



Cite this: *Environ. Sci.: Processes Impacts*, 2014, **16**, 2758

# Polychlorinated biphenyl (PCB) and dioxin concentrations in residential dust of pregnant women†

A. L. Hinwood,<sup>\*a</sup> A. C. Callan,<sup>a</sup> J. Heyworth,<sup>b</sup> D. Rogic,<sup>c</sup> J. de Araujo,<sup>c</sup> R. Crough,<sup>c</sup> G. Mamahit,<sup>c</sup> N. Piro,<sup>c</sup> A. Yates,<sup>c</sup> G. Stevenson<sup>c</sup> and J. Ø. Odland<sup>d</sup>

Polychlorinated biphenyls (PCBs) and dioxins are well known for their persistence in the environment. PCBs can be found in the residential environment long after the use of these chemicals in domestic products and industrial processes has ceased. Dioxins have been assessed in Australia as being of very low concentrations. Despite concerns about residential dust as a source of human exposure to persistent chemicals, there has been limited testing of PCBs and dioxins in dust in Australia. As part of an assessment of maternal exposure to a variety of persistent toxic substances, we analysed 30 residential dust samples from a variety of geographical settings for their dioxin and PCB concentrations. PCBs were found in most samples, the median and range concentrations ( $\text{pg g}^{-1}$ ) of dominant congeners of PCB were as follows: PCB118 (315; <35.0–29 000), PCB105 (130; 14.0–16 000) and PCB156 (440; <5.00–2800). Dioxin concentrations were generally low with median concentrations for the total sum of dioxin-like polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) of  $3.75 \text{ pg g}^{-1}$  each. There was a very high percentage of non-detects. Concentrations of both PCBs and dioxins were low compared with most studies reporting residential dust concentrations internationally. Age of dwelling was the only factor observed to influence both PCB congener concentrations and dioxin isomers in multivariate regression analyses. No other housing or sociodemographic variables, including proximity to industry, were important predictors in multivariate linear regression models.

Received 11th July 2014  
Accepted 30th September 2014

DOI: 10.1039/c4em00383g

rsc.li/process-impacts

## Environmental impact

The residential environment is a significant source of environmental exposure for the developing child. The presence of persistent chemicals PCBs and dioxins in dust is suggested to represent a significant source of maternal and children's exposure in the home. This study found generally low concentrations of PCBs and dioxins in the homes of non-occupationally exposed pregnant women with those residing in older homes having higher concentrations. This study provides useful baseline information on residential dust concentrations of pregnant women and individual data from the home environment. Such data are vital to enable an understanding of the significance of sources for vulnerable groups in our society.

## Introduction

Polychlorinated biphenyls (PCBs) along with dibenzodioxins and dibenzofurans (dioxins) are persistent organic pollutants (POPs) that result largely from anthropogenic activities. There are 209 individual congeners of PCBs with approximately 130 of those congeners having widespread anthropogenic use prior to

1977 (ATSDR<sup>1</sup>). The term dioxin relates to polychlorinated dibenzodioxin (PCDD) compounds (chlorinated derivatives of *p*-dibenzodioxin) plus polychlorinated dibenzofurans (PCDF).<sup>23</sup>

PCBs were previously used in domestic products such as carpets and upholstery, paint, sealants, coolants, lubricants, pesticides and electronic equipment, which resulted in the extensive distribution of PCBs in the environment.<sup>1,2</sup> Other sources of these pollutants include the burning of waste in industrial incinerators and leakage from sealants and paint in older buildings.<sup>2,4</sup>

Although the manufacturing, processing and distribution of PCBs has been prohibited in almost all industrial countries since the 1980s, their entry into the environment still occurs, especially due to improper disposal practices or leaks in electrical equipment and hydraulic systems which may still be in use.<sup>2</sup> They are found in soils, dust, biota and hence in food due

<sup>a</sup>Centre for Ecosystem Management, Edith Cowan University, 270 Joondalup Drive, Joondalup, WA, Australia. E-mail: a.hinwood@ecu.edu.au; Tel: +61 8 6304 5372

<sup>b</sup>School of Population Health, The University of Western Australia, 35 Stirling Highway, Crawley, WA 6009, Australia

<sup>c</sup>National Measurement Institute Australia, 105 Delhi Rd, North Ryde, NSW 2113, Australia

<sup>d</sup>Department of Community Medicine, University of Tromsø, NO-9037 Tromsø, Norway

† Electronic supplementary information (ESI) available. See DOI: 10.1039/c4em00383g

to their persistence in environmental media.<sup>5</sup> In Australia, PCBs were never manufactured and importation was banned in 1975 (<http://www.npi.gov.au>).

PCBs are largely insoluble in water with the main exposure pathways in humans being the ingestion of food and inhalation of dust.<sup>2,6,7</sup> Based on the evidence in animal studies PCBs have been classified as probable human carcinogens (2A) with selected congeners considered to cause cancer (group 1). (<http://monographs.iarc.fr/ENG/Classification/>).

Dioxins are formed from activities such as industrial incineration and power generation, and also natural processes such as volcanic activity and forest fires which contribute to the presence of dioxins in the air, soil and sediment.<sup>5</sup> Pentachlorophenol (PCP) in textiles has also been identified as a source of PCDD/Fs.<sup>9</sup> Products using these textiles and other pesticides reported to contain PCDD/F could therefore contribute these compounds to dust in homes.<sup>3,9</sup>

The toxicity of a dioxin is determined by the number and configuration of chlorine (Cl) atoms in the compound, with 2,3,7,8-tetrachlorodibenzodioxin (TCDD) being the most toxic dioxin compound.<sup>8</sup> Whilst there are many PCB congeners, 12 of these are described as having dioxin-like characteristics and have either one (mono-*ortho*) or no (non-*ortho*) chlorine atoms at the *ortho* positions. Dioxins and the dioxin-like PCBs share the characteristic of binding to the aryl hydrocarbon receptor (AhR), an intracellular ligand-dependent transcription factor found in many tissues, although the toxicity of the dioxins and dioxin-like PCBs varies.<sup>8</sup> Any dioxin compound with a toxic response similar to TCDD is referred to as a dioxin-like compound and the toxicity of these compounds is expressed relative to TCDD by toxic equivalence factors.<sup>8</sup> A toxic equivalence factor (TEF) has been assigned to individual PCB and dioxin congeners based on results from *in vitro* and *in vivo* studies.<sup>8</sup>

As with PCBs, the main exposure pathways of humans to dioxins are through the ingestion of food containing these chemicals and inhalation of contaminated dust particles.<sup>2,10,11</sup> Other pathways include dermal contact, inhalation of re-entrained dust and accidental ingestion of soil and dust contaminated with PCBs and dioxins.<sup>12–14</sup> In the home, where most people spend over 80% of their time, residents may be exposed to PCB and dioxin concentrations that may increase the potential for adverse health effects which include adverse birth outcomes, neurodevelopmental effects and leukaemia.<sup>1,5,15</sup> Pregnant women and children may be more vulnerable to the effects of persistent organic pollutants and so are considered an at-risk group.<sup>5</sup>

This opportunistic study aimed to determine the concentrations of dioxins and dioxin-like PCBs in the residential homes of pregnant women. Demographic and housing characteristics were explored for their contribution to measured concentrations.

## Methods

This study was a cross-sectional assessment of residential dust concentrations of dioxins and PCBs. Dust samples were provided by participants of the Australian Maternal Exposure to

Toxic Substances (AMETS) study during the third trimester of their pregnancy.<sup>16</sup> Thirty samples were randomly selected from a possible 167 samples across the State of Western Australia.

Ethics approval was obtained from the Edith Cowan University Human Research Ethics Committee, WA Country Health Service, St John of God Health Care (Subiaco and Bunbury), Joondalup Health Campus and King Edward Memorial Hospital. All participants provided written informed consent.

### 2.1 Data collection

Each participant of the AMETS study completed a questionnaire which included information about their home, furniture and proximity to industry and roads (ESI Table S1†). Participants also collected a sample of dust from their vacuum cleaner.

### 2.2 Sample collection and preparation

The collection of dust is described in the study by Stasinska *et al.*,<sup>17</sup> and is summarised here. Participants were instructed to empty the contents of their vacuum cleaner into a provided plastic bag labelled with the participant code and date. Participants then posted the dust samples *via* Australia Post, directly to Edith Cowan University for storage and analysis.

The dust samples were received in individual plastic bags and each sample was mixed thoroughly within the plastic bag before being air dried in the laboratory for a minimum of 48 hours. The dried samples were individually shaken in a closed sifting pan to homogenise the <600  $\mu\text{m}$  fraction. Particles >600  $\mu\text{m}$  in size were removed and disposed of accordingly. To minimise cross-contamination between individual samples, the sieves were cleaned with a brush and fresh paper tissue and rinsed with acetone.

### 2.3 Analysis of PCDDs, PCDFs & PCBs

Analysis of dioxins and dioxin-like PCBs was undertaken at the National Measurement Institute, New South Wales. High resolution mass spectrometry was used to determine PCDDs, PCDFs & the dioxin-like PCBs concentrations ( $\text{pg g}^{-1}$ ) based on US EPA methods 8290, 1613B, 1668B & 3545 (USEPA<sup>18–21</sup>). The dust samples were dried with an inert drying agent, 20 g accurately weighed, homogenised and then spiked with 15 <sup>13</sup>C isotopically labelled PCDD/Fs and 12 <sup>13</sup>C isotopically labelled DL PCBs in order to act as surrogates. Toluene was used in the Dionex ASE 100 & ASE 300 auto extraction systems (150 °C, 1500 psi) to extract, concentrate and split all samples. The initial clean-up included acid back-extraction and gel permeation chromatography to remove sulphur contamination (as per the EPA method 3640A). Acidic/basic/neutral silica gel, alumina and activated carbon column clean-up was undertaken using FMS Power-Prep. Non-*ortho* substituted PCBs were separated from mono-*ortho* substituted PCBs and PCDD/Fs. Two PCDD/Fs and four isotopically labelled PCBs were added to each extract prior to analysis to determine surrogate recoveries.

The qualitative/quantitative analysis of PCDD/Fs, non-*ortho* substituted PCBs and mono-*ortho* substituted PCBs (congeners) was undertaken using a HP 6890 high-resolution gas chromatograph (coupled with a Finnigan Mat A200S auto sampler),

a Finnigan MAT 95XL high-resolution mass spectrometer (HRMS) (maintained at >10 000) and a computerised data system in accordance with US EPA methods 1613/1668. PCDD/Fs and PCB congener analyte identification occurred when two ions within the allowable abundance ratio were detected within the prescribed retention time window. The isotopically labelled surrogates were used for quantification of PCDD/Fs and PCBs. Single column analysis on HRMS allowed for co-elution of interfering compounds, so a second column was used for mono-*ortho* substituted PCBs and several dioxin/furans. The list of compounds analysed in dust in this study and the respective detection limits achieved for this study are shown in the ESI Table S2.†

## 2.4 Statistical analysis

PCB and dioxin concentrations were highly skewed and the data were log transformed prior to analysis. Statistical analysis was undertaken using SPSS version 21 (IBM). All samples recording less than detectable concentrations were assigned half the detection limit. Only those congeners with >70% detectable concentrations were investigated for factors or housing characteristics that may influence dust PCB and dioxin concentrations. Pearson correlation coefficients (continuous variables), Spearman rank correlation (ordinal variables) and Kruskal–Wallis tests (categorical variables) were used to assess the association of household and regional characteristics collected from questionnaire information on dust concentration data. Linear regression models were run on natural log transformed total concentrations of PCBs and dioxins. The initial set of predictors for inclusion in the regression modelling were those collected *via* questionnaire (ESI†) which included questions on factors identified in previous studies as being important for the presence of PCBs and dioxins. The variables considered for inclusion in the final models during formal model selection were those found to be significant in univariate analyses ( $p < 0.05$ ) and included age of home, region of WA and urban and rural categories for PCBs and age of home and building material of home for dioxins. The model was based on forward selection using a probability F cut off of  $p = 0.05$  for entry and  $p = 0.10$  for removal, with age of home being the only variable included in the final models.

## Results

The dust samples were collected from participants who lived in mainly brick and tile homes in urban areas away from industry (Table 1). Over 60 percent of homes were older than 10 years with twenty percent older than 50 years (Table 1). Many residents had undertaken renovation over the past few years (55%). All participants were non-smokers and none reported a family member or visitor smoking inside the home, however 46% of participants reported visitors smoking outside (Table 1).

Residential dust samples had total PCB concentrations ranging from less than the detection limit to 54 600 pg g<sup>-1</sup>. The congener with the highest concentration was PCB118, followed by PCB156 and PCB105 (Table 2).

Table 1 Housing characteristics ( $n = 30$  unless otherwise specified)

Characteristic	% of samples/ responses
Age of home (years) ( $n = 29$ )	
<2 years	6.9
2–10 years	24.1
10–50 years	48.3
>50 years	20.7
Type of home	
House	93.3
Duplex/townhouse/villa	3.3
Flat/unit/apartment	3.3
Urban dwelling (% of participants)	40.0
Rural and outback (% of participants)	60.0
Main building materials	
Brick/brick veneer/tile	70.0
Timber	13.3
Fibrocement/asbestos	16.7
Percentage of participants reporting living within 1 km of industry	30.8
Main heating source	
Electric	3.3
Gas	40.0
Wood	13.3
RC air conditioner	40.0
No heating	3.3
Reported renovations in past year (yes)	55.2
Reported visitor smoking outside	46.7

Table 2 Median, geometric mean and range for non-*ortho* and mono-*ortho* substituted PCB congeners (pg g<sup>-1</sup>)

Compound	Median (range)	% samples below DL	TEF (WHO 2005)
<b>Non-<i>ortho</i> substituted PCBs</b>			
TePCB77	39.5 (<1.5–3710)	3.3	0.0001
TePCB81	1.70 (<1.0–170)	33.3	0.0003
PePCB126	4.00 (<1.0–82.0)	26.7	0.1
HxPCB169	<1.0 (<1.0–9.0)	76.7	0.03
<b>Mono-<i>ortho</i> substituted PCBs</b>			
PePCB105	130 (14–16 000)*	0	0.0003
PePCB114	9.38 (0.5–1060)*	0	0.0003
PePCB118	315 (<35.0–29 000)	3.3	0.0003
PePCB123	6.55 (<1.0–550)	6.7	0.0003
HxPCB156	44.0 (<5.0–2790)	6.7	0.0003
HxPCB157	10.2 (<1.0–550)	10.0	0.0003
HxPCB167	16.5 (<1.00–720)	3.3	0.0003
HxPCB189	3.6 (<0.30–140)	6.7	0.0003
Total sum PCBs	572 (<DL – 54 600)		

The influence of household and regional characteristics on dust PCB concentrations was examined. Each individual PCB and total PCBs were used in the analysis of these relationships, with only age of home shown to be important, with increased age of home being associated with increased dust PCB concentrations (Fig. 1).

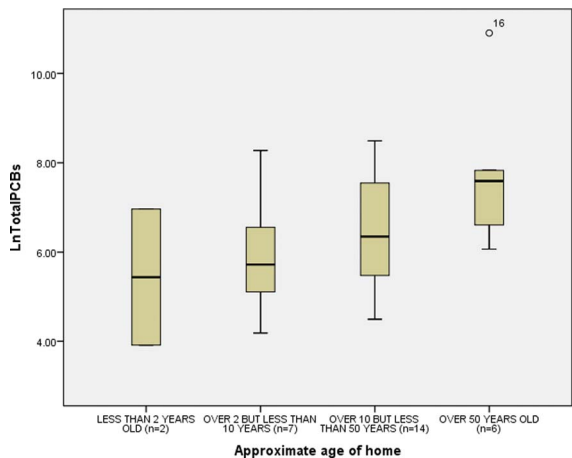


Fig. 1 Natural log of the sum of PCBs ( $\text{pg g}^{-1}$ ) by categories of age of home.

The regression analysis undertaken revealed that approximate age of home accounted for nearly 18% in the variation in total PCB concentrations (ESI Table S3†). No other factors were identified that significantly influenced either individual PCB congeners or total PCB concentrations.

The PCB congener profiles were examined and similar patterns were observed across all samples indicating widespread sources across Western Australia (Fig. 2). The PCB profile was dominated by mono-ortho PCB congeners, namely PCB-118 (42–59%) and PCB-105 (20–30%), followed by HxPCB-156 (3–13%). PCB-77 was the most dominant non-ortho PCB with some variation (2–17%) due to non-detection in some dust samples.

The concentrations of dioxins were low with a high percentage of non-detects for individual congeners (Tables 3 and 4). The PCDD congener profiles (Fig. 3) were similar for all dust samples across the urban and rural sites indicating similar contamination sources. The samples were dominated by octachlorodibenzo-*p*-dioxin (OCDD) (62–94%) followed by 1,2,3,4,6,7,8-HpCDD (5–29%) congeners. There were two slightly different samples; no. 15 (urban) and sample no. 24 (rural) where additional congeners were detected (0.1–24%), of

Table 3 Chlorinated dibenzo-*p*-dioxin concentrations ( $\text{pg g}^{-1}$ ) ( $n = 30$ )

Compound	Median (range)	% samples below DL
2,3,7,8-TCDD	<0.10 (<0.10–4.80)	70.0
1,2,3,7,8-PeCDD	<0.50 (<0.50–13.0)	56.7
1,2,3,4,7,8-HxCDD	<0.50 (<0.50–23.0)	73.3
1,2,3,6,7,8-HxCDD	1.75 (<1.0–37.0)	43.3
1,2,3,7,8,9-HxCDD	<1.50 (<1.50–21.0)	70.0
1,2,3,4,6,7,8-HpCDD	49.0 (<10.0–590)	16.6
OCDD	325 (<20.0–2960)	3.33
Total TCDD	1.50 (0.40–60.0)	0
Total PeCDD	3.80 (0.4–160)	0
Total HxCDD	20.0 (<1.5–450)	13.3
Total HpCDD	99.0 (2.5–1090)	3.3

Table 4 Chlorinated dibenzofuran concentrations ( $\text{pg g}^{-1}$ ) ( $n = 30$ )

Compound	Median (range)	% samples below DL
2,3,7,8-TCDF	<2.00 (<2.0–30.0)	73.3
1,2,3,7,8-PeCDF	<1.0 (<1.00–13.0)	73.3
2,3,4,7,8-PeCDF	<3.0 (<3.0–32.0)	73.3
1,2,3,4,7,8-HxCDF	<3.0 (<3.0–64.0)	80.0
1,2,3,6,7,8-HxCDF	<4.0 (<4.0–57.0)	83.3
1,2,3,7,8,9-HxCDF	<0.3 (<0.3–4.40)	1.00
2,3,4,6,7,8-HxCDF	<3.0 (<3.0–74.0)	13.3
1,2,3,4,6,7,8-HpCDF	<20.0 (<20.0–340)	60
1,2,3,4,7,8,9-HpCDF	<4.0 (<4.00–46.0)	86.7
OCDF	18.5 (<5.0–290)	26.7
Total TCDF isomers	5.85 (<2.0–660)	10.0
Total Pe CDF	3.80 (0.45–390)	30.0
Total Hx CDF	5.70 (<2.0–640)	26.7
Total Hp CDF	9.45 (0.5–530)	6.7

1,2,3,6,7,8-HxCDD, 1,2,3,7,8,9-HxCDD, 1,2,3,4,7,8-HxCDD and 1,2,3,7,8-PeCDD suggesting that they may each have a local source compared with other samples (Fig. 3).

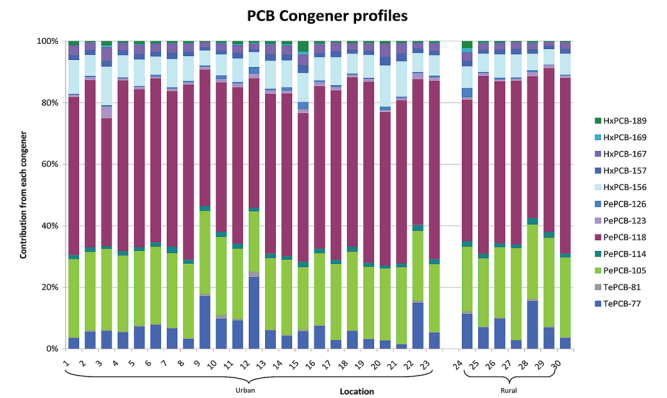


Fig. 2 Contribution of PCB congeners to total mass of dioxin-like-PCBs (<DL samples recorded as DL).

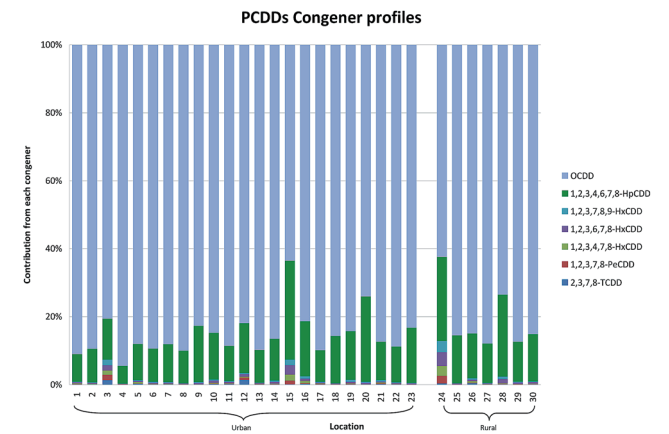


Fig. 3 Contribution of PCDD congeners to total mass of PCDDs (<DL samples recorded as DL).



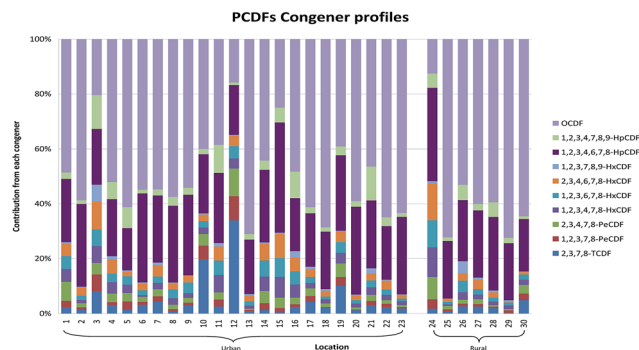


Fig. 4 Contribution of PCDF congeners to total mass of PCDFs (<DL samples recorded as DL).

There was a greater variation in PCDF congener profiles (Fig. 4), which may indicate several local sources such as combustion or industrial emissions, although the distance to road or industry was not observed to be a contributor to the dust concentrations.

The PCDF congener profile was dominated by OCDF (13–72%) > 1,2,3,4,6,7,8-HpCDF (15–40%) > 2,3,4,6,7,8-HxCDF (2–13%). Sample no. 24 (rural) once again differs from the rest of the dust samples by having the lowest proportion of the OCDF congener (13%).

Associations with household and regional characteristics were examined and it was found that age of home was important for total TCDF, HxCDF and HpCDF (ESI Table S3†).

Concentrations of PCDD and PCDF tended to be higher in the dust samples collected from the homes of participants who reported residing in proximity to industry, but this relationship was not statistically significant (ESI Table 3†).

## Discussion

The results confirm the presence of PCBs in residential homes, and hence the potential for exposure, long after the use of these chemicals in products have ceased and the increased concentrations in older homes supports the work of other researchers.<sup>22</sup> Concentrations in this study (collected between 2009 and 2011) were low compared with concentrations of PCBs in dust samples from homes collected during similar time periods (2006 and 2008) in Canada, New Zealand, the UK and the USA.<sup>6,22</sup> The concentrations of both PCBs and dioxins were however similar to those reported by Tue *et al.*,<sup>14</sup> who also used participant collected dust in New York State, USA.

The dominant PCB in this study was PCB118, as has been found in other studies.<sup>24</sup> Whitehead *et al.*,<sup>23</sup> sampled carpet dust and reported PCB118 concentrations that were nearly 10 times greater than the concentrations in this study. PCB105 concentrations were also higher in the Whitehead *et al.*, study.<sup>23</sup> Knobloch *et al.*,<sup>22</sup> also reported higher concentrations in US homes and an increased concentration in older homes, predominantly built between 1959 and 1970, following which concentrations decreased. Concentrations of PCBs in this study were also lower

than those reported in classrooms and outdoor environments.<sup>3,10</sup>

The dioxin concentrations for the different congeners were also low with most congeners below the limit of detection. Concentrations were low compared with most studies reviewed<sup>6,14,25</sup> and also lower than those of the study of O'Connor and Sabarsula<sup>26</sup> who analysed dust from houses in the US, at what were considered to be background concentrations.

PCDD/F congener profiles of soil samples around Australia are dominated by OCDD, which contributes 60–90% of total PCDD/F congener profiles, with HpCDD being the next most dominant congener.<sup>27</sup> A similar profile was identified in household dust in this study. The interpretation of the congener profiles in this study has shown that for most homes tested, the PCDD/Fs and dioxin like-PCBs contamination may be from similar, diffuse sources, but identified individual samples with different profiles, indicating local sources. The results are consistent with the findings of other researchers where the most abundant isomer OCDD has been reported due to its presence in soils and from atmospheric deposition.<sup>28</sup> The finding of increased dioxin concentrations with age of home was interesting given that these substances have traditionally been reported to be associated with emissions from combustion sources. However PCDDs have also been identified as being associated with pesticides which may have degraded over time to produce dioxins which have resulted in increasing concentrations in residential dust over time.<sup>3</sup>

In a ranking of indoor semi-volatile compounds in dust in homes based on the literature reported toxicity, Bonvallot *et al.*,<sup>7</sup> considered PCBs found in household dust as a risk factor for infants and children, although the concentrations used in the ranking were those by Harrad *et al.*<sup>6</sup> which were substantially higher than those reported here.

This study was limited by the small sample size and cross-sectional design as well as the lack of repeated measurements. The use of a 600 µm sieve fraction during sample preparation may have underestimated concentrations as smaller size fractions are associated with higher concentrations.<sup>29</sup> Compared with other studies, the size fraction of dust analysed in this study was larger (600 µm) compared with a variety of mesh sizes down to 150 µm used in other studies.<sup>6,14,15,23</sup>

## Concluding remarks

PCBs and dioxins were found in household dust in homes of non-occupationally exposed pregnant women. The concentrations were low and may have been underestimated due to the larger sieve size used. To enable more definitive comparisons with other studies and to aid in the application of this data to an assessment of risk, the use of standard collection methods, dust size fraction and preparation and analysis should be undertaken.

The concentrations of PCBs and dioxins found in household dust were low in this study compared with concentrations reported in the international literature.

## Acknowledgements

This study was funded by an ARC linkage grant in partnership with the Arctic Monitoring and Assessment Program (AMAP). The ARC was not involved in the design, conduct or reporting on this study. The research team would like to thank all participants who provided samples.

## References

- Agency for Toxic Substances and Disease Registry (ATSDR), Atlanta, GA, USA, 2000, <http://www.atsdr.cdc.gov/toxprofiles/tp17.pdf>.
- K. Srogi, *Environ. Chem. Lett.*, 2008, **6**, 1–28.
- A. Franzblau, L. Zwica, K. Knutson, Q. Chen, S. Y. Lee, B. Hong, P. Adriaems, A. Demond, D. Garabrant, B. Gillespie, J. Lepkowski, W. Luksemburg, M. Maier and T. Towey, *J. Occup. Environ. Hyg.*, 2009, **6**, 188–199.
- C. Tlustos, M. Sheridan, D. O'Sullivan, W. Anderson and A. Flynn, *Food Addit. Contam.*, 2012, **29**(1), 128–138.
- D. O. Carpenter, *Rev. Environ. Health*, 2011, **26**(1), 61–69.
- S. Harrad, C. Ibarra, M. Robson, L. Melymuk, X. Zhang, M. Diamon and J. Douwes, *Chemosphere*, 2009, **76**(2), 232–238.
- N. Bonvallot, C. Mandin, F. Mercier, B. Le Bot and P. Glorennec, *Indoor Air*, 2010, **20**, 458–472.
- M. Van den Berg, L. S. Birnbaum, M. Denison, M. De Vito, W. Farland, M. Feeley, H. Fiedler, H. Halansson, A. Hanberg, L. Haws, M. Rose, S. Safe, D. Schrenk, C. Tihyama, A. Tritscher, J. Tuomisto, M. Tysklind, N. Walers and R. E. Peterson, *Toxicol. Sci.*, 2006, **93**(2), 223–241.
- M. Horstmann and M. S. McLachlan, *Organohalogen Compd.*, 1994, **20**, 251–254.
- S. Harrad, E. Goosey, J. K. Desborough and A. Covaci, *Environ. Sci. Technol.*, 2010, **44**, 4198–4202.
- L. Roosens, M. A. Abdallah, S. Harrad, H. Neels and A. Covaci, *Environ. Sci. Technol.*, 2010, **44**(8), 2870–2875.
- G. Suzuki, H. Takigami, K. Nose, S. Takahashi, M. Asari and S.-I. Sakai, *Environ. Sci. Technol.*, 2007, **41**, 1487–1493.
- R. F. Herreck, M. D. McClean, J. D. Meeker, L. K. Baxter and G. A. Weymouth, *Environ. Health Perspect.*, 2004, **117**, 1051–1053.
- T. M. Tue, G. Suzuki, S. Takahashi, K. Kannan, H. Takigami and S. Tanabe, *Environ. Pollut.*, 2013, **181**, 75–80.
- M. H. Ward, T. S. Colt, C. Metayer, R. B. Gunier, J. Lubin, V. Crouse, M. G. Nishioka, P. Reynolds and P. A. Buffer, *Environ. Health Perspect.*, 2009, **117**(6), 1007–1013.
- A. L. Hinwood, A. C. Callan, M. Ramalingam, M. Boyce, J. Heyworth, P. McCafferty and J. O. Odland, *Environ. Res.*, 2013, **126**, 118–124.
- A. Stasinska, A. Reid, A. Hinwood, G. Stevenson, A. Callan, J. O. Odland and J. Heyworth, *Chemosphere*, 2013, **91**, 187–193.
- USEPA, 1997 US EPA Method 1613, [http://water.epa.gov/scitech/methods/cwa/organics/dioxins/upload/2007\\_07\\_10\\_methods\\_method\\_dioxins\\_1613.pdf](http://water.epa.gov/scitech/methods/cwa/organics/dioxins/upload/2007_07_10_methods_method_dioxins_1613.pdf), accessed July 2014.
- USEPA 2007a. US EPA Method 8290A <http://www.epa.gov/osw/hazard/testmethods/sw846/pdfs/8290a.pdf>, accessed July 2014.
- USEPA, 2007b US EPA Method 3545A <http://www.epa.gov/epawaste/hazard/testmethods/sw846/pdfs/3545a.pdf>, accessed July 2014.
- USEPA, 2008 US EPA Method 1668B [http://water.epa.gov/scitech/methods/cwa/bioindicators/upload/2009\\_01\\_07\\_methods\\_method\\_1668.pdf](http://water.epa.gov/scitech/methods/cwa/bioindicators/upload/2009_01_07_methods_method_1668.pdf), accessed July 2014.
- L. Knobeloch, M. Turyk, P. Imm and H. Anderson, *Chemosphere*, 2012, **86**(7), 735–740.
- T. P. Whitehead, J. R. Nuckols, M. H. Ward and S. M. Rappaport, *Emerging themes in Epidemiology*, 2012, vol. 9, p. 2, <http://www.etc-online.com/content/9/1/12>.
- M. Kohler, M. Zennegg and R. Waeber, *Environ. Sci. Technol.*, 2002, **36**, 4735–4740.
- Y. Kang, K.-C. Cheung, Z.-W. Cai and M. H. Wong, *Ecotoxicol. Environ. Saf.*, 2011, **74**, 947–952.
- R. O'Connor and J. Sabrsula, *Environ. Forensics*, 2005, **6**, 283–287.
- J. Müller, K. Mülle, M. Goudkamp, M. Mortimer, and D. Haynes, Department of Environment and Heritage. Technical report no. 5, 2004 pp. 1–42.
- N. M. Tue, G. Suzuki, S. Takahashi, T. Isobe, P. T. K. Trang, P. H. Viet, S. Tanabe, *Environ. Sci. Technol.*, 2010, **44**, 9195–9200.
- W. Wang, M. J. Huang, J. S. Zheng, K. C. Cheung and M. H. Wong, *Sci. Total Environ.*, 2013, **463–464**, 1201–1209.