary, dust samples (1 g) were treated with appropriate quantities of PCB internal/surrogate standards (PCB #s: 34, 62, 119, 131, and 173) before extraction using hexane with an accelerated solvent extraction (ASE) system (ASE 300, Dionex), using a 66 mL cell filled from the bottom with: Florisil (1.5 g), sample, and Hydromatrix (Varian Inc.). The extraction conditions were: temperature: 150 °C, pressure: 1500 psi, heat time: 7 min, static time: 5 min, flush volume: 50%, purge time: 100 s, static cycles: 1.

Following extraction, crude extracts were concentrated to approximately 2 mL, treated with 2 mL concentrated sulfuric acid, subjected to liquid:liquid back extraction using dimethyl sulfoxide (DMSO), prior to elution through a column containing 1 g Florisil (Aldrich Chemicals; 60–120 mesh, pesticide grade) topped with 1 g anhydrous sodium sulfate with 20 mL hexane. The eluate was reduced to incipient dryness, prior to addition of 20 μL of nonane containing 10 ng each of PCBs 29 and 129 as recovery determination (or syringe) standards, which are used to determine the recoveries of internal/surrogate standards for QA/QC purposes. PCB analyses were conducted on a Fisons' MD-800 GC/MS system fitted with a 60 m VF5 MS column (0.25 mm id, 0.25 μm film thickness). Both injector and interface temperatures were 280 °C. The oven temperature program for PCBs was: 140 °C for 2 min, 5 °C $min^{-1}$ to 215 °C and held for 5 min, then 2 °C $min^{-1}$ to 280 °C and held for 15 min. The mass spectrometer was operated in EI+ SIM mode; with monitored $m/z$ values as reported previously (Ayris et al., 1997).

To ensure accurate and precise measurement, peaks were only accepted if the following criteria were met:

- Signal to noise ratios for the least abundant ion exceeded 3:1.
- Peaks eluted within 5 s of standards run in the same batch as the samples.
- Isotope ratios for peaks were within 20% of those obtained for standards run in the same batch as the samples.

Method blanks ($n$ = 12) for the dust analysis procedures consisting of sodium sulfate were found to contain concentrations of target PCBs no greater than 5% of the concentrations found in the corresponding samples. Our data are thus not corrected for blank concentrations.

### 2.4. Determination of PCBs in indoor air samples

Analysis of all air samples was conducted at the University of Toronto. Field blanks ($n$ = 3) consisting of a PUF disk (treated in identical fashion to those used for sampling, except that no air was aspirated through them), and method blanks ($n$ = 3) (i.e. as field blanks but PUF disks were not transported to/fro sampling site) were found to contain concentrations of target PCBs no greater than 2.5% of the concentrations found in the corresponding samples. Our data are thus not corrected for blank concentrations.

All air samples were treated with the same suite of internal standards as dust samples, and extracted by soxhlet for 16 h with dichloromethane. Following extraction, crude extracts were solvent exchanged to hexane, washed with 2 mL of concentrated sulfuric acid and cleaned up using a column composed of 8 g of activated alumina and 2 g of silver nitrate impregnated alumina. Following this the sample was reduced in volume to 25 μl prior to GC/MS analysis. This was conducted on a 6890 N/5975B Agilent GC/MS system fitted with a 60 m J&W DB5 column (0.25 mm id,

0.25 μm film thickness). The temperature programme and MS acquisition parameters were as described above for dust samples.

### 2.5. Statistical analysis

Statistical analysis of the data was conducted using Excel (Microsoft Office for Mac OS X) to generate descriptive statistics, with all other statistical procedures conducted using SPSS version 13.0 for Mac OS X. Where concentrations of a given congener were below detection limits, the concentration entered for statistical analysis was assumed to equal half the detection limit. The distribution of each data set was evaluated using both the Kolmogorov–Smirnov goodness of fit test and visual inspection. In contrast to the findings for recent surveys of brominated flame retardants in indoor dust (e.g. Harrad et al., 2008), the results revealed concentrations in all data sets to be normally distributed. Hence, *t*-test, ANOVA and post-hoc tests were performed on untransformed concentrations. Principal component analysis of congener patterns was conducted on the fractional contribution of each individual congener to ΣPCB in each sample.

### 2.6. Exposure assessment methods

For each of the countries studied, external exposure (i.e. assuming 100% absorption of all intakes) of both adults and toddlers to ΣPCBs via dust ingestion and inhalation of indoor air was estimated as follows. To estimate exposure via dust ingestion, we have used average adult and toddler dust ingestion figures of 20 and 50 mg $d^{-1}$, and high dust ingestion figures for adults and toddlers of 50 and 200 mg $d^{-1}$ (Jones-Otazo et al., 2005). We have then estimated various plausible dust ingestion exposure scenarios, using 5th percentile, median, arithmetic mean, and 95th percentile concentrations in the dust samples reported here. A similar approach has been taken to estimate a range of inhalation exposures for Canadians, assuming inhalation of the indoor air concentrations reported in Table 2 and daily adult and toddler respiration rates of 20 and 3.8 $m^3$ $d^{-1}$, respectively (Wilford et al., 2005). Inhalation exposures for the UK are taken from a previous study (Harrad et al., 2006). In the absence of relevant data on concentrations of PCBs in indoor air from New Zealand and the USA, coupled with the similarity between concentrations in house dust from New Zealand and the UK, and Canada and the USA; we have assumed exposure via inhalation in New Zealand to equal that in the UK, and that in the USA to be identical to that in Canada. With respect to dietary exposure in the USA, the most recent estimate based on data on foodstuffs from the early 1990s (Dougherty et al., 2000) appears substantially higher than that reported for Canada (Health Canada, 2008). Hence, we have assumed that dietary exposure in the USA is identical to that in Canada. Canadian dietary exposure was calculated using the most recent national data derived from the total diet study conducted in Vancouver in 2002 (Health Canada, 2008). We calculated that a 10 kg toddler ingests 7.22 ng ΣPCB $kg^{-1}$ body weight $d^{-1}$ (i.e. for the age group 1–4 years male and female), and that a 60 kg adult ingested 1.87 ng ΣPCB kg body weight $d^{-1}$ (i.e. for males in age group 40–64 years). Dietary exposures for New Zealand and UK adults are taken from previous total diet surveys (Wearne et al., 1996; New Zealand MoE, 1998), with those for toddlers in those countries assumed to equal 57% of adult exposures (Wilford et al., 2005).

## 3. Results and discussion

### 3.1. Concentrations of PCBs in dust from different countries

Table 1 summarises the concentrations of ΣPCBs and selected congeners in dust samples taken in this study from each of the four cities studied. Statistical analysis of concentrations via ANOVA reveals that while concentrations of ΣPCBs and congeners 28 + 31, 52, 101, 118, 138, 153, and 180 in Canadian and US dusts are statistically indistinguishable, dusts from both countries are significantly more contaminated ($p < 0.05$) than both New Zealand and UK dusts. No statistical difference was detected between concentrations in New Zealand and UK dust. There are very few data pertaining to the concentrations of PCBs in house dust, but those available are compared with those recorded in this study in Table 1. Regardless of sampling location, concentrations detected in this study exceed substantially those recorded in the most recent previous study of 31 house dust samples in Singapore (Tan et al., 2007), but in contrast, are well below those reported in earlier studies of house dust from northeastern USA (Vorhees et al., 1999; Rudel et al., 2003). The reason(s) for the lower concentrations reported for US house dust in this study are unclear. However, the differences may reflect one or more of the following: (1) differences in the ages of buildings sampled; (2) the fact that the Vorhees et al., 1999 study was conducted in a city with a known "hotspot" of PCB contamination (although the concentrations cited in Table 1 from that study were from "comparison neighbourhoods" distant from the hotspot); (3) regional variations in the use of PCBs; (4) a temporal decline in PCB contamination as PCB-contaminated building materials are replaced during renovations (the earlier studies were of samples taken in 1994–1995 (Vorhees et al., 1999) and 1999–2001 (Rudel et al., 2003)); (5) methodological differences in sampling and/or analysis between studies, or (6) could simply be due to the comparatively small sample numbers.

### 3.2. Congener patterns of PCBs in indoor dust – clues for source attribution

As well as international differences in concentrations of PCBs, we examined the data for differences in congener patterns via PCA. The first two principal components (PCs) accounted for 37% and 19%, respectively, of the total variance within the dataset. PC1 is driven in a positive direction by high proportions of mid- and high molecular weight congeners that predominate in the Aroclor 1254 and 1260 commercial formulations. In contrast, high factor scores for PC2 are caused by elevated proportions of tri- and tetrachlorinated PCBs; congeners that predominate in the Aroclor 1242 formulation. Fig. 1 plots the factor scores obtained for each sample entered into the PCA.

While the Canadian, New Zealand, and US samples in the main display positive factor 1 and negative or low factor 2 scores; the UK samples display lower scores for factor 1 and higher scores for factor 2. This suggests that while the UK dust samples are more influenced by Aroclor 1242-like congeners; dust from the other three countries are impacted more strongly by Aroclor 1254 and/or 1260-like congeners.

### 3.3. Concentrations of PCBs in domestic indoor air from Toronto

The concentrations of PCBs in air from Toronto homes are summarized in Table 2. For comparison, concentrations recorded in indoor air from 31 UK homes (Harrad et al., 2006), and in outdoor air from three urban Toronto locations are included also (Motelay-Massei et al., 2005). The indoor concentrations are elevated significantly compared to those reported outdoors previously. This is consistent with previous surveys in the UK that show a substantial indoor:outdoor increment (Currado and Harrad, 1998; Harrad et al., 2006). Concentrations in the Toronto indoor air samples were compared via a *t*-test with those reported previously for UK indoor air using identical PUF disk samplers.

**Table 1**
Summary of concentrations (ng ΣPCB $g^{-1}$) in domestic dust samples from selected countries.

| Location (reference)/statistical parameter | Congener | Minimum | 5th percentile | Median | Average | 95th percentile | Maximum |
|---|---|---|---|---|---|---|---|
| Amarillo, Austin, TX, US (this study) | 28 + 31 | 1.6 | 2.3 | 5.1 | 9.5 | 24 | 37 |
| | 52 | 1.7 | 2.0 | 6.2 | 7.9 | 17 | 28 |
| | 101 | 1.9 | 3.6 | 8.7 | 10 | 25 | 29 |
| | 105 | <dl | 0.89 | 2.6 | 4.5 | 14 | 20 |
| | 118 | 1.8 | 2.6 | 5.5 | 10 | 29 | 44 |
| | 138 | 1.1 | 1.7 | 6.5 | 8.6 | 28 | 31 |
| | 153 | 1.0 | 1.6 | 7.1 | 8.4 | 21 | 22 |
| | 180 | 0.71 | 0.84 | 2.6 | 4.5 | 14 | 20 |
| | ΣPCB | 47 | 67 | 200 | 220 | 520 | 620 |
| Birmingham, UK (this study) | 28 + 31 | 0.52 | 0.62 | 3.4 | 6.3 | 26 | 39 |
| | 52 | 0.26 | 0.41 | 1.8 | 5.6 | 15 | 53 |
| | 101 | 0.09 | 0.27 | 1.2 | 6.1 | 13 | 73 |
| | 105 | 0.02 | 0.08 | 0.41 | 1.9 | 3.1 | 24 |
| | 118 | 0.06 | 0.17 | 0.92 | 4.3 | 7.9 | 56 |
| | 138 | 0.05 | 0.18 | 1.1 | 4.1 | 8.8 | 50 |
| | 153 | 0.06 | 0.23 | 1.2 | 3.3 | 7.2 | 32 |
| | 180 | 0.06 | 0.21 | 0.89 | 1.8 | 6.7 | 8.1 |
| | ΣPCB | 5.7 | 9.0 | 48 | 110 | 270 | 860 |
| Toronto, Canada (this study) | 28 + 31 | 3.5 | 3.6 | 7.3 | 10 | 23 | 29 |
| | 52 | 3.4 | 3.5 | 7.2 | 12 | 35 | 37 |
| | 101 | 1.9 | 2.6 | 8.8 | 15 | 45 | 60 |
| | 105 | 0.49 | 0.67 | 4.4 | 7.1 | 20 | 23 |
| | 118 | 1.1 | 1.7 | 8.7 | 13 | 42 | 55 |
| | 138 | 1.0 | 1.8 | 9.5 | 12 | 34 | 49 |
| | 153 | 0.92 | 1.8 | 9.9 | 11 | 27 | 36 |
| | 180 | 0.47 | 1.1 | 6.8 | 8.5 | 21 | 24 |
| | ΣPCB | 56 | 65 | 260 | 290 | 720 | 820 |
| Wellington, New Zealand (this study) | 28 + 31 | 0.76 | 0.79 | 2.3 | 3.3 | 8.3 | 11 |
| | 52 | 0.43 | 0.54 | 1.4 | 2.6 | 9.9 | 13 |
| | 101 | 0.39 | 0.46 | 1.6 | 3.1 | 8.1 | 21 |
| | 105 | <dl | 0.13 | 0.43 | 0.87 | 2.2 | 5.3 |
| | 118 | <dl | <dl | 0.95 | 1.9 | 4.9 | 14 |
| | 138 | 0.27 | 0.44 | 1.8 | 2.8 | 8.6 | 11 |
| | 153 | 0.35 | 0.40 | 1.4 | 2.7 | 7.0 | 12 |
| | 180 | <dl | 0.24 | 1.3 | 2.3 | 5.7 | 9.6 |
| | ΣPCB | 11 | 13 | 46 | 67 | 154 | 260 |
| Boston, USA (Vorhees et al. (1999)) | ΣPCB | 260 | – | 710 | 690[a] | – | 3600 |
| Cape Cod, MA, USA (Rudel et al. (2003)) | 52 | <dl | – | <dl | 170 | <dl | 16000 |
| | 105 | <dl | – | <dl | 250 | <dl | 17000 |
| | 153 | <dl | – | <dl | 540 | 400[b] | 35000 |
| Singapore (Tan et al. (2007)) | 28 + 31 | <dl | – | 0.2 | 0.3 | – | 2.9 |
| | 101 | <dl | – | 0.5 | 0.6 | – | 2.2 |
| | 118 | <dl | – | 0.3 | 0.7 | – | 8.1 |
| | 153 | <dl | – | 0.5 | 0.8 | – | 5.5 |
| | 180 | <dl | – | 0.2 | 0.3 | – | 2.0 |
| | ΣPCB | <dl | – | 5.6 | 9.2 | – | 44 |

–, statistical parameter or congener not reported.
<dl, below detection limit.
[a] Geometric mean.
[b] 90th percentile.

Out of the congeners summarized in Table 2, concentrations of PCBs 52, 101, 118, 138, and ΣPCB were all significantly higher ($p < 0.05$) in Toronto than Birmingham domestic indoor air (Hazrati, 2006). Concentrations of PCBs 28 + 31, 153, and 180 were statistically indistinguishable. Although they should not be over-interpreted as only 10 Toronto homes were monitored; combined with the significantly higher PCB concentrations in Toronto compared to Birmingham dust, these data suggest that indoor contamination of Canadian homes exceeds that in the UK. Potential causes of this difference are: (a) greater PCB usage, (b) lower indoor:outdoor air exchange rates in Canada compared to the UK, and (c) a comparative oversampling in Canada compared to the UK of homes constructed during the peak period of PCB use (1960s–1970s). However, as the proportion of homes constructed during that period was roughly equal (~30%) in both the Toronto and Birmingham campaigns, the latter does not seem a likely explanation.

### *3.4. Exposure to PCBs via dust ingestion and inhalation of indoor air relative to each other and to dietary exposure*

Table 3 summarises estimated external exposure of both adults and toddlers to ΣPCBs via dust ingestion and inhalation of indoor air both in absolute terms and relative to each other and to dietary exposure. While we recognise that the relative significance of different exposure pathways will vary on a congener-specific basis (e.g. inhalation is likely to assume proportionally greater significance for PCB 28, while diet will likely be of greater importance for PCB 180), there were no suitable data available on dietary exposure on a congener-specific basis. It is stressed that the range of exposure estimates via dust ingestion and air inhalation thus derived are only an indication of the likely range within the population. No attempt has been made to estimate the range of dietary exposures, but we acknowledge that it will be appreciable. Furthermore, while the inhalation exposures for Canada (and

**Table 2**
Comparison of concentrations (pg ΣPCB $m^{-3}$) in domestic indoor air from Toronto (this study) with concentrations in Toronto outdoor air and in domestic indoor air from Birmingham, UK.

| Location (reference)/statistical parameter | Congener | Minimum | 5th percentile | Median | Average | 95th percentile | Maximum |
|---|---|---|---|---|---|---|---|
| Toronto (this study) | 28 + 31 | 130 | 140 | 580 | 1100 | 2400 | 2500 |
| | 52 | 100 | 120 | 610 | 630 | 1200 | 1200 |
| | 101 | 19 | 22 | 110 | 150 | 320 | 330 |
| | 118 | 10 | 15 | 35 | 55 | 110 | 120 |
| | 138 | 3.8 | 4.0 | 17 | 22 | 44 | 46 |
| | 153 | 3.2 | 3.9 | 21 | 24 | 48 | 49 |
| | 180 | <dl | <dl | 3.8 | 3.1 | 7.2 | 7.3 |
| | ΣPCB | 1100 | 1400 | 4900 | 6900 | 14200 | 14400 |
| Toronto outdoor air[a] (Motelay-Massei et al. (2005)) | 28 | 9.7 | 13 | 28 | 39 | 86 | 101 |
| | 52 | 19 | 21 | 69 | 71 | 149 | 165 |
| | 101 | 9.7 | 13 | 40 | 59 | 143 | 169 |
| | 118 | 3.9 | 5.5 | 20 | 24 | 57 | 61 |
| | 138 | 2.9 | 4.4 | 17 | 27 | 70 | 85 |
| | 153 | 3.2 | 5.4 | 21 | 31 | 75 | 96 |
| | 180 | 0.22 | 0.55 | 3.1 | 5.8 | 16 | 21 |
| | ΣPCB | 100 | 130 | 350 | 510 | 1200 | 1400 |
| Birmingham, UK indoor air, homes only (Hazrati, 2006) | 28 | 88 | 99 | 300 | 490 | 700 | 2100 |
| | 52 | 29 | 34 | 110 | 140 | 370 | 470 |
| | 101 | 4.0 | 6.0 | 28 | 33 | 68 | 140 |
| | 118 | 2.0 | 2.5 | 9.0 | 11 | 23 | 36 |
| | 138 | 0.25 | 1.0 | 2.0 | 3.2 | 7.0 | 15 |
| | 153 | 1.0 | 2.0 | 7.0 | 11 | 19 | 108 |
| | 180 | <dl | <dl | 1.0 | 2.1 | 5.0 | 20 |
| | ΣPCB | 490 | 590 | 1800 | 2800 | 8900 | 9800 |

[a] Based on concentrations reported for all three quarterly samples taken at sites Urban 1, 2, and 4.

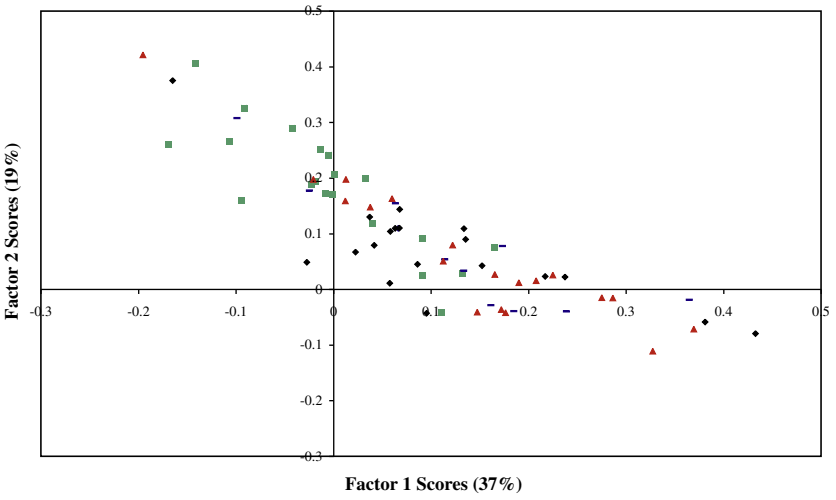


**Fig. 1.** Plot of factor scores for principal component 1 versus principal component 2 for dust samples. Canadian, New Zealand, UK, and US samples are marked in blue dashes, black diamonds, green squares, and red triangles, respectively.

the USA) are based on domestic air only; those for UK (and New Zealand) adults incorporate inhalation of 40 h $week^{-1}$ of office air – which has been shown to be significantly more contaminated with PCBs than domestic air in the UK (Harrad et al., 2006). It is likely therefore, that inhalation may be more important for Canada and the USA than indicated in Table 3. Within these constraints, Table 3 gives a useful indication of the range both of absolute magnitude and relative significance of indoor and dietary exposures for adults and toddlers, as well as international differences. Perhaps the most important overall point from these exposure estimate scenarios is that indoor exposures (dust ingestion and air inhalation combined) make a significant contribution to overall exposure, which may in fact exceed dietary exposures in some scenarios. The importance of indoor exposures is less marked for the UK owing to higher dietary exposure, and lower indoor air and dust concentrations than in Canada. More specifically, under the low dust ingestion scenarios for adults, it is evident that ingestion of dust contributes at most a few per cent of overall exposure in all countries studied, with inhalation and diet making fairly equal contributions – the former making the major contribution to those individuals frequenting more highly contaminated buildings. For toddlers, diet is the most important exposure pathway except for those inhaling air contaminated at the 95th percentile concentration in New Zealand, where the absolute magnitude of dietary exposure is lowest. Under the high dust ingestion scenarios, dust is still a relatively minor contributor to overall adult exposure. For toddlers however, dust ingestion is substantially more important, especially in Canada and the USA, where it represents the most significant pathway in the most contaminated homes.

**Table 3**
Summary of relative significance of exposure of Canadian, New Zealand, UK, and US adults and toddlers to ΣPCBs via dust ingestion, inhalation, and diet under various scenarios.

| Country | | Adult | | | | Toddler (6–24 months) | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | 5th percentile | Median | Average | 95th percentile | 5th percentile | Median | Average | 95th percentile |
| Magnitude of exposure (ng ΣPCB $d^{-1}$) (% contribution to overall exposure) | | | | | | | | | |
| *Mean dust intake scenario* | | | | | | | | | |
| Canada | Air | 28 (19.8%) | 99 (45.8) | 138 (53.9) | 285 (69.3) | 5.2 (6.2) | 19 (18.3) | 26 (23.0) | 54 (33.3) |
| | Dust | 1.3 (0.9) | 5.2 (2.4) | 5.8 (2.3) | 14 (3.4) | 3.2 (4.0) | 13 (12.5) | 15 (13.3) | 36 (22.2) |
| | Diet | 112 (79.3) | 112 (51.8) | 112 (43.8) | 112 (27.3) | 72 (89.8) | 72 (69.2) | 72 (63.7) | 72 (44.4) |
| New Zealand | Air | 15 (14.3) | 60 (39.8) | 150 (62.2) | 586 (86.3) | 2.8 (5.1) | 11 (17.1) | 28 (34.0) | 111 (65.4) |
| | Dust | 0.26 (0.2) | 0.91 (0.6) | 1.3 (0.5) | 3.1 (0.5) | 0.64 (1.2) | 2.3 (3.6) | 3.3 (4.0) | 7.7 (4.5) |
| | Diet | 90 (85.5) | 90 (59.6) | 90 (37.3) | 90 (13.3) | 51 (93.7) | 51 (79.3) | 51 (62.0) | 51 (30.1) |
| UK | Air | 15 (4.2) | 60 (15.0) | 150 (30.5) | 586 (62.9) | 2.8 (1.4) | 11 (5.3) | 28 (12.3) | 111 (34.8) |
| | Dust | 0.18 (0.1) | 0.95 (0.2) | 2.3 (0.5) | 5.4 (0.6) | 0.45 (0.2) | 2.4 (1.2) | 5.6 (2.5) | 14 (4.4) |
| | Diet | 340 (95.7) | 340 (84.8) | 340 (69.0) | 340 (36.5) | 194 (98.4) | 194 (93.5) | 194 (85.2) | 194 (60.8) |
| US | Air | 28 (19.8) | 99 (46.0) | 138 (54.2) | 285 (70.0) | 5.2 (6.2) | 19 (18.8) | 26 (23.9) | 54 (35.5) |
| | Dust | 1.3 (0.9) | 4.0 (1.9) | 4.4 (1.7) | 10 (2.5) | 3.3 (4.1) | 10 (9.9) | 11 (10.1) | 26 (17.1) |
| | Diet | 112 (79.3) | 112 (52.1) | 112 (44.0) | 112 (27.5) | 72 (89.7) | 72 (71.3) | 72 (66.1) | 72 (47.4) |
| *High dust intake scenario* | | | | | | | | | |
| Canada | Air | 28 (19.6) | 99 (44.2) | 138 (52.1) | 285 (65.8) | 5.2 (5.6) | 19 (13.3) | 26 (16.7) | 54 (20.0) |
| | Dust | 3.2 (2.2) | 13 (5.8) | 15 (5.7) | 36 (8.3) | 13 (14.4) | 52 (36.4) | 58 (37.2) | 144 (53.3) |
| | Diet | 112 (78.2) | 112 (50.0) | 112 (42.3) | 112 (25.9) | 72 (80.0) | 72 (50.3) | 72 (46.2) | 72 (26.7) |
| New Zealand | Air | 15 (14.2) | 60 (39.4) | 150 (61.7) | 586 (85.7) | 2.8 (5.0) | 11 (15.5) | 28 (30.4) | 111 (57.5) |
| | Dust | 0.64 (0.6) | 2.3 (1.5) | 3.3 (1.3) | 7.7 (1.1) | 2.6 (4.6) | 9.1 (12.2) | 13 (14.1) | 31 (16.1) |
| | Diet | 90 (85.2) | 90 (59.1) | 90 (37.0) | 90 (13.2) | 51 (90.4) | 51 (71.7) | 51 (55.4) | 51 (26.4) |
| UK | Air | 15 (4.2) | 60 (14.9) | 150 (30.3) | 586 (62.3) | 2.8 (1.4) | 11 (5.1) | 28 (11.4) | 111 (30.9) |
| | Dust | 0.45 (0.1) | 2.4 (0.6) | 5.6 (1.1) | 14 (1.5) | 1.8 (0.9) | 9.5 (4.4) | 23 (9.4) | 54 (15.0) |
| | Diet | 340 (95.7) | 340 (84.5) | 340 (68.6) | 340 (36.2) | 194 (97.7) | 194 (90.4) | 194 (79.2) | 194 (54.0) |
| US | Air | 28 (19.5) | 99 (44.8) | 138 (52.9) | 285 (67.4) | 5.2 (5.6) | 19 (14.5) | 26 (18.3) | 54 (23.5) |
| | Dust | 3.3 (2.3) | 10 (4.5) | 11 (4.2) | 26 (6.1) | 13 (14.4) | 40 (30.5) | 44 (31.0) | 104 (45.2) |
| | Diet | 112 (78.2) | 112 (50.7) | 112 (42.9) | 112 (26.5) | 72 (80.0) | 72 (55.0) | 72 (50.7) | 72 (31.3) |

Exposure estimates derived from PCB concentrations in domestic dust and Toronto domestic air (this study) except:
Canadian dietary exposure taken from Health Canada (2008) assuming 60 kg adult and 10 kg toddler.
New Zealand dietary exposure from New Zealand Ministry for the Environment (1998).
New Zealand inhalation exposures assumed to equal those for UK.
UK dietary exposure taken from Wearne et al. (1996).
UK inhalation exposures taken from Harrad et al., (2006).
US dietary and inhalation exposures assumed to equal those for Canada.
Air inhalation rates assumed to be 20 $m^3$ $d^{-1}$ (adults), 3.8 $m^3$ $d^{-1}$ (toddlers).
Mean dust ingestion rates assumed to be 20 mg $d^{-1}$ (adults), 50 mg $d^{-1}$ (toddlers).
High dust ingestion rates assumed to be 50 mg $d^{-1}$ (adults), 200 mg $d^{-1}$ (toddlers).
UK and New Zealand toddler dietary intakes assumed to equal 57% of adults.

Overall, the combination of the two indoor exposure pathways of dust ingestion and air inhalation delivers PCBs across the full range of chlorination, as well as adding substantially to the exposure received via diet. Fig. 2 shows how the proportional contribution of dust ingestion to overall indoor exposure (i.e. from dust ingestion and air inhalation combined) varies with Log$K_{ow}$ (Hawker and Connell, 1988), using Canadian toddler exposures for PCBs 28/31, 52, 101, 118, 138, 153, and 180 as an illustration. This reveals that dust ingestion contributes proportionally more to exposure to the higher molecular weight and generally less easily metabolized (Thomas et al., 2006) congeners with higher $K_{ow}$ values and lower vapour pressures than does inhalation of air; for which exposure is predominantly in the guise of the more volatile and more easily metabolised tri- and tetrachlorinated congeners. Hence, because dust delivers proportionally more of the less easily metabolized higher molecular weight congeners, dust ingestion may translate more effectively into human body burdens of ΣPCBs than the inhalation pathway. However, while inhalation delivers primarily lower molecular weight PCBs that are generally more easily metabolized; the health impacts of such PCB metabolites and thus exposures arising from the inhalation of indoor air are as yet unclear.

This study augments significantly our knowledge of the extent of contamination with PCBs of domestic dusts in Australasia, Europe, and North America. The evidence presented here adds to the weight of evidence that even three decades since the introduction of restrictions on the manufacture and new use of PCBs, they

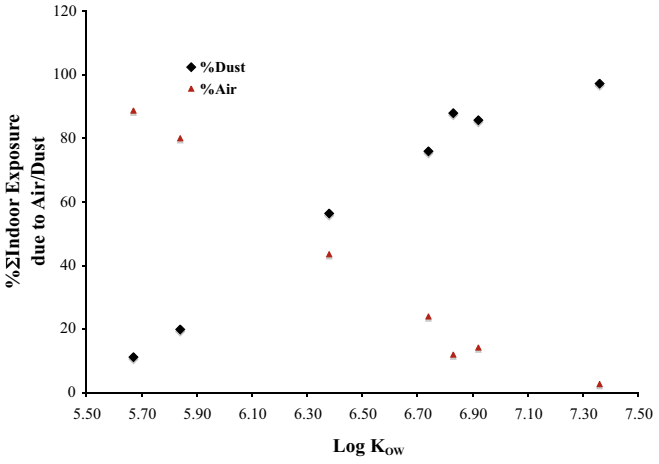


**Fig. 2.** Plot of the relative contribution of dust ingestion and air inhalation to overall Canadian toddler "Indoor" exposures (expressed as a percentage of the sum of dust and air exposures) versus Log$K_{ow}$ for PCBs 28/31, 52, 101, 118, 138, 153, and 180. Dust ingestion calculated using average concentrations and a dust ingestion rate of 50 mg $d^{-1}$. Log$K_{ow}$ values taken from Hawker and Connell (1988).

remain in our homes and diet leading to appreciable exposures. These findings underline the importance of on-going international efforts to eradicate PCBs and suggest that similar efforts may be required if the legacy of more recently-controlled chemicals with significant indoor applications, such as PBDEs and perfluorinated substances, is not to be similarly long-lasting.

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