A Screening Level Risk Assessment of Polybrominated Diphenyl Ethers In Young Children Up To 4 Years of Age in New Zealand

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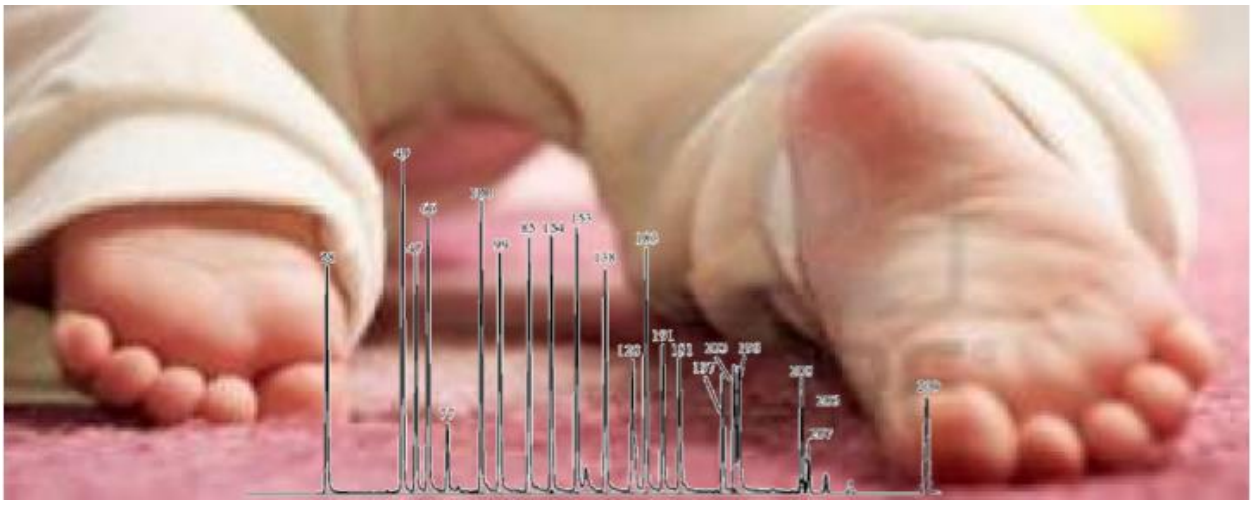

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A SCREENING LEVEL RISK ASSESSMENT OF POLYBROMINATED DIPHENYL ETHERS IN YOUNG CHILDREN UP TO 4 YEARS OF AGE IN NEW ZEALAND

Prepared as part of a Ministry of Health
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EXECUTIVE SUMMARY

We present a hazard index approach to characterise the risks associated with New Zealand infants and toddler exposures to polybrominated diphenyl ethers (PBDEs), focusing on house dust, car dust, and diet (including breast milk) as the main exposure routes. PBDE hazards were characterized using the current US Environmental Protection Agency's Reference Doses, and the aggregate risk estimated using a grouping of PBDE congeners into three hazard categories: 1) tri-pentaBDE, 2) hexa-nonaBDE, and 3) DecaBDE. Using New Zealand specific breast milk PBDE concentrations, dietary PBDE intakes from the UK Food Standards Agency, New Zealand specific house dust PBDE concentrations, and car dust PBDE estimates from international data sources, exposure scenarios were constructed for infants 0-6 months, toddlers 6-12 months, and children 1-4 years old.

Results show that, for all three scenarios, under typical daily dust intake estimates, the risk of an aggregate exposure from these routes is below the threshold of concern, with a hazard index (HI) of less than 1.0 (Figure ES-1). The estimated HI for typical exposures were: 0.2, 0.1, and 0.1 for the 0-6 month, 6-12 month, and 1-4 year old scenarios, respectively. Under typical daily dust ingestion scenarios, exposures to maximum PBDE concentrations were still estimated to fall below an HI of 1.0, with values of 0.9, 0.5, and 0.2 for the three age groups. However, in the extreme case of high daily dust ingestion (200 mg/day), combined with the highest reported PBDE concentrations, overall HIs were 1.5, 0.8, and 0.4 for the 0-6 month, 6-12 month, and 1-4 year age groups, respectively. PBDE intakes from breast milk were the driving factor for the estimate of aggregate PBDE risk for infants, while house and car dust exposures constituted the most prominent contributors for 1-4 year olds.

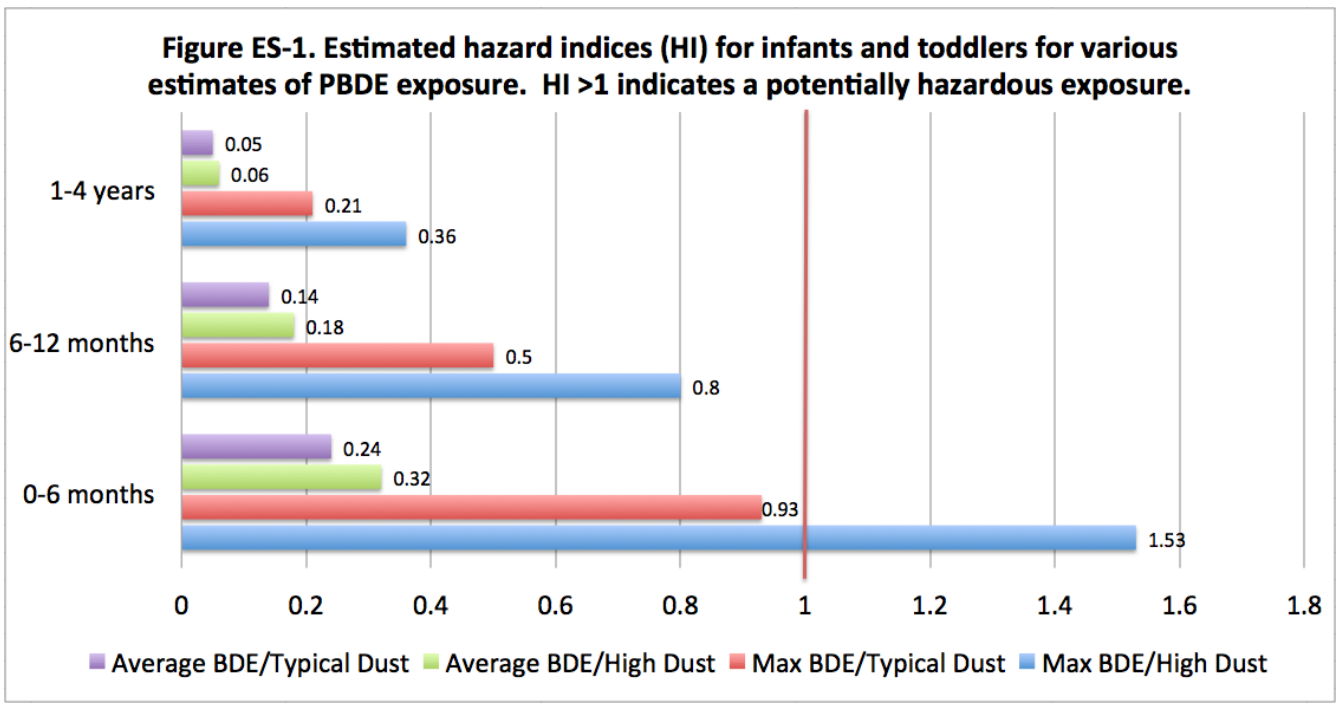

Figure ES-1. Estimated hazard indices (HI) for infants and toddlers for various estimates of PBDE exposure. HI >1 indicates a potentially hazardous exposure.

A hazard index of less than 1.0 is an indication that the exposure is unlikely to be of toxicological significance. It is important to note that these estimates did not include minor contributing routes including inhalation and dermal absorption. The direct mouthing contact of plastic toys was also not quantitatively factored into this assessment. In addition, biomonitoring studies internationally appear to consistently indicate the presence of a higher daily dose of PBDEs than risk assessment approaches predict. Finally, as scientists learn more about new classes of chemicals, the hazard levels set by regulatory agencies tend to decrease over time, sometimes quite dramatically. Thus, while the current assessment finds risks to be generally below levels of concern, the additional qualitative considerations mentioned above leave some questions open about exposures and risks to infants exposed to the higher end of the characterized exposures.

1. INTRODUCTION

Polybrominated diphenyl ethers (PBDEs) are chemicals added to commercial items that contain foams, fabrics and plastics to reduce the likelihood of fires. PBDEs have emerged, over the past fifteen years, as persistent environmental contaminants of health concern, now commonly reported in biomonitoring studies worldwide. These compounds exist globally in the environment due to the production of three commercial products: Pentabromodiphenyl ether (PentaBDE), Octabromodiphenyl ether (OctaBDE), and Decabromodiphenyl ether (DecaBDE). The pentaBDE and OctaBDE products have become either banned or restricted in use, leaving only the DecaBDE formulation being actively produced in some areas. The three commercial products are mixtures of brominated diphenyl ether (PBDE) congeners that are commonly found in biological and environmental matrices. Of the 209 theoretical congeners possible, the congeners most prominently encountered in environmental or human sampling include: BDE-28, BDE-47, BDE-99, BDE-100, BDE-138, BDE-153, BDE-154, BDE-183, and BDE-209. Additional congeners constitute largely trace levels in blood and environmental samples but are sometimes included in biomonitoring studies for completeness and because it has been shown that higher brominated PBDEs can degrade into lower congeners (Shih and Wang 2009).

Congeners BDE-47, 99, 100, 153, 154 are tetra-, penta-, and hexabromodiphenyl ether components of the “penta” commercial mixture, which has primarily been used as an additive in polyurethane foams in carpet padding and furniture. In 2004, this mixture was banned in Europe and voluntarily removed from use in the United States because of persistence in the environment and concerns about safety (Harrad and Porter 2007; Daniels et al 2010). Manufacturers in the United States and Europe have voluntarily ceased, or agreed to cease, the production and use of commercial grade PBDEs (USEPA 2010).

Congener BDE-209 is the main component of the “deca” commercial mixture, which has been used in hard plastic housings for electronic equipment, as well in upholstery fabric, plastic furniture and plastic toys. Deca was increasingly used after the ban on products that contain lower molecular weight congeners, but was banned in Europe in 2008 and is being phased out of use in the U.S. (Harrad and Porter 2007; Fowles and Morgott 2013). BDE-209 is not considered a persistent organic pollutant under the Stockholm convention due to its short biological half-life (Thuresson et al 2006; NICNAS 2007). However, its environmental persistence is considerably longer, and BDE-209 is believed to break down into lower congeners that have longer biological half-lives (Shih and Wang 2009).

Due to their environmental persistence and ability to bioaccumulate, their legacy and continued production, and their presence in commercial products, people continue to experience daily exposure to PBDEs worldwide (Harrad and Porter 2007; Law et al 2014; Sjodin et al 2008; Wong et al 2013). However, the pattern and magnitude of PBDE exposures vary from country to country, as illustrated by the results of the various international biomonitoring and environmental sampling data available. Harrad and Porter (2007) first reported on blood serum PBDE levels in New Zealanders, finding the levels to be consistent with reports from the UK and Europe.

The primary health concern with PBDEs, is that the youngest age groups have been repeatedly documented to carry the highest PBDE body burdens worldwide and to have the highest estimated exposures in the population (Coakley et al 2013; Fischer et al 2006; Gomara et al 2006; Roosens et al 2010). Exposure assessments have determined that these high early exposures are due largely to the combination of breast milk and house dust exposures to infants and toddlers (Hays et al 2006; Sjödin et al 2008; Johnson-Restrepo et al 2009; Roosens et al 2010; Lunder et al 2010; Johnson et al 2010; Trudel et al 2011). Adding to this concern is that the most critical toxicological effects of these compounds are also found in animal toxicology or human epidemiology studies that examine exposures to the very young, most notably when exposures occur *in utero* or early post-natally (Blanco et al 2013; Branchi et al 2002; Johansson et al 2008; Rice et al 2009; Suvorov et al 2009; Viberg et al 2003a, Viberg et al 2003b, Viberg et al 2004; Viberg et al 2005; Zhang et al 2013). Combined, these two characteristics lead to concerns about the risk to the young, susceptible, developing human nervous and endocrine systems from nearly ubiquitous passive exposures (Lorber 2008; Fowles and Morgott 2013).

House dust and car dust have emerged as highly significant sources for PBDE mobility into humans (Coakley et al 2013; Harrad et al 2008; Lorber 2008; Mannetje et al 2013; Stapleton et al 2005; Toms et al 2009a). International house dust PBDE concentrations from these studies are illustrated in Figure 1.

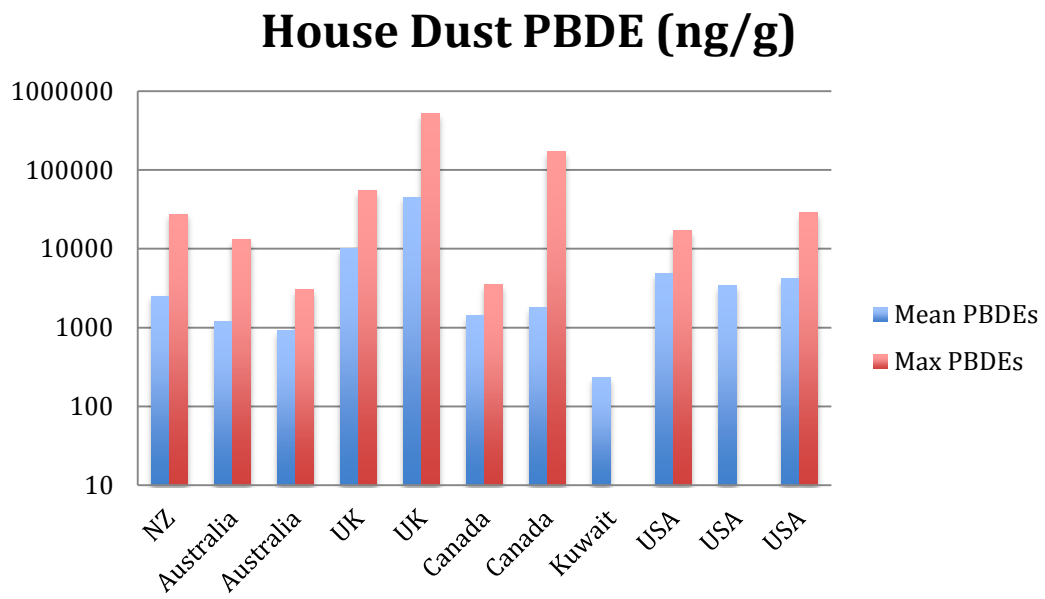

Figure 1 House dust total PBDE concentrations reported in NZ, Australia, Canada, Kuwait, USA, and UK (mean values)

House dust is now known to be an important source of PBDE exposure, and has been estimated to be a highly significant contributor to daily intakes of PBDEs to infants and toddlers, particularly in reference to the higher brominated PBDE congeners (Sjodin et al 2008; Toms et al 2009a). Chief among the higher brominated PBDE congeners, BDE-209 specifically is found in very high

concentrations in house dust in New Zealand, Australia, Kuwait, the UK, and Europe. The relative contributions of BDE-209 vs. BDE-99 to total PBDE qualitatively separate North American exposures from most other countries that have been studied (Coakley et al 2013; Gevao et al 2006; Sjodin et al 2008; Trudel et al 2011).

This fact creates difficulties in interpreting many studies that have left out BDE-209 from the summed PBDE exposure assessment. For example, Toms and associates reported on biomonitoring trends for PBDE in Australia, using 4 PBDE congeners (47, 99, 100, and 153), and found that exposures did not change markedly between 2002/03 and 2008/09. Yet, BDE-209, the most prominent PBDE congener in house and car dust, was not included in their analysis (Toms et al 2012). Similarly, Hwang and colleagues reported on high levels of several PBDE congeners in US house dust, but did not include BDE-209 (Hwang et al 2008). The difficulty posed by leaving the critical BDE-209 congener out of exposure assessments was described by Lorber in a comprehensive exposure assessment paper of PBDEs (Lorber 2008).

A recent investigation by the New Zealand Ministry for the Environment (MfE) into the prevalence of PBDE flame retardants in New Zealand evaluated products used by children in the 0-5 year age group and found PBDE presence in most footwear and toys with levels not exceeding 0.04%, or 400 ppm (measured as total bromine) (MfE 2010). The MfE found, using an x-ray fluorescence detector, that the percentage PBDE in automotive interiors was 0.069%, or 690 ppm (MfE 2010).

A previous risk assessment examined the estimated exposure and risk of PBDEs to infants and toddlers in New Zealand from dust formed from the foam materials used in child car seats (Fowles and Morgott 2013). In this assessment, using conservative assumptions, a highly exposed New Zealand child's exposure to PBDEs emanating from car seats, or in the car environment, was estimated to be less than the US Environmental Protection Agency's (US EPA) Reference Doses for chronic long-term exposures to PBDEs. However, the exposure was significant, considering that the contribution to the overall daily PBDE intake from other sources was not included in the calculations.

In a New Zealand study by Coakley and colleagues, PBDE intakes from house dust and breast milk were calculated and total PBDE intakes summed (Coakley et al 2013). Their assessment noted higher intakes of BDE-47 in the 0-3 month old infants, and higher intakes of BDE-209 in older age groups.

The aim of the present assessment was to qualitatively summarise key considerations in PBDE risk assessments, and to provide quantitative risk estimates of likely and worst case exposures to infants and toddlers up to 4 years of age, for the two most prominent known PBDE exposure routes: dust (car and house) and diet (breast milk and other foods). Our approach differs from previous assessment in that it incorporates the assumption of dose addition and use of a cumulative hazard index across three PBDE congener groupings.

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2. METHODS

A literature search was conducted to identify the scientific and medical research papers published on intakes of PBDEs around the world. PubMed was used as the primary search engine. ToxNet, Google and Google Scholar were used as supplements to identify papers and reports from sources outside the traditional scientific literature, including dietary and exposure reports from the governments of Australia and New Zealand, and abstracts from scientific conferences. Search terms used included combinations of "polybrominated diphenyl ether", "PBDE", "exposure," "dust," "diet," "oral," and "New Zealand". References of identified papers were reviewed, and relevant papers from the references were also obtained. A total of 210 research papers, reports, and abstracts from scientific conferences were identified in this manner as potentially useful for this report, and these were reviewed in detail. The Mekentosj Papers software program was used for reference management.

2.1 Hazard Index Approach and Hazard Assessment

Because different reference dose or hazard values exist for the tetra, penta, hexa, and decaBDE congeners, and because not all PBDE congeners have published hazard values specifically associated with them, these PBDE congeners were grouped, based on structure. The groups' hazard potencies were estimated using conservative assumptions for the tri- through penta- congeners, and for the hexa- through nona- congeners. BDE-209 was calculated separately, and has a significantly different hazard value (Table 1). A combined hazard index was calculated assuming dose-addition, through the assumption of a currently unknown common adverse outcome pathway for all PBDE congeners, differing only in potency. This approach allows for consideration of individual PBDE congener potency while assessing the potential cumulative impact of simultaneous exposure to the mixture of congeners.

The equation describing how to utilise congener-specific data for risk assessment of the mixture of congeners by combining hazard values (RfD) and specific congener exposure estimates (CBDE) is shown below:

$$HI = Sum [CBDE_1/RfD_1 + CBDE_2/RfD_2 + \ldots. CBDE_n/RfD_n/]$$

where a Hazard Index (HI) ratio greater than 1.0 indicates exposures that collectively exceed a risk level of concern as defined by the Reference Doses used.

The toxicological hazards of PBDEs, for the purposes of risk assessment, were previously reviewed (Fowles and Morgott 2013). An updated review of the toxicology and epidemiology literature on PBDEs confirmed that the critical toxicological adverse outcome for PBDEs in the earlier assessment was altered neurological development and behaviour, as measured in studies on rodents by numerous laboratories and supported by epidemiological studies (Herbstman et al 2010; Viberg et al 2003a; Viberg et al 2003b; Viberg et al 2004).

The US EPA Reference Doses for PBDEs 47, 99, 153, and 209 were thus considered to represent the most appropriate hazard values for the risk assessment

(IRIS 2008a; IRIS 2008b; IRIS 2008c; IRIS 2008d). The expected NTP chronic carcinogenicity study on PBDE99 was not yet released at the time of this report. Preliminary data from the NTP study website indicated that genotoxicity studies were negative (http://ntp.niehs.nih.gov/?objectid=BDC849E4-123F-7908-7BBBD74E7E1CC1E0 - site accessed 08 May, 2014).

Based on the updated hazard review, the hazard values used for the screening level risk assessment are based on US Environmental Protection Agency Reference Dose (RfD) values, as shown in Table 1.

Table 1 Hazard values used for screening level risk assessment.

PBDE Congener Names	Congener Range	Reference Value for Screening RA	Source for Reference Value
lower PBDEs (tri, tetra, penta)	BDE 16-127	0.1 µg/kg/d	IRIS 2008a,b
mid-range PBDEs (hexa, hepta, octa and nonaBDE)	BDE 128-208	0.2 µg/kg/d	IRIS 2008c
decaBDE	BDE 209	7 µg/kg/d	IRIS 2008d

Since the 2013 Fowles and Morgott report on PBDE exposures in cars, a correlative study from Adgent and colleagues on 304 mothers with breastfeeding infants found imprecise associations with PBDE (BDE-28, BDE-47, BDE-99, BDE-100, and BDE-153) exposures on increased anxiety, increased withdrawal and improved cognition using breast milk collected at 3 months of age and neurological development measured at 36 months of age. In this study, only breast milk as a source of PBDE exposure was used and house dust PBDE exposure was not considered (Adgent et al 2014). BDE-209 was not included in their analysis.

A review of environmental data up to 2012 found that, despite the phase out or ban of some PBDEs regionally, and the decreasing trend for pentaBDE, BDE-209 levels are still increasing globally, potentially serving as a reservoir for lower brominated congeners in future years (Law et al 2014).

In a mechanistic toxicology study, Pereira et al (2014) found that BDE-154 interfered with rat mitochondrial function through ATP depletion *in vitro*.

The toxicity of particulate-bound PBDEs on the pulmonary tract was investigated in rats (Kim et al 2014). In this study, increased pulmonary inflammation and neutrophil recruitment into the lung was observed in rats exposed to PBDEs bound to particulates.

These most recent studies are of interest, but are consistent with the selection of the USEPA RfD values based on postnatal neurological development.

2.2 Exposure Assessment

A semi-quantitative multipathway exposure model for PBDEs was constructed which considered the following major sources of exposure to infants and toddlers (Figure 2):

* Diet (including breast milk)
* House Dust
* Car Dust

Exposure scenarios were constructed to provide estimates of daily typical (mean) and high (qualitatively defined) intake of grouped PBDEs. Wherever possible, studies which measured a "full suite" of PBDE congeners were used, ideally including at least PBDEs: 28, 47, 99, 100, 138, 153, 154, 183, and 209.

The current risk assessment aims to estimate PBDE exposures from the two routes that are considered to be the most significant, diet and dust, to add to and complete a more general exposure and risk assessment of PBDEs to New Zealand infants and toddlers, up to 4 years of age. Exposures to inhaled airborne PBDEs, dermal absorption from skin contact with hard plastics, or PBDEs in drinking water were considered qualitatively to represent relatively minor contributors to overall PBDE exposure pathways.

PBDE exposures pathways and scenarios described in this report are shown in Figure 2 below.

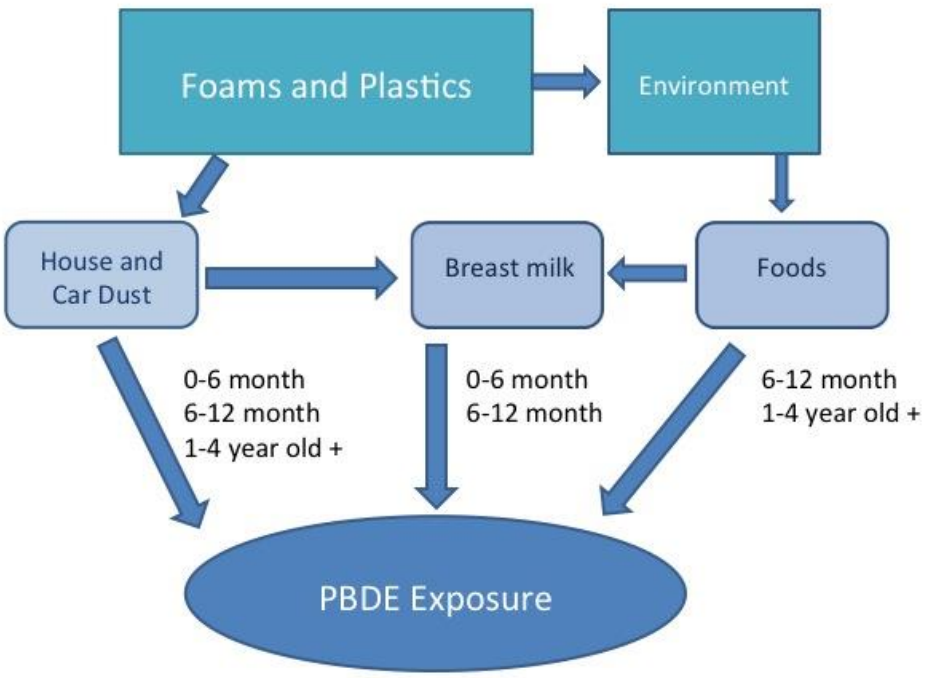

Figure 2 PBDE exposure pathways, with an emphasis on children up to age 4.

2.3 Exposure Parameters

The exposure parameters used in this assessment are largely generic and taken from various published sources, as described in Table 2. The amount of time spent in a car seat was determined by Fowles and Morgott to be 65 minutes/day (mean value), and this was extended by a factor of 2 in the case of 0-6 month old infants, based on the published report by Callahan and Sisler (1997) which found that 94% of infants less than 5 months of age spend 30 minutes or more restrained in a removable car seat carrier potentially constructed with foam containing PBDEs.

Table 2 Key parameters used in PBDE exposure scenarios.

Key parameter	Value	Reference
Scenario 1 (0 – 6 months old)		
Body weight	6.5 kg	USEPA exposure factors handbook 2011, Table ES-1
Breast Milk consumption	770 ml/day	Coakley et al 2013, 3-6 months
Breast Milk lipid content	3.7 %	Harden et al 2005 (average value)
Dust intake (all routes)	0.06 g/day	USEPA exposure factors handbook 2011, Table ES-1
Dust absorption rate	0.82	Fowles and Morgott, 2013
Fraction of day in car + car seat	0.08	Fowles and Morgott, 2013
Fraction of day in home	0.92	1-fraction in car
Scenario 2 (6-12 months old)		
Body weight	9.2 kg	USEPA exposure factors handbook 2011, Table ES-1
Breast Milk consumption	385 ml/day	Coakley et al 2013, 3-6 months
Breast Milk lipid content	3.7 %	Harden et al 2005 (average value)
Dust intake (all routes - typical)	0.06 g/day	USEPA exposure factors handbook 2011, Table ES-1
Dust intake (all routes - high)	0.2 g/day	Harrad et al 2008
Dust absorption rate	0.82	Fowles and Morgott, 2013
Food consumption (all relevant)	NA	Half of reported intake ng/kg/day from UK FSA
Fraction of day in car + car seat	0.04	Fowles and Morgott, 2013
Fraction of day in home	0.96	1-fraction in car
Scenario 3 (1 – 4 years old)		
Body weight	13.8 kg	USEPA exposure factors handbook 2011, Table ES-1
Dust intake (all routes - typical)	0.1 g/day	USEPA exposure factors handbook 2011, Table ES-1
Dust intake (all routes high)	0.2 g/day	Harrad et al 2008

Key parameter	Value	Reference
Dust absorption rate	0.82	Fowles and Morgott, 2013
Food consumption (all relevant)	NA	Reported intake ng/kg/day from UK FSA
Fraction of day in car + car seat	0.04	Fowles and Morgott, 2013
Fraction of day in home	0.96	1-fraction in car

2.4 Dietary intakes

A total of 36 published papers were identified describing intakes of PBDEs from diet. Of these, 14 included data relevant to children and toddlers (who along with infants represent the most vulnerable populations). These papers are reviewed in detail in Table 3. These studies are discussed in more detail in the discussion section of this report, along with descriptions of variations in dietary intakes by geographic region. None of these studies were conducted in New Zealand.

Although dietary intake studies specific to New Zealand were not found, several researchers have described the diets of toddlers in New Zealand (Theodore et al 2006; Harrad and Porter 2007; Rush et al 2008; Bristow 2010; Liu, 2010). The New Zealand Total Diet Study (NZTDS) contains a simulated typical diet for toddlers (Vannoort et al 2009). For this risk analysis, for children over one year of age, the diets were considered to follow a Western model diet, because the foods eaten by toddlers in New Zealand are similar to those eaten by toddlers from other countries following Western-type diets (Szymlek-Gay et al 2010).

In some places, seafood consumption is a major contributor to PBDE exposure (Bocio et al 2004; Meng et al 2007; Guo et al 2010; Lee et al 2013; Mannetje et al 2013; Coakley et al 2013). However, it is not clear if this is the case among young children in New Zealand. In Australia, PBDE congeners were measured in foods analysed as part of the 22nd Australian Total Diet Study (FSANZ, 2007). Table 5 shows the results of this analysis. Foods with high levels of PBDEs in Australia include eggs, cream, and meats, particularly fatty meats such as pork chops and bacon (FSANZ, 2007). PBDE levels in fish fillets were not particularly high compared to other foods, at 173 pg/g (0.173 ng/g). However, Shanmuganathan et al (2011) collected seafood samples from fish markets in Adelaide, Australia, and found PBDE concentrations ranging from 1.01 to 45 ng/g fresh weight. The highest levels were observed in silver fish from Vietnam and prawns from Thailand, and were lower in local Australian fish. For the purposes of this risk analysis, seafood consumption was considered to be just one among many potential dietary sources of PBDE exposure.

2.5 Breast milk exposures

Dietary sources of PBDE exposure include breast milk for infants, and, for the purposes of the current report, breastfeeding as an exposure route was applied to children up to 1 year of age. A total of 10 published papers were identified describing intakes of PBDEs from breast milk. These papers are reviewed in detail in Table 4 and discussed in more detail in the discussion section of this report. The study by

Mannetje et al (2013) measured breast milk PBDE concentrations in New Zealand, but did not calculate PBDE intakes.

In Tables 3a and 3b, where intakes were only reported in ng/day, a ng/kg/day value was calculated and is presented along with the reported values for ease of comparison. If insufficient data were available, these calculated values are not presented (i.e., where only one congener was measured, or PBDE intakes were only from seafood and not from other food sources). Body weights used for these calculations are Infants 0-6 months, 6.5kg; Infants 7-12 months, 9.2 kg; Toddler 1-4 years, 13.8 kg; Toddler 2-5 years, 15 kg; and child 4-9 years, 20 kg, based approximately on USEPA exposure factors handbook 2011, Table ES-1 (USEPA 2011).

2.6 Limit of Detection

One area of uncertainty when measuring levels of PBDEs and other chemicals is how to handle values below the detection limit. There are various ways that researchers calculate summary values when some measurements fall below the detection limit. Throughout this report, summary measures described as “Lower Bound” assumes results reported as being below the detection limit are zero. “Upper Bound” values assume results reported as being below the detection limit are at the detection limit and “Middle Bound” assumes results reported as being below the detection limit are 50% of the detection limit.

Table 3 Dietary intakes for toddlers and young children

Reference	Exposure Media	Age Group	Reported Exposure Dose	Mid level ng/kg/day	High level ng/kg/day		Comments
Wilford 2005	diet	6–24 months	median- 25 ng/day, P95- 25 ng/day	1.67	1.67	*	Canada. Total intakes from food, dust ingestion, and air inhalation are median 124 ng/day P95 579 ng/day; values 3.5 times higher with dust ingestion rate of 0.2 g/day
Allen 2007	diet	1–5 years	mean –30.9 ng/day	2.24	NA	*	US. 11 PBDEs including BDE 209.
Roosens 2010	diet	3–6 years	median- 2.1 ng/kg/day	2.1	NA		Belgium. Based on food samples and published literature. PBDE 28,47,100,99,154,153,210
Schecter 2006b	diet	2–5 years	"Typical" intake- 2.7 ng/kg/day	2.7	NA		US, based on market basket survey and consumption rates. "Typical" intakes calculated using median consumption rates for foods and mean PBDE concentrations
Johnson-Restrepo 2009	diet	1–5 years	mean- toddlers 2.76 ng/kg/day	2.76	NA		US; based on USDA food intake surveys, measured and previously published PBDE levels. Total intakes from dust ingestion and air inhalation 13.3 ng/kg/day
Harrad 2006	diet (does not include breast milk)	6–24 months	mean- 51.6 ng/day, P95- 51.6 ng/day	3.44	3.44	*	UK; assumed to be 57% of UK adult exposure as described in Harrad 2004. Food contributes 80.8% total PBDE intake in mean dust ingestion scenario, and 54.3% in high dust ingestion scenario
Bocio 2004	diet	4–9 years	mean- 74.6 ng/day	3.73	NA	*	Spain (Catalonia). Based on food samples and survey consumption rate
de Winter-Sorkina 2006	diet	2 years	median- 4.37 ng/kg/day. P95- 7.54 ng/kg/day	4.37	7.54		Netherlands, based on food samples and consumption rates estimated from 2003-4 data, middle estimate

Table 3 Dietary intakes for toddlers and young children

Reference	Exposure Media	Age Group	Reported Exposure Dose	Mid level ng/kg/day	High level ng/kg/day		Comments
UK Food Standards Agency 2006	diet (whole diet)	4–6 years	mean- 11 ng/kg/day, High level- 26 ng/kg/day	11	26		UK. 19 composite food group samples collected for the 2003 and 2004 Total Diet Studies. PBDE-17, 28, 47, 49, 66, 71, 77, 85, 99, 110, 199, 126, 138, 153, 154, 183, 209. Used upper bounds of average and high level (very similar to lower bounds)
UK Food Standards Agency 2006	diet (whole diet)	3.5–4.5 years	mean- 12 ng/kg/day, High level- 29 ng/kg/day.	12	29		UK. 19 composite food group samples collected for the 2003 and 2004 Total Diet Studies. PBDE-17, 28, 47, 49, 66, 71, 77, 85, 99, 110, 199, 126, 138, 153, 154, 183, 209. Used upper bounds of average and high level (very similar to lower bounds)
UK Food Standards Agency 2006	diet (whole diet)	2.5–3.5 years	mean- 13 ng/kg/day, High level- 33 ng/kg/day.	13	33		UK. 19 composite food group samples collected for the 2003 and 2004 Total Diet Studies. PBDE-17, 28, 47, 49, 66, 71, 77, 85, 99, 110, 199, 126, 138, 153, 154, 183, 209. Used upper bounds of average and high level (very similar to lower bounds)
UK Food Standards Agency 2006	diet (whole diet)	1.5–2.5 years	mean- 13 ng/kg/day, High level- 34 ng/kg/day.	13	34		UK. 19 composite food group samples collected for the 2003 and 2004 Total Diet Studies. PBDE-17, 28, 47, 49, 66, 71, 77, 85, 99, 110, 199, 126, 138, 153, 154, 183, 209. Used upper bounds of average and high level (very similar to lower bounds)
FSANZ 2007	diet	2–5 years, female	mean- 1306 ng/day, P95- 2798 ng/day	87.07	186.53	*	Australia; based on food sampling and food survey. Middle bound

Table 3 Dietary intakes for toddlers and young children

Reference	Exposure Media	Age Group	Reported Exposure Dose	Mid level ng/kg/day	High level ng/kg/day		Comments
FSANZ 2007	diet	2–5 years, male	mean- 1474 ng/day, P95- 3468 ng/day	98.27	231.20	*	Australia; based on food sampling and food survey. Middle bound
Lee 2013	diet (seafood)	<2 years	mean- 1.09 ng/kg/day	NA	NA		Korea. Predominant congeners in seafood were BDEs 47, 99 and 100 and those in dust samples were BDE 209
Meng 2007	diet (seafood)	2–5 years	median- 5.2 ng/day males, 1.7 ng/day females. Max: 22.5 ng/day males, 7.4 ng/day females	NA	NA		China. Sigma11 PBDEs
Guo 2010	diet (seafood)	2–5 years	male mean- 6.4 ng/day; female mean- 4.7 ng/day	NA	NA		South China, based on fish tissue data and consumption rates. Includes key tetra-, penta-, and decaBDEs
Bakker 2008	diet	2 years	median- 0.23 ng/kg/day; P90- 0.34 ng/kg/day	NA	NA		Netherlands, based on food samples and consumption rates. PBDE 99 only
Bakker 2008	diet	2 years	median- 1.40 ng/kg/day; P90- 2.69 ng/kg/day	NA	NA		Netherlands, based on food samples and consumption rates. PBDE 47 only

*ng/kg/day calculated using estimated body weights 13.8 kg for child 1-4 years, 15 kg for child 2-5 years, and 20 kg for child 4-9 years.
NA = Not applicable

Table 4 Breast milk and dietary intakes for infants

Reference	Exposure Media	Infant Age	Reported Exposure Dose	Mid level ng/kg/day	High level ng/kg/day		Comments
Tan 2007	diet	6–24 months	mean – 25 ng/day	3.85	NA	*	Singapore.
Meng 2007	breast milk only	0–1 years	median- 48.2 ng/day; Upper- 99.1 ng/day	7.42	15.2	*	China. Sigma7 PBDEs
FSANZ 2007	half infant formula, half solid foods	9 months	mean- 99 ng/day, P95- 247 ng/day	10.76	26.8	*	Australia; based on food sampling and food survey. Middle bound
de Winter-Sorkina 2003	breast milk only	6 months	mean- 15.5 ng/kg/day	15.5	NA		Netherland. Sum of 6 PBDES: 28, 47, 99, 100, 153, 183.
Darnerud 2001	breast milk only	not specified	mean- 110 ng/day	16.92	NA	*	Sweden, calculated from survey data
Roosens 2010	breast milk only	0–6 months	median- 20.7 ng/kg/day; P90- 26.2 ng/kg/day	20.7	26.2		Belgium. Based on milk samples and estimated average 130 g/kg/day milk consumption during first 6 months. PBDE 28,47,100,99,154,153,209. Based on P95 levels. P50 levels are lower
FSANZ 2007	half breast milk, half solid foods	9 months	mean- 274 ng/day, P95- 616 ng/day	29.78	67.0	*	Australia; based on food sampling and food survey. Middle bound

Table 4 Breast milk and dietary intakes for infants

Reference	Exposure Media	Infant Age	Reported Exposure Dose	Mid level ng/kg/day	High level ng/kg/day		Comments
FSANZ 2007	breast milk only	3 months	mean- 324 ng/day	49.85	NA	*	Australia; based on food sampling and food survey. P95 not available. No lower, middle, upper bound available because all pooled samples contained detectable PBDEs.
Johnson-Restrepo 2009	including breast milk as well as other food sources such as milk, meat, fish	<1 yr	mean-infants 78.2 ng/kg/day	78.2	NA		US; based on USDA food intake surveys, measured and previously published PBDE levels. Breast milk accounts for 99% of the exposure. Total intakes from dust ingestion and air inhalation 86.4 ng/kg/day
Jones-Otazo 2005	breast milk only	0–6 months	mean-1800.0 ng/day	276.92	NA	*	Canada, breast milk accounts for 92% of the total including dust inhalation and air ingestion, which is 1963.5 ng/day
Schecter 2006b	breast milk only	0–1 years	"Typical" intake- 307 ng/kg/day	307	NA		US, based on market basket survey and consumption rates. "Typical" intakes calculated using median consumption rates for foods and mean PBDE concentrations
Toms 2009a	breast milk only	0–1 years	range- 10 to 440 ng/day	NA	NA		Australia. BDE-47 only
*ng/kg/day calculated using estimated body weights 6.5 kg for 0-6 months and 9.2 kg for 7-12 months							

Table 5 PBDE congener concentrations (fresh weight pg/g) in foods analysed, 22nd Australian Total Diet Study, 2007c (FSANZ 2007)

	BDE 47	BDE 99	BDE 100	BDE 153	BDE 154	BDE 209	LB PBDE*	UB PBDE*
Water, tap**	<700	<500	<100	<40	<20	<800	0	3.12
Canola Oil	<200	<300	<50	<20	<50	<200	0	1022
Milk, full fat	<20	<30	<3	<3	<2	<6	0	75.3
Milk, full fat (duplicate)	<30	<20	<3	<2	<1	<6	0	72.5
Salt, table, non-iodised	<20	<20	<3	<1	<1	<8	0	68
Milk, modified, low fat (duplicate)	<20	<20	<2	<0.8	<0.8	<6	0	60.2
Milk, modified, low fat	<20	<20	<2	<0.8	<0.8	<5	0	59.5
Potato, cooked	<10	<9	<2	<1	<1	<5	1.7	39.1
Tuna, canned in brine	15	<6	1.9	1.8	2.3	<2	24.5	39.2
Infant Dessert, fruit	33	<10	<2	<0.6	<0.4	<6	52.3	73.8
Carrots, cooked	22	9.4	2	0.75	0.64	5.9	57.6	60.1
Infant Dessert, dairy based	25	19	3.4	1.5	1.3	13	73.0	75.3
Yoghurt, fruit, full fat	21	16	<2	1.6	1.1	42	83.6	95.8
Infant Dinner	34	16	3.5	2.4	1.4	<10	86.8	101.7
Infant Formula, prepared	41	22	4.1	1.5	1.2	<10	88.3	100.6
Infant Cereal, mixed	29	19	3.9	1.7	1.4	15	90.9	91.8
Ice Cream, full fat, vanilla	49	39	6.9	4.6	2.6	<8	104.0	133.8
Cheese, cheddar, full fat	54	50	8.4	6	2.9	<70	121.3	265.7
Peanut butter	85	<30	<5	<2	<2	<30	125.4	232.4
grilled Margarine	74	68	11	5.2	4.1	<100	164.1	383.5

Table 5 PBDE congener concentrations (fresh weight pg/g) in foods analysed, 22nd Australian Total Diet Study, 2007c (FSANZ 2007)

	BDE 47	BDE 99	BDE 100	BDE 153	BDE 154	BDE 209	LB PBDE*	UB PBDE*
Fish fillets	79	24	16	4.3	9.3	28	172.6	208.0
Butter, regular	95	76	<8	10	5.1	<70	189.1	357.8
Beef steak, grilled	65	51	11	5.4	3.6	26	189.8	196.1
Bread, white	73	65	14	7.5	4.5	19	194.4	225.4
Sheep liver	110	43	<6	4.4	2.9	<50	231.7	358.7
Coconut, desiccated	110	<30	<7	<3	<3	<40	243.6	364.6
Chicken Breast	40	48	10	8.4	5.6	120	285.3	288.9
Potato crisps	140	<40	<4	5.3	<2	46	300.4	374.8
Chocolate, milk	<20	<20	<4	<2	<1	290	309.0	377.2
Pizza	110	140	15	24	13	<60	324.3	409.3
Sausage, beef (duplicate)	90	80	8.6	8.6	5.5	120	343.2	364.2
Sausage, beef	98	88	11	9	5.7	110	352.8	373.8
Lamb Chops, loin,	160	41	14	6.1	2.7	23	360.9	371.2
Hamburger	100	110	11	21	9.7	77	361.8	372.3
Cream, pure (not thickened)	200	160	16	17	10	<30	469.0	512.5
Bacon	160	150	30	22	12	110	514.3	568.3
Pork Chops, grilled (duplicate)	220	260	36	50	26	32	681.6	702.4
Pork Chops, grilled	230	280	54	48	27	<10	693.4	720.2
Eggs, boiled	170	360	100	99	63	84	909.7	954.6

*Lower Bound (LB) and Upper Bound (UB) PBDE sums include all congeners, not just the six listed here. However, the six congeners listed here are the primary contributors to the totals.
**Water was measured in pg/mL

2.7 Exposure Values for Diet and Breast Milk

For this risk assessment, values from the UK Food Standards Agency 2006 report were used (UK Food Standards Agency 2006). These values were based on 19 composite food group samples collected for the 2003 and 2004 Total Diet Studies. Congeners measured included BDE-17, 28, 47, 49, 66, 71, 77, 85, 99, 110, 199, 126, 138, 153, 154, 183, and 209. PBDE intakes from diet were reported separately for toddlers (1.5–2.5 years), toddlers (2.5–3.5 years), toddlers (3.5–4.5 years), and young children (4–6 years). PBDE intakes were similar in all children's age categories and the highest values were used in this analysis, which allows for a conservative assessment of risk. Upper bound calculations were used; these were very similar to lower bound values.

For infants 6–12 months old, diet was assumed to consist of half breast milk and half toddler diet (using data for the youngest toddlers 1.5–2.5 years old from the UK Food Standards Agency (Hites 2004; UK Food Standards Agency 2006; Trudel et al 2011). For infants 0–6 months old, diet was assumed to consist entirely of breast milk. Older infants ages 6–12 months were split out separately in this risk analysis because of the hypothesized elevated exposures that could result from a cohort that is exposed both via breast milk consumption as well as via dust ingestion from crawling around on the floor.

For the purposes of this risk assessment, PBDE concentrations were summed by congener type based on data reported in breast milk in New Zealand (Mannetje et al 2013) using the lower bound. For infants, breast milk consumption was assumed to be 770 ml/day (USEPA 2011), and to be composed of 3.7% lipid, the average value reported by Harden 2005 in Australia (Harden et al 2005).

2.8 House Dust

Estimates of mean intake of PBDEs from dust and diet in New Zealand infants and toddlers from 3 months of age up to 24 months were made by Coakley et al (2013). In their exposure assessment of 33 homes, intakes of tri-hexa BDE congeners were on average 21.4 ng/kg BW/d for 3–6 month olds, while BDE-209 intake was estimated to be 11.7 ng/kg BW/d on average. For the 12–24 month old toddlers, intakes for the tri- to hexa- BDE congeners had fallen to 0.6 ng/kg BW/d, but were 13.2 ng/kg BW/d for BDE-209 (Coakley et al 2013). These exposure estimates were compared to US EPA RfD values of 100, 200, or 7000 ng/kg BW/day for tri- through pentabrominated congeners, hexabrominated congeners, or decaBDE, respectively. These authors concluded that the average exposure to a toddler did not present significant risks when using the US EPA RfD as a comparison. Exposures to PBDEs from riding in cars or car seats were not included in their assessment, and high end exposures were not characterised. In this study, like most others, BDE-209 concentrations were the most variable, with maximal BDE-209 concentrations roughly 10-fold higher than the reported mean values (Coakley et al 2013).

It should be noted that dust intake estimates vary significantly in the literature. The US EPA Exposure Factors Handbook employs a default value of 30 mg/day for infants less than 12 months old, and 60 mg/day for the 12–24 month old age groups (USEPA 2011). However, Lorber cites significant uncertainty in these house dust consumption values, and employed a more precautionary value of 100 mg/day (Lorber 2008). However, there is scant empirical data on actual observed dust ingestion in infants and toddlers. Therefore, significant uncertainty exists surrounding the estimation of a high end intake scenario. Intake values as high as 200 mg/day have been proposed (Harrad et al 2008).

Table 6 House dust PBDE concentrations - international comparisons.

Reference	House dust PBDE (mean and max) (ng/g)	PBDE congeners	Location
Coakley et al 2013	2,756 (29,213)	17, 28, 47, 49, 66, 85, 99, 100, 153, 154, 183, 196, 197, 203, 206, 209	New Zealand (Wellington, Christchurch, Wairarapa, North Canterbury)
Sjodin et al 2008	1,200 (13,000)	47, 99, 100, 153, 154, 183, 209	Australia
Toms et al 2009c	904 (3,062)	47, 99, 100, 153, 183, 206, 207, 209	Australia
Sjodin et al 2008	10,000 (54,000)	47, 99, 100, 153, 154, 183, 209	UK
Harrad et al 2008	45,000 (520,000)	28, 47, 99, 100, 153, 154, 183, and 209	Birmingham, UK
Harrad et al 2008	1,400 (3,500)	28, 47, 99, 100, 153, 154, 183, and 209	Toronto, Canada
Wilford et al 2005	1,800 (170,000)	28, 47, 99, 100, 153, 154, 183, and 209	Ottawa, Canada
Gevao et al 2006	233	28, 47, 85, 99, 100, 153, 154, 183, 209	Kuwait
Harrad et al 2008	4,800 (17,000)	28, 47, 99, 100, 153, 154, 183, and 209	Amarillo and Austin, Texas, USA
Imm et al 2009	3,385	47, 99, 100, 153, 154, 206, 207, 209	Wisconsin, USA
Sjodin et al 2008	4200 (29,000)	47, 99, 100, 153, 154, 183, 209	USA

2.9 Car Dust

Exposures of New Zealand infants and toddlers to PBDEs in the automobile environment were estimated in an earlier report (Fowles and Morgott 2013). According to this assessment, uptake parameters for PBDEs in automobiles from dermal, oral and inhalation routes are described by the following equations (Fowles and Morgott 2013):

$$Uptake_{dermal} = \frac{C_{dust} \times BSA_{exposed} \times SAS_{skin} \times t_{car} \times f_{dermal}}{BW} \quad (1)$$

$Uptake_{dermal}$ – internal dose by dermal route (ng/kg/day)
C_{dust} – car dust concentration (ng/g)
$BSA_{exposed}$ – exposed body surface area (cm^2)
SAS_{skin} – dust adhering to skin (mg/cm^2)
t_{car} – time spent in car (min/day)
f_{dermal} – dermal absorption factor (unitless)
BW – body weight (kg)

C_{dust} = 95% LCL, mean, 95% UCL
t_{car} = 95% LCL, mean, 95% UCL
$BSA_{exposed}$ =2564 cm^2
SAS_{skin} = 0.096 mg/cm^2
f_{dermal} = 0.62
BW = 9.2 kg

$$Uptake_{oral} = \frac{C_{dust} \times R_d \times t_{car} \times f_{oral}}{BW} \quad (2)$$

$Uptake_{oral}$ – internal dose by oral route (ng/kg/day)
C_{dust} – car dust concentration (ng/g)
t_{car} – time spent in car (min/day)
R_d – daily dust ingestion (mg/day)
f_{oral} – oral absorption factor (unitless)
BW – body weight (kg)

C_{dust} = 95% LCL, mean, 95% UCL
t_{car} = 95% LCL, mean, 95% UCL
R_d = 60 mg/day
f_{oral} = 82.2
BW = 9.2 kg

$$Uptake_{inhalation} = \frac{C_{air} \times VR_{lung} \times t_{car}}{BW} \quad (3)$$

$Uptake_{inhalation}$ – internal dose by inhalation route (pg/kg/day)
C_{air} – car vapor concentration (pg/m^3)
t_{car} – time spent in car (min/day)
VR_{lung} – pulmonary ventilation rate (m^3/day)
BW – body weight (kg)

C_{air} = 95% LCL, mean, 95% UCL
t_{car} = 95% LCL, mean, 95% UCL
VR_{lung} = 8.9 m^3/day
BW = 9.2 kg

From these parameters, using defined and published input parameters, mean combined route (oral, dermal, and inhalation) intakes of ΣPBDE were 10.6 ng/kg/d, while the maximum was 92.1 ng/kg/d for a child (6–11 months) of 9.2 kg (Fowles and Morgott 2013).

The estimated dust PBDE concentrations in vehicles from the assessment by Fowles and Morgott were used in the present assessment, summed across the three congener groupings as shown in Table 7.

Table 7 Dust concentrations used for intake calculations**.**

	House Dust PBDE concentrations[1] (ng/g)		Car dust PBDE concentrations[2] (ng/g)	
BDEs	**mean**	**max**	**mean**	**max**
ΣTri-penta	97.4	374.8	1573	11220
ΣHexa-nona	153	1443.6	2519	19062
Deca	2505.2	27394.3	31403	264758

[1] Coakley et al 2013
[2] Fowles and Morgott, 2013

2.10 Other sources of exposure

A total of 15 published papers were identified describing intakes of PBDEs from sources other than diet and dust. These papers are reviewed in Table 8. None of these studies were conducted in New Zealand. The routes of exposures described included air and vapor inhalation, dermal ingestion, oral contact with toys, and water ingestion. These intake sources are expected to be very minor contributors to PBDE levels in New Zealand, and particularly in the most vulnerable populations (infants, toddlers, and young children), who may be exposed to high levels of PBDEs in breast milk, and may also experience high intakes through dust ingestion, due to increased hand-to-mouth behaviours and time spent crawling around on the floor.

Wilford et al (2005) sampled air in 74 homes in Ottawa, Canada, during the winter of 2002–2003 using polyurethane foam passive air samplers and compared air intakes to intake from house dust and diet. For children ages 6–24 months, they reported a median air intake of 0.33 ng/day and a 95th percentile of 3.6 ng/day. Based on the median levels found in these homes, the authors concluded that the daily exposure via the inhalation pathway for children would be 0.26% of overall daily intake assuming a mean dust ingestion of 55 mg/day, and 0.085% assuming a high dust ingestion of 200 mg/day. (For adults, the estimated maximum daily exposure via the inhalation pathway was about 4% of overall daily intake). Also in Canada, Jones-Otazo et al (2005) found a similar PBDE intake of 0.8 ng/day from home air inhalation among infants 0–6 months.

In the U.K., Harrad et al (2006) reported a median PBDE intake among toddlers (ages 6-24 months) from air inhalation of 0.16 ng/day, with a 95th percentile of 1.7 ng/day. This accounted for 0.3% of total PBDE intake from diet, dust ingestion, and air inhalation in a mean dust ingestion scenario (median total intake 56.6 ng/day) and 0.2% of total PBDE intake in a high dust ingestion scenario (median total intake 69.2 ng/day). (For adults, the estimated median daily exposure via the air inhalation pathway was about 1% of overall daily intake in either dust ingestion scenario) (Harrad et al 2006). In U.K. cars, Harrad and Abdallah (2011) found an intake among toddlers from inhalation of car dust to be 0.21 ng/day for BDE-209 only, compared to 390 ng/day from ingestion of car dust.

In Belgium, Roosens et al (2010) reported a total intake of 0.057 ng/kg/day from indoor, outdoor, and vehicle air inhalation among children ages 3–6 years old. Indoor air was the primary source of this exposure. The combined indoor, outdoor, and vehicle air intake accounted for less than 3% of the total intake for children ages 3–6 from all sources, including dust ingestion and diet, which was 2.19 ng/kg/day, and was driven by dietary intake.

Petito Boyce et al (2009) investigated intake of BDE-209 via dermal contact with dust. They found the highest intake in the U.K (mean dermal intake 51 ng/kg/day, compared to 400 ng/kg/day from ingestion) and lower intakes elsewhere in Europe (mean dermal intake 0.31 ng/kg/day, compared to 1.9 ng/kg/day from ingestion) and in North America (mean dermal intake 0.79 ng/kg/day, compared to 4.8 ng/kg/day from ingestion) (Petito Boyce et al 2009).

In the US, Johnson-Restrepo at al. (2009) reported PBDE intakes from air inhalation in children that decreased with age, with an intake of 0.23 ng/kg/day in older children ages 6–11 years, 0.29 ng/kg/day among toddlers ages 1–5, and 0.60 ng/kg/day among infants. Dermal absorption of dust in children also decreased with age, with an intake of 0.46 ng/kg/day in older children ages 6–11 years, 0.70 ng/kg/day among toddlers ages 1-5, and 0.77 ng/kg/day among infants (Johnson-Restrepo et al 2009).

One contributing source of PBDE exposures to infants and toddlers is plastic toys. Ionas and colleagues found BDE-99 and BDE-209 in toys found in Belgium made from recycled plastics with a maximum of 140 μg/g, and BDE-209 as the dominant congener (Ionas et al 2014). The transfer rate of PBDEs from toys to an ingested dose for a toddler mouthing the toys or dermally and/or orally from hand contact, is not precisely known, however, it is likely a significant source for some children. Chen and colleagues (2009) measured median PBDE concentrations of 53,000 ng/g and maximum concentrations of 4,232,000 ng/g in toys purchased from South China's Guangdong province. Most of the PBDE residues were from BDE-209. Mouthing of these toys was determined to be the most significant contributor to exposure in 3–18 month old children, compared with inhalation, dermal contact, or hand to mouth ingestion. Mouthing exposure was estimated to be 0.005, 3.9, and 5.0 ng/kg/day for the tri-penta, hexa-nona, and deca BDE congeners, respectively. Because it is estimated that 70% of the world's toys are manufactured in China, these findings are of likely significance to New Zealand and other parts of the world.

Table 8 Other intakes for infants and young children

Reference	Exposure Media	Age Group	Exposure Dose	Comments
Wilford 2005	air inhalation	Children ages 6–24 months	median- 0.33 ng/day, P95-3.6 ng/day	Canada; air is 0.26% of overall daily intake assuming a mean dust ingestion of 55 mg/day, and 0.085% assuming a high dust ingestion of 200 mg/day
Harrad 2006	air inhalation	Children ages 6–24 months	median- 0.16 ng/day, P95-1.7 ng/day	UK; air is 0.2-0.3% of total PBDE intake
Meng 2007	air inhalation	Infants (0–1 yrs)	mean – 2.7 ng/day; upper – 31.8 ng/day	China, Sigma11 PBDEs
Meng 2007	air inhalation	Toddlers and young children (2–5 yrs)	mean – 4.6 ng/day; upper – 53.7 ng/day	China, Sigma11 PBDEs
Meng 2007	air inhalation	Children and adolescents (6-17 years)	mean – 8.4 ng/day males, 7.1 ng/day females; upper – 98.9 ng/day males, 83.3 ng/day females	China, Sigma11 PBDEs
Toms 2009a	air inhalation	Infants (0–1 yrs)	range- 0.2–1.1 ng/day	Australia, BDE-47 only
Johnson-Restrepo 2009	indoor air inhalation	Infants (<1 yr)	median- 0.60 ng/kg/day	US; based on EPA exposure factors for ingestion and inhalation, measured and previously published PBDE levels
Johnson-Restrepo 2009	indoor air inhalation	Toddlers (1–5 yr)	median- 0.29 ng/kg/day	US; based on EPA exposure factors for ingestion and inhalation, measured and previously published PBDE levels

Table 8 Other intakes for infants and young children

Reference	Exposure Media	Age Group	Exposure Dose	Comments
Johnson-Restrepo 2009	indoor air inhalation	Children (6–11 yr)	median- 0.23 ng/kg/day	US; based on EPA exposure factors for ingestion and inhalation, measured and previously published PBDE levels
Roosens 2010	outdoor air inhalation	Children (3–6 yrs)	0.51 E-3 ng/kg/day	Belgium, includes BDE 209 along with 5 other congeners. Based on P50 levels from the literature
Roosens 2010	home air inhalation	Children (3–6 yrs)	54.6 E-3 ng/kg/day	Belgium, includes BDE 209 along with 5 other congeners. Based on P50 levels from the literature
Jones-Otazo 2005	home air inhalation	Infants (0–6 months)	mean- 0.8 ng/day	Canada. Outdoor air was also evaluated but contributed ~0
Allen 2007	air inhalation	Child (1–5–yrs)	mean – 5.2 ng/day	US. 11 PBDEs including BDE 209.
Tan 2007	air inhalation	Infant (6-24 months)	mean – 0.33 ng/day	Singapore.
Roosens 2010	vehicle air inhalation	Children (3–6 yrs)	1.81 E-3 ng/kg/day	Belgium, includes BDE 209 along with 5 other congeners. Based on P50 levels from the literature
Harrad and Abdallah 2011	inhalation of vehicle dust	Toddler (age not specified)	mean – 0.21 ng/day	UK, BDE 209 only
Chen 2010	inhalation from electronic household products	Infant (0–3 yrs)	mean – 0.61 ng/kg/day	China, include ΣPBDE for 16 congeners

Table 8 Other intakes for infants and young children

Reference	Exposure Media	Age Group	Exposure Dose	Comments
Chen 2010	inhalation from electronic household products	Child (4–12 yrs)	mean – 0.32 ng/kg/day	China, include ΣPBDE for 16 congeners
Hays 2006	inhalation of particulates released from electronics	Infants and young children (0–2 yrs)	midrange- 0.31 ng/kg/day; upper- 0.63 ng/kg/day	US, deca-BDE only
Petito Boyce 2009	dermal absorption of dust	Child (3–4 yrs)	mean – 51 ng/kg/day; 95th – 126 ng/kg/day	United Kingdom, BDE 209 only
Petito Boyce 2009	dermal absorption of dust	Child (3–4 yrs)	mean – 0.79 ng/kg/day; 95th – 3.1 ng/kg/day	North America, BDE 209 only
Petito Boyce 2009	dermal absorption of dust	Child (3–4 yrs)	mean – 0.31 ng/kg/day; 95th – 1.2 ng/kg/day	mainland Europe, BDE 209 only
Johnson-Restrepo 2009	dermal absorption of dust	Infants (<1 yr)	median- 0.77 ng/kg/day	US; based on EPA exposure factors for ingestion and inhalation, measured and previously published PBDE levels
Johnson-Restrepo 2009	dermal absorption of dust	Toddlers (1–5 yr)	median- 0.7 ng/kg/day	US; based on EPA exposure factors for ingestion and inhalation, measured and previously published PBDE levels

Table 8 Other intakes for infants and young children

Reference	Exposure Media	Age Group	Exposure Dose	Comments
Johnson-Restrepo 2009	dermal absorption of dust	Children (6–11 yr)	median- 0.46 ng/kg/day	US; based on EPA exposure factors for ingestion and inhalation, measured and previously published PBDE levels
Chen 2009	inhalation, dermal, and oral from toys	Infant (3–12 months) toddlers (1–3 yrs)	mean – 107 ng/kg/day; mean – 176 ng/kg/day	China, include penta-decaBDE exposures
Hays 2006	oral from mouthing electronics	Infants and young children (0–2 yrs)	midrange- 4.3 ng/kg/day; upper- 250 ng/kg/day	US, deca-BDE only

[Intentionally Blank]

3. RESULTS

3.1 Diet

Values used in this semi-quantitative risk assessment were obtained from total dietary intakes calculated by the UK Food Standards Agency (UK Food Standards Agency 2006). PBDE intakes were similar in all age categories 1.5–4.5 and the highest values were used in this analysis (Table 9). PBDEs were found in all 19 food groups. Meats and fish were the most significant contrinbutors to PBDE intakes, with PBDE 209 higher in meat, and PBDE 47 highest in fish.

Table 9 Risk Assessment Parameters: Dietary Intake, ng/kg/day. (Based on UK Food Standards Agency 2006)**.**

	Mean	Max
Sum tri-penta	2.96	5.68
Sum hexa-nona	0.39	0.87
Sum deca	10	30

- Intakes for each PBDE congener were summed across food groups, as presented in UK FSA 2006.

The intake values used in this risk assessment are similar to but somewhat higher than most of the other dietary intakes presented in Table 3, including other data from the UK and Europe, North America, and Asia. This allows for a conservative assessment of risk. One exception to this general pattern is a 2007 report by Food Standards of Australia and New Zealand (FSANZ 2007), which contains intake values much higher than those observed elsewhere. Furthermore, the data for the 2007 FSANZ report displays a different age pattern than the other studies in Table 3 and 3b, showing dietary PBDE intakes increasing with increasing child age. This report was considered an outlier and so was not used in risk assessment calculations, and is discussed in more detail in the discussion section of this report.

3.2 Breast milk and solid food intake estimates

The analysis of the data reported by Mannetje et al (2013), yielded a mean (max) breast milk PBDE content of 4.07 ng/g lipid (12.42 ng/g lipid) for the tri-penta BDEs, 1.17 ng/g lipid (5.72 ng/g lipid) for the hexa-nona BDEs, and 0.35 ng/g lipid (3.14 ng/g lipid) for the deca BDE (Table 10). For these calculations, results reported as being below the detection limit are set to zero (lower bound).

Table 10 Breast milk PBDE content, ng/g lipid (from Mannetje et al 2013).

PBDEs	mean	max
Sum tri-penta	4.07	12.42
Sum hexa-nona	1.17	5.72
Sum deca	0.35	3.14

Using the dietary PBDE concentration data from UK Food Standards Agency, 2006 and breast milk concentrations from Mannetje et al 2013, intake scenarios were constructed. The intake estimates of PBDEs for the three exposure scenarios are presented in Table 11. The tri- penta BDE congeners are the biggest contributor to PBDE intake from diet, both in terms of total weight of PBDE congeners (Table 11), as well as when considering potency of the congener groups (Table 13).

Table 11 PBDE intake estimates (ng/kg bw/day) to infants and toddlers from diet, including breast milk and solid food, based on data from UK Food Standards Agency 2006 and Mannetje et al 2013.

	Intakes Using Mean PBDE (ng/kg/d)			Intakes Using Maximum PBDE (ng/kg/d)		
	0–6 months					
	Σtri-penta	Σhexa-nona	Deca	Σtri-penta	Σhexa-nona	Deca
Breast milk	17.83	5.13	1.55	54.45	25.09	13.76
	6–12 months					
Breast milk	8.91	2.57	0.78	27.22	12.54	6.88
Solid foods	1.48	0.20	5.00	2.84	0.44	15.00
	1–4 years					
Solid foods	2.96	0.39	10.00	5.68	0.87	30.00

3.3 Dust PBDE intakes

Using the limited NZ-specific house dust PBDE concentration data from Coakley and associates, and the car dust PBDE exposure estimates from Fowles and Morgott, dust intake scenarios were constructed. The intake estimates of PBDEs for the three exposure scenarios are presented in Table 12. The sum total PBDE exposures in our analysis indicate that infants less than 1 year of age are expected to have higher daily doses, on a body weight basis, than toddlers 1–4 years old. (Table 12).

Table 12 shows that, daily intake, in terms of the total weight of PBDE congeners, is driven by DecaBDE, followed by the hexa-nona group. However, when considering potency of the congener groups, the PBDE congener group contributing the most to risk calculations in Table 13 were the tri- penta PBDE congeners (see Appendix A).

Risk estimates varied from 5 to 24% of the summed hazard index (HI) for an expected typical scenario using mean PBDE concentrations in dust. However, since typical and extreme dust intakes are not well characterised in the literature, an extreme intake case is presented which assumes the maximal PBDE dust concentrations found in NZ, combined with a 200 mg/day dust intake. Additionally, the extreme case considers maximal PBDE concentrations in breast milk and diet. In this case, the HI exceeds 1.0 for the 0-6 month infant and is shown in boldface type (Table 13). Even if one assumes a dust intake of 60 mg/day for a 0–6 month old infant, houses with the highest PBDE concentrations in dust, and also with high PBDE concentrations in breast milk may approach unacceptable risk levels, as shown by the HI of 0.9 in Table 13. The use of maximum reported PBDE concentrations in dust and breast milk results in an approximately 4-fold increase in PBDE risk estimates, the highest being in the 0–6 month old group with a combined HI = 0.9.

Table 12 PBDE intake estimates (ng/kg bw/day) to infants and toddlers from house and car dust based on data from Coakley et al 2013 and Fowles and Morgott 2013.

	Intakes Using Mean PBDE in Dust (ng/kg/d)			Intakes Using Maximum PBDE in Dust (ng/kg/d)		
	0–6 months					
	Σtri-penta	Σhexa-nona	Deca	Σtri-penta	Σhexa-nona	Deca
House Dust (60 mg dust/d)	0.68	1.06	17.38	2.60	10.02	190.07
House Dust (200 mg dust/d)	2.25	3.54	57.94	8.67	33.39	633.58
Car Dust (60 mg dust/d)	0.99	1.59	19.81	7.08	12.02	167.00
Car Dust (200 mg dust/d)	3.31	5.30	66.03	23.59	40.08	556.67
	6–12 months					
House Dust (60 mg dust/d)	0.50	0.79	12.86	1.92	7.41	140.64
House dust (200 mg dust/d)	1.67	2.62	42.87	6.41	24.70	468.80
Car Dust (60 mg dust/d)	0.34	0.54	6.72	2.40	4.08	56.64
Car Dust (200 mg dust/d)	1.12	1.80	22.39	8.00	13.59	188.78
	1–4 years					
House Dust (100 mg dust/d)	0.55	0.87	14.27	2.13	8.22	156.00
House Dust (200 mg dust/d)	1.11	1.74	28.53	4.27	16.44	311.99
Car Dust (100 mg dust/d)	0.39	0.62	7.77	2.78	4.72	65.55
Car Dust (200 mg dust/d)	0.78	1.25	15.55	5.56	9.44	131.10

Table 13 Summary of Hazard Indices from Dietary and Dust Exposure Scenarios. Hazard Index >1 indicates a potentially hazardous exposure.

	Average PBDE Values in Dust and Diet		Max PBDE Values in Dust and Diet	
Age group	**Typical Dust Intake[1]**	**High Dust Intake[2]**	**Typical Dust Intake[1]**	**High Dust Intake[2]**
0–6 months	0.2	0.3	0.9	**1.5**
6–12 months	0.1	0.2	0.5	0.8
1–4 years	0.1	0.1	0.2	0.4

[1]Typical dust intake assumed to be 60 mg/day for <1 year olds; 100 mg/day for 1–4 year olds. (USEPA 2011; Coakley et al 2013)

[2]High dust daily intake from all sources assumed to be 200 mg/day (Harrad et al 2008)

3.4 Risk Characterisation

The use of hazard indices has allowed for a comparison of the relative contribution of the exposure routes reviewed in this report on total risk from PBDEs. The charts shown in Figures 3–8 illustrate the changing contributions from diet, breast milk, and dust to infant and toddler PBDE risks under different scenarios. For infants, exposure via breast milk is the dominant source of PBDE exposures, accounting for 83% of the PBDE risk (Fig 3). Even if one assumes maximum PBDE concentrations in house or car dust, breast milk still accounts for 71% of the BDE HI. As the child ages (Figs 4 and 5), the overall HI declines due to lower overall intake hazard of the congener profile, with increasing amounts of BDE 209 relative to BDE 47 and BDE 99, and dust becoming a significant exposure pathway in ages 1–4 years. Overall, the 0–6 month old exposure scenario presents with the highest mean risk estimate at HI = 0.2 (Fig 3).

When considering high end, worst case exposure situations, the overall HI was 0.9 for 0–6 month old infants, with 72% of the risk accounted for by breast milk (Fig 6). As maximal values in both diet and dust are factored into the risk calculations, the relative importance of dust increases, yet still remains a lesser contributor to overall risk for the youngest age groups. Only in the 1–4 year old age group does house and car dust dominate the worst case risks, but in this scenario, the HI was 0.2, significantly less than 1.0.

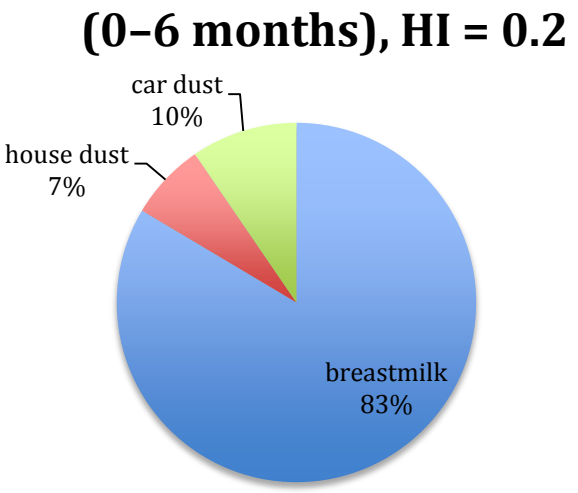

Figure 3 Relative contribution to PBDE risks for infants, age 0–6 months, assuming mean dietary and dust intake levels combined with mean PBDE concentrations.

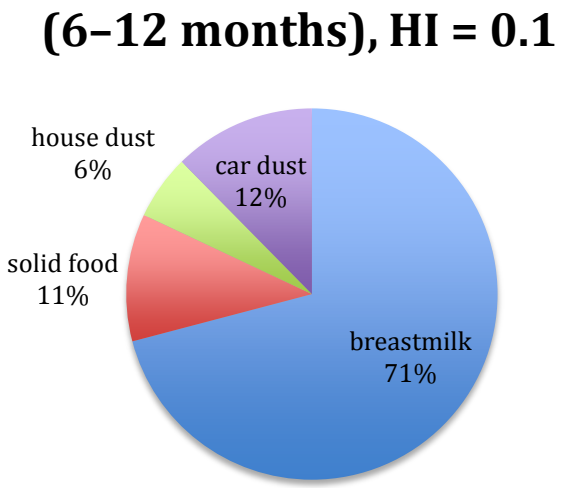

Figure 4 Relative contribution to PBDE risks for infants and toddlers, age 6–12 months, assuming mean dietary and dust intake levels combined with mean PBDE concentrations.

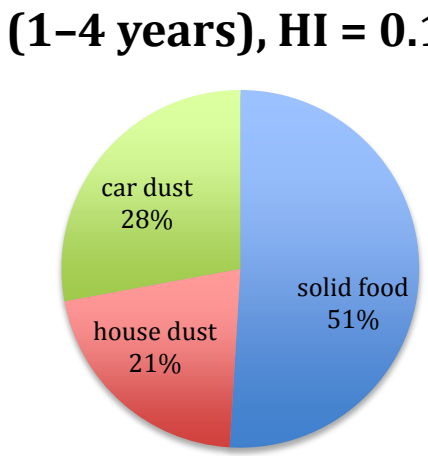

Figure 5 Relative contribution to PBDE risks for children, age 1–4 months, assuming mean dietary and dust intake levels combined with mean PBDE concentrations.

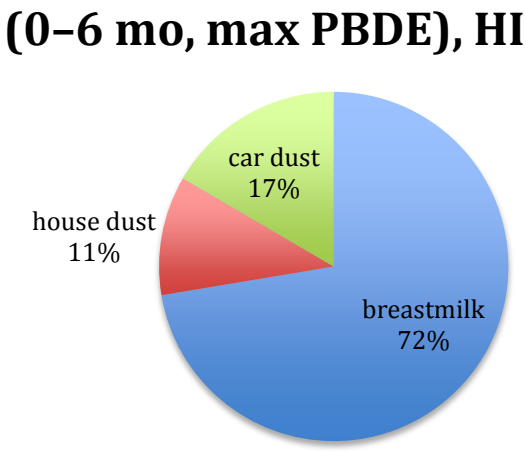

Figure 6 Relative contribution to PBDE risks for infants, age 0–6 months, assuming mean dietary and dust intake levels combined with maximum PBDE concentrations.

(6–12 mo, max PBDE), HI = 0.5

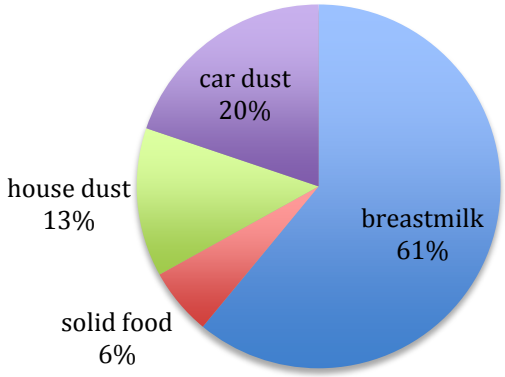

Figure 7 Relative contribution to PBDE risks for infants and toddlers, age 6–12 months, assuming mean dietary and dust intake levels combined with maximum PBDE concentrations.

(1–4 yr, max PBDEs), HI = 0.2

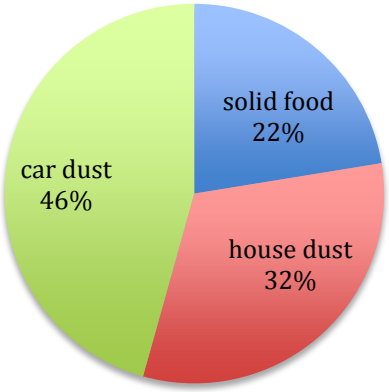

Figure 8 Relative contribution to PBDE risks for children, age 1–4 months, assuming mean dietary and dust intake levels combined with maximum PBDE concentrations.

4. DISCUSSION

We present here a semi-quantitative risk assessment of PBDEs on New Zealand infants and young children. Rather than simply presenting summed PBDE exposure estimates, which ignore relative potencies, we include a grouping by congener type with a dose-addition Hazard Index approach to illustrate from where the most significant exposures originate from a risk standpoint. Our analysis shows that the relative contribution to risk of PBDE exposure is significant for both diet and dust exposures, with the latter becoming more prominent as breastfeeding is replaced by solid foods, and the contribution of dust PBDEs increases in significance. While total exposure (ng/kg/day) of PBDEs is highest in the toddler age group, taking into account the relatively lower potency of BDE-209, the actual risk estimate (HI) is less than in breastfeeding infants. In our study, total exposures across the three age groups show that infants less than 1 year old are likely to have higher risks than toddlers aged 1–4 years old.

Previous studies had concluded that PBDE exposure in infants and young children was higher than that in adults (Fischer et al 2006; Toms et al 2008; Lunder et al 2010), and the high levels of PBDEs observed in breast milk samples and breastfeeding infants worldwide makes our finding of the highest exposures in the youngest infants seem plausible. However, this finding contrasts with the only study to use 6 month age brackets to calculate exposure in children from birth to age 4, by Toms et al (2009b), who found in biomonitoring of 4 PBDE congeners (BDE-47, BDE-99, BDE-100, and BDE-153) that peak exposures in Australian children occurred at 2.6–3 years of age (Appendix B). Notably, this pattern was not observed in modeled data in an earlier study by the same authors (Toms et al 2008), and in the biomonitoring study, the age pattern was not observed for polychlorinated biphenyls and persistent pesticides in these same samples of children. This discrepancy may indicate that the routes of exposure are different for PBDEs than for traditional persistent organic pollutants, and this may not have been fully captured in the current modeled analysis. Furthermore, our modeled analysis may result in an underestimate of exposure in toddlers age 1–4 years, as suggested by Toms et al regarding their earlier modeled analysis (Toms et al 2008; Toms et al 2009b), because the half-life values used may be inaccurate for children, and missing PBDE sources may exist that contribute to intake levels during early years. Other reasons for the discrepancy might include lower breastfeeding rates or breast milk PBDE concentrations in the population investigated by Toms and colleagues, or an overall higher PBDE content in New Zealand house or car dust generally as compared with Australia, as has been described by Coakley et al (2013).

In Australia, Toms et al (Toms et al 2009a) reported PBDE-47 intake from breast milk at a range of 10 to 440 ng/day from 10 breast milk samples. The highest intake from breast milk was described in the US and Canada (Jones-Otazo et al 2005; Schecter et al 2006b; Johnson-Restrepo et al 2009). Jones-Otazo et al (Jones-Otazo et al 2005) reported mean PBDE intake from breast milk of 1800.0 ng/day among infants age 0–6 months. Breast milk accounted for 92% of the total exposure including dust ingestion and air inhalation.

In North America, four studies (Jones-Otazo et al 2005; Schecter et al 2006b; Lorber 2008; Johnson-Restrepo et al 2009) described dietary PBDE intakes in infants and children, and found relatively low levels of intakes compared to those reported in Australia, the U.K, and other places. This may seem surprising considering the well-documented higher levels observed in North America (Hites, 2004; Trudel et al 2011), and may indicate that dust ingestion is a more important exposure route than diet in North America. Schecter et al conducted a market basket survey of U.S. food and used mean PBDE concentration of sample foods to obtain a PBDE intake estimate for a “typical” man or woman (one whose food intake is at the 50th percentile) for each age group. PBDE intakes reported in toddlers were 2.7 ng/kg/day (Schecter et al 2006b). Allen et al reported a mean PBDE intake of 30.9 ng/day along toddlers in the U.S. (Allen et al 2007). Johnson-Resrepo et al reported mean PBDE intake separately for dust (house and car), ingestion, dermal uptake, and food/milk consumption. These estimates were based on US EPA exposure factors for ingestion and inhalation, United States Department of Agriculture (USDA) food intake surveys, and measured and previously published PBDE levels. For food and milk intakes alone, mean intake level reported in U.S. infants was 78.2 ng/kg/day, and lower levels were reported in U.S. children and toddlers (2.76 ng/kg/day and 1.92 ng/kg/day, respectively (Johnson-Restrepo et al 2009). Among toddlers ages 6–24 months, Wilford et al (Wilford et al 2005) reported median combined intake from food, dust ingestion, and air inhalation of 124 ng/day, with food contributing 25 ng/day, or 20% of total intake. When a higher dust ingestion rate of 0.2 g/day was used, PBDE intake values are 3.5 times higher, at 385 ng/day, with food contributing the same 25 ng/day, or 6.5% of total intake (Wilford et al 2005).

Dietary intakes reported from Asia were generally difficult to compare with values from other places due to differences in methodology. In China, Meng et al (Meng et al 2007) reported a median intake of 48.2 ng/day from breast milk among nursing infants and 5.2 and 1.7 ng/day among male and female toddlers, respectively, from seafood consumption. Guo et al (2010) reported a dietary intake from seafood among children ages 2-5 of 6.4 ng/day for males and 4.7 ng/day for females. In Korea, Lee at al. (2013) reported PBDE intake of 11.4 ng/kg/day among toddlers (<2 years) from seafood consumption and dust ingestion. In Singapore, Tan et al (2007) reported a median intake of 25 ng/day from breast milk among nursing infants.

In Europe, estimated PBDE intakes from diet among infants and children were generally higher than those reported in North America. Harrad et al (2006) reported a mean intake of 63.8 ng/kg/day among U.K. toddlers (6–24 months) from diet, dust ingestion, and air inhalation in a mean dust ingestion scenario, with 81% of that (51.6 g/day) coming from diet. Using a high dust ingestion scenario, food contributed 54% of total PBDE intake. In Belgium, median PBDE intake from diet among children ages 3–6 years was 2.1 ng/kg/day, and from breast milk among infants ages 0–6 months was 20.7 ng/kg/day (Roosens et al 2010). In Spain, mean PBDE intake among children ages 4–9 years was 74.6 ng/kg/day (Bocio et al 2004). In Sweden, mean PDBE intakes among infants from breast milk were estimated at 110 ng/kg/day (Darnerud et al 2001). In the Netherlands, estimated PBDE exposures were 4.37 ng/kg/day among toddlers (aged 2 years), and 2.26 ng/kg/day among children (aged 10 years) (de Winter-Sorkina et al 2006), and 15.5 ng/kg/day among infants (aged 6 months) (de Winter-Sorkina et al 2003). A 2006 report by the U.K Food Standards Agency (UK Food Standards Agency 2006) found a mean level in toddlers and children (ages 1.5-6) between 11 and 13 ng/kg/day, with a high level between 26 and 34 ng/kg/day. This high value was ultimately chosen as a representative value for the purposes of this risk assessment.

A 2007 report by FSANZ included a dietary exposure assessment, based on the 1995 National Nutrition Survey (NNS) (McLennan et al 1998), that surveyed 13,858 Australians aged 2 years and above using a 24-hour food recall methodology. The authors also measured PBDE levels in food samples obtained as part of the 22nd Australian Total Diet Study (ATDS) (FSANZ, 2008), and used these values to characterise the risk.

PBDE intakes calculated for young children in the FSANZ (2007) report were surprisingly high, compared to a similar risk assessment conducted in the UK (UK Food Standards Agency, 2006) as well as other values reported in the literature and included in Tables 3a and 3b. Detailed analyses presented in the report (based on the data shown here in Table 5) appear to indicate large contributions to PBDE intakes from foods other than animal products, such as breads and pizza, and possibly oils and fats. To our knowledge, this has not been described elsewhere in the scientific literature. Furthermore, the data for the 2007 FSANZ report displays a different age pattern than the other similar risk assessments (UK Food Standards Agency 2006; de Winter-Sorkina et al 2006; Bakker et al 2008), showing dietary PBDE intakes increasing with increasing child age. For these reasons, the PBDE intake values in this report were considered outliers that may not reflect New Zealand exposures and were not used in risk assessment calculations.

A review and exposure assessment of PBDEs by Lorber, concluded that indoor dust exposure constituted 80–90% of total exposures in toddlers over the age of one year (Lorber 2008). Dietary exposures comprised the next most significant exposure source. Other reviews and studies have varied in

this assessment, but, in all cases, dust and diet seem to be the two most prominent routes of exposure to PBDEs (Johnson-Restrepo et al 2009; Trudel et al 2011). Breast milk intakes to young infants were not included in the Lorber review paper. To answer the question about relative contributions of various sources requires an understanding of the differential occurrence of the various PBDE congeners in different environmental media. For example, BDE-47 has been shown to be a prominent contributor to adult and child PBDE intakes due to its occurrence in some foods as well as in breast milk (Mannetje et al 2013). In contrast, BDE-209 is not found in high concentrations in foods or breast milk, but is very prominent in house and car dusts (Harrad et al 2008; Webster et al 2008; Fowles and Morgott, 2013; Coakley et al 2013). A probabilistic exposure model that included house dust and food sources of PBDE exposures in continental Europe, the UK, and North America was developed by Trudel et al (2011). The Trudel model demonstrated the differential contribution of BDE-209 to total PBDE exposures in the UK and Europe compared to North America, where BDE-47 and BDE-99 dominate the sum of PBDE exposures. The relative contribution of BDE-209 to summed PBDE exposures everywhere increased with higher modeled PBDE dose percentiles, especially in the estimated exposures above the 70^{th} percentile (Trudel et al 2011). One recent study reported BDE-209 levels in the environment to be increasing globally (Law et al 2014). Thus, any consideration of the most highly exposed individuals requires careful consideration of the specific hazards and risks of all of the major PBDE congeners, including BDE-209. New Zealand was not included in the international comparative model by Trudel, but there is evidence that New Zealand exposure patterns are better reflected by Australian and UK PBDE congener profiles and levels in the environment than those in North America (Coakley et al 2013; Harrad et al 2008).

Coakley and colleagues (2013) found high levels of BDE-209 in New Zealand house dust. The high levels of BDE-209 in house dust in New Zealand are consistent with findings in Australia, Asia, and parts of Europe. However, the overall PBDE concentrations were higher than has been reported in Australia. These high levels in dust are likely to remain for many years, until older furniture is replaced with furniture lacking added PBDEs.

The ingestion of dust parameter, used in virtually every published risk assessment for PBDEs, has age-specific values ranging from 30 to 100 mg/day, given in the USEPA Exposure Factor Handbook (USEPA 2011). But the reliability of this number is unknown, and there seems to be very little empirical data upon which to estimate with any precision what this value should be. Harrad et al (2008) proposed to use a significantly higher value of 200 mg/day, again not based upon empirical evidence. We have used this range of values in this report, but emphasise that should empirical data become available to quantify a toddler's daily dust intake, the calculations in this report may need revisiting.

Due to these numerous factors, the variability of PBDE levels in house dust is extremely high and difficult to characterise without a large sample size. Stasinska and colleagues report house dust levels for the seven most common PBDE congeners in Australian house dusts to range from 60 to 82,400 ng/g, with a median of 571 ng/g (Stasinska et al 2013). Harrad and colleagues reported an astounding range of BDE-209 house dust concentrations of a median of 2,800 to a maximum of 520,000 ng/g from just 16 households in the UK (Harrad et al 2008). Fowles and Morgott reported that car dust PBDE variation was so great that non-standard statistical distributions were required to describe the variability (Fowles and Morgott 2013). Additionally, the number of variables surrounding dust exposure intake parameters and activity patterns with infants and toddlers increases the variability and uncertainty in characterizing the likely doses, particularly toward the highest exposed individuals.

While PBDEs from polyurethane foam furniture has been thought to be the major source of PBDEs in house dust, electronic goods have recently also been found to significantly contribute to indoor dust PBDE levels through processes that are poorly understood but are under study (Kefeni and Okonkwo 2014).

In New Zealand, no brominated compounds were found in new carpets (MfE, 2010). This may indicate that PBDE exposures in New Zealand are coming from older carpets, as well as older furniture and housing.

One aspect of BDE-209 is the documented degradation to lower brominated PBDEs that are more rapidly bioaccumulative (Lagalante et al 2011). One limitation of many earlier studies is that they did not include BDE-209 in the analysis.

For example, a 2007 Australian report, *Interim Public Health Risk Assessment of Certain PBDE congeners* (NICNAS, 2007) included exposure assessment for BDE-47, 99, 100, 153, and 154, but not BDE-209. Estimated exposures were compared to Lowest Observed Adverse Effect Levels (LOAELs) from animal studies, which were 700 μg/kg/day for BDE-47, 800 μg/kg/day for BDE-99, and 450 μg/kg/day for BDE-153. These values are 2–7 thousand-fold higher than US EPA's RfDs (0.1 μg/kg/day for PBDEs 47 and 99 and 0.2 μg/kg/day for BDE-153) (IRIS 2008a,b,c). The report concluded that Australian infants are not exposed to PBDEs at levels of health concern, based on comparison to experimental LOAEL values.

Another Australian report, from 2007, titled: *Polybrominated Diphenyl Ethers (PBDE) in Food in Australia*, did include BDE-209, but concentrations of the 26 congeners evaluated were not weighted to take account of differences in their relative toxicity (FSANZ 2007). Despite the high intake estimates, the

report concluded that dietary exposure of Australian children and adults to PBDEs in food is low, with the margin of exposures (MOEs) for the majority of population groups at or above 1,000 and therefore not of concern. The authors discounted the one relatively low MOE of 600 calculated for 2–5 year old males due to the overly conservative nature of the estimate, particularly as the majority of samples contained no detectable PBDEs. For MOE calculations, the 2007 FSANZ report used a threshold dose of 100 μg/kg/day for calculating margins of exposure (FSANZ 2007). This level was set by Joint FAO/WHO Expert Committee on Food Additives (JECFA, 2006) and is a thousand-fold lower than the US EPA's RfD for lower brominated PBDEs (IRIS 2008a,b).

Although our conclusions are somewhat consistent with those in the 2007 NICNAS and FSANZ reports (FSANZ 2007; NICNAS 2007), we did find some scenarios under which a significant risk could exist, and others that approach the hazard index threshold of 1.0. It is important to note that advances in scientific understanding of PBDEs since 2007, specifically about the neurodevelopmental effects of PBDEs on young children, have led to US EPA's release of reference doses for PBDE congeners (IRIS 2008a,b,c,d) which are orders of magnitude lower than the threshold dose used in the earlier Australian reports which were set by JECFA in 2005 or the LOAELs from animal studies. This is similar to what has been observed as scientific understanding grows in relation to impacts of other environmental chemicals on neurodevelopment, such as in the case of lead (CDC 2012; New York Times 2012), and indicates the importance of revisiting risk assessments as new scientific information on the toxicity of environmental chemicals is produced. In our exposure assessment, Hazard Indices do approach 1 and even exceed 1 in certain high exposure scenarios. Even a small downward revision of the Reference Dose by US EPA or other agencies could have a large impact on our conclusions.

In addition to PBDEs, numerous brominated and chlorinated flame retardants are now found in indoor dust throughout the world (Fromme et al 2014). The toxicological significance and environmental movement of these compounds into the human body are under study, with significant data gaps remaining.

5. CONCLUSION

This report compiles child exposure, hazard, and risk assessment estimates for PBDE congeners from dietary, house dust, and car dust sources found in New Zealand. While typically expected exposure patterns and risks to children from PBDE exposures were not found to exceed levels of concern in our assessment, scenarios were constructed, particularly in relation to households with high PBDE dust content, that conceivably present significant health concerns for infants. The significance of this finding is underscored by the observation in the scientific literature that conventional risk assessment methodologies have been observed to under-predict the observed PBDE biomonitoring (body burden) data internationally, by several fold (Wong et al 2013). Thus, our understanding of exposure pathways or toxicodynamics for PBDEs is imperfect, and it may be that bioavailability, exposure to dusts, or partitioning of PBDEs from particulates into the vapour phase is not completely understood. Given our study findings, the following conclusions can be reached.

- New Zealand infants and toddlers have PBDE exposures of the same magnitude as many other countries around the world.

- Breast milk, diet, house dust, and interior car dust represent the most significant known sources of PBDE exposure to infants and toddlers. The possibility exists that some toys may be significant contributors to exposure, but there is insufficient reliable information to quantitatively assess this currently.

- PBDE exposures vary greatly depending on the specific conditions within each household and vehicle, and with the amount of dust ingested.

- Typical exposure scenarios do not result in risks above a level of concern for infants and toddlers, although they approach such levels in certain scenarios.

- These scenarios include the highest measured PBDE dust and breast milk concentrations and intakes, where risks can become significant for breast feeding infants, with Hazard Indices greater than 1.0 for the combined exposure to all PBDE congeners.

- These conclusions are based on current knowledge of hazard potencies of PBDEs. As more toxicology and epidemiology research clarifies thresholds for onset of toxic effects, these conclusions may

change. Given the amount of on-going research in this area, it is recommended to maintain continued awareness of the scientific literature in this area.

The University of Birmingham in the UK has initiated a project called INFLAME, which aims to elucidate and better define the environmental fate parameters and behaviour of flame retardants in the indoor environment that lead to human exposure and to better understand the potential for health effects that these exposures represent (http://www.birmingham.ac.uk/research/activity/inflame/index.aspx).

Boston University has a similar active research program in their School of Public Health funded by the National Institute of Environmental Health Sciences in the USA (http://www.bu.edu/sph/research/research-landing-page/exposure-biology-research-group-ebrg/projects/assessing-pbde-exposure-pathways). Continued research in this area will undoubtedly shed further light on these areas of uncertainty, and a watching brief with regular updates to assumptions and intake parameters is suggested.

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APPENDIX A. BREAKDOWN OF CALCULATED HAZARD INDICES BY SCENARIO AND ROUTE OF EXPOSURE

Summary of Exposure Scenarios with Estimated Intakes and Hazard Indices

		Mean (ng/kg BW/day)			**Maximum** (ng/kg BW/day)		
		tri-penta	hexa-nona	deca	tri-penta	hexa-nona	deca
Scenario 1	**Infant 0-6 months old**						
	Breastmilk	17.83	5.13	1.55	54.45	25.09	13.76
	House Dust (typical)	0.68	1.06	17.38	2.60	10.02	190.07
	House Dust (high)	2.25	3.54	57.94	8.67	33.39	633.58
	Car Dust (typical)	0.99	1.59	19.81	7.08	12.02	167.00
	Car Dust (high)	3.31	5.30	66.03	23.59	40.08	556.67
	Total (mean dust)	19.50	7.78	38.74	64.12	47.13	370.84
	Total (high dust)	23.39	13.97	125.52	86.71	98.56	1204.01
	Total HI (mean dust)	*0.19*	*0.04*	*0.01*	*0.64*	*0.24*	*0.05*
	Total HI (high dust)	*0.23*	*0.07*	*0.02*	*0.87*	*0.49*	*0.17*
Scenario 2	**Infant 6-12 months old**						
	Breastmilk	8.91	2.57	0.78	27.22	12.54	6.88
	Solid foods	1.48	0.20	5	2.84	0.44	15
	House Dust (typical)	0.50	0.79	12.86	1.92	7.41	140.64
	House dust (high)	1.67	2.62	42.87	6.41	24.70	468.80
	Car Dust (typical)	0.34	0.54	6.72	2.40	4.08	56.64
	Car Dust (high)	1.12	1.80	22.39	8.00	13.59	188.78
	Total (mean dust)	11.23	4.09	25.35	34.39	24.47	219.16
	Total (high dust)	13.18	7.18	71.04	44.48	51.28	679.47
	Total HI (mean dust)	*0.11*	*0.02*	*0.00*	*0.34*	*0.12*	*0.03*
	Total HI (high dust)	*0.13*	*0.04*	*0.01*	*0.44*	*0.26*	*0.10*
Scenario 3	**Toddler 1-4 years old**						
	Solid foods	2.96	0.39	10	5.68	0.87	30
	House Dust (typical)	0.55	0.87	14.27	2.13	8.22	156.00
	House Dust (high)	1.11	1.74	28.53	4.27	16.44	311.99
	Car Dust (typical)	0.39	0.62	7.77	2.78	4.72	65.55
	Car Dust (high)	0.78	1.25	15.55	5.56	9.44	131.10
	Total (mean dust)	3.90	1.88	32.04	10.59	13.81	251.55
	Total (high dust)	4.46	2.76	46.31	15.50	26.75	473.09
	Total HI (mean dust)	*0.04*	*0.01*	*0.00*	*0.11*	*0.07*	*0.04*
	Total HI (high dust)	*0.04*	*0.01*	*0.01*	*0.16*	*0.13*	*0.07*

[Intentionally Blank]

APPENDIX B. REVIEW OF BIOMONITORING LITERATURE

A comprehensive review of international biomonitoring data is beyond the scope of this report. However, a brief review of the most relevant data is presented here.

Young children have been shown in biomonitoring studies to have the highest body burdens of PBDEs, with blood levels peaking at 2.6–3 years of age (Toms et al 2009b). In their study, Toms and colleagues reported that exposures independent of breastfeeding must account for these higher body burdens. This observation is consistent with intense hand to mouth activity of toddlers with house dust and articles that contain PBDEs. It should be noted that this study did not include BDE-209, which would almost certainly have added even more strength to this conclusion.

Sjodin and colleagues determined that a 2.5 year old toddler would be expected to be exposed to far more BDE-209 than any other PBDE congener in Australia, the UK, or the USA. Exposures in Germany were, in sharp contrast, much lower for seven PBDE congeners, including BDE-209 (Sjodin et al 2008). The precise reason for the vast discrepancy in house dust PBDE concentrations between European countries is not known. A correlative study from Wisconsin, of 44 adults (mean age 58 years) found associations between dust PBDEs and blood serum levels, except for BDE-209 (Imm et al 2009). Thus, while BDE-209 is an important congener in terms of infant and toddler exposures to house and car dust PBDEs, this does not reflect the situation for older adults, where dietary exposures may predominate.

In New Zealand, biomonitoring levels of PBDEs are available from 23 blood serum donors collected in 2001 in Wellington (Harrad and Porter, 2007). The sum of human blood levels for congeners BDE-47, 99, 100, 153, 154 and 183 in serum was 7.17 ng/g lipid weight. Table A1 shows serum values for individual congeners and excludes BDE-183 for ease of comparison with other studies. The sum for five congeners was 6.9 ng/g lipid weight, specifically BDE-47=3.8 ng/g, BDE-99=0.9 ng/g, BDE-100=0.9 ng/g, BDE-153=1.2 ng/g, BDE-154=0.1 ng/g. BDE-209 was not included in the study. One donor displayed concentrations that were significantly elevated, greater than two standard deviations above others in the study (Harrad and Porter, 2007). These levels lower than but comparable to values from Australia (also shown in Table A1). Serum values from Australia are based on 2420 blood serum samples collected from South East Queensland in 2006–7. The samples were stratified by age and sex, and pooled for analysis of PBDEs (Toms et al 2009b). The PBDE serum levels observed in New Zealand are similar to levels reported in Europe (Gomara et al 2006; Roosens et al 2010;

Guo et al 2010), and lower than those observed in North America (Sjödin et al 2008; Lunder et al 2010; Johnson et al 2010; Lee et al 2013).

Harrad and Porter suggest that the most likely sources of PBDE exposures in New Zealand are imported foods and imported consumer goods such as furnishings and electronics (Harrad and Porter 2007). The lower levels of PDBEs observed in New Zealand blood and dust samples compared to the U.S. and North America are not unexpected, since New Zealand has never required PDBEs for fire protection, as was done in the U.S., nor have PBDEs ever been produced, used or imported as a raw product in New Zealand (Harrad et al 2006; Mannetje et al 2013; Coakley et al 2013).

Biomonitoring data for breast milk is also available for New Zealand. Mannetje et al. measured PBDEs in breast milk samples collected in 2007–10 from 39 donors from New Zealand. The four study sites included Wellington (urban North Island), Wairarapa (rural North Island), Christchurch (urban South Island) and North Canterbury (rural South Island). As shown in Table A1, the sum of breast milk concentrations for congeners BDE-47, 99, 100, 153, 154 and 209 in blood serum was 4.7 ng/g lipid weight in Zealand, about half the value observed in Australia (10.0 ng/g, excluding BDE-209), with a similar congener profile. Breast milk data from Australia is based on 157 pooled samples collected throughout Australia in 2002–03 and is also shown in Table A1. Values in Australia were similar and somewhat higher than values observed in Europe (Fängström et al 2008; Colles et al 2008; Thomsen et al 2010; Roosens et al 2010) and 3–6 times lower than values observed in the U.S. (Schecter et al 2003; Bocio et al 2004; Schecter et al 2006a, Darnerud et al 2001; Daniels et al 2010).

BDE-209 levels presented for New Zealand breast milk are likely to represent recent exposure, but may be underestimates of the true body burden of BDE-209 exposure because of the less efficient transfer of highly brominated congeners from blood to breast milk (Mannetje et al 2013). Higher concentrations of BDE-209 were reported in New Zealand for urban vs. rural regions, suggesting that urban environments may contain more sources of higher brominated PBDEs, which would be consistent with their use in items such as electronics (Kefeni and Okonkwo, 2014).

Table A1 Comparison of PDBE levels measured in serum and breast milk samples in Australia and New Zealand. Concentrations in ng/g lipid weight.

	Congener							
Sample	BDE47	BDE99	BDE100	BDE153	BDE154	BDE209	ΣBDE	Reference
Australia								
0–0.5 year	7	3.8	1.5	1.8	-	-	14	Toms 2009b
0.5–1 year	19	7.5	3.8	2.8	-	-	33	Toms 2009b
1–1.5 years	21	7.9	5.1	3.4	-	-	37	Toms 2009b
1.5–2 years	21	7.8	1.96	4.5	-	-	39	Toms 2009b
2.1–2.5 years	20	7.1	5.2	4.3	-	-	36	Toms 2009b
2.5–3 years	26	12	7.1	5.9	-	-	51	Toms 2009b
3.5–4 years	15	5.2	4.8	5.6	-	-	31	Toms 2009b
>16 years	8.4	2.5	2.0	3.3	-	-	16	Toms 2009b
Breast milk	5.6	1.9	1.3	1.1	0.1	-	10	Harden 2005
New Zealand								
Adult serum	3.8	0.9	0.9	1.2	0.1	-	7	Harrad & Porter 2007
Breast Milk	2.5	0.5	0.5	0.7	0.0	0.4	5	Mannetje et al. 2013

Table A2 provides a brief summary of international PBDE biomonitoring data that has been reported to date. This comparison of PBDE concentrations in human serum from different countries was taken directly from a recent report by Gari and Grimalt (Garí et al 2013).

Table A2 Median PBDE concentrations (in ng/g lipid) found in human serum from different countries. From Gari and Grimalt (2013).

Location	Year	n	BDE-28	BDE-47	BDE-85	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	BDE-209	ΣPBDEs[a]	Reference
Catalonia	2002	731	0.71	2.6	0.81	1.2	0.83	0.94	0.44	0.21	3.7	15.4 (8.0)	Present study
Norway	1998	69[b]	0.14	2.2	NA	0.45	0.45	0.54	0.26	ND	NA	4.0	Thomsen et al. (2002)[c]
Norway	1999	29[b]	0.24	1.5	NA	0.31	0.35	0.59	0.35	ND	NA	3.3	Thomsen et al. (2002)
Sweden	2000	15	0.070	0.83	<0.010	0.19	0.17	0.56	0.040	0.060	NA	2.1	Guvenius et al. (2003)
Sweden	2000	50	NA	0.91	NA	0.20	0.29	1.1	0.33	NA	0.46	(3.6)	Weiss et al. (2006)
UK	2003	154	<0.14	0.82	<0.13	<0.16	0.76	1.7	0.60	0.30	<15	5.6 (4.7[d])	Thomas et al. (2006)
Belgium	1999	11	0.10	1.2	ND	0.40	0.27	1.6	ND	0.21	11.1	3.8[e]	Covaci and Voorspoels (2005)
Belgium	2004	8[b]	NA	0.97	NA	0.080	0.20	1.4	1.3	0.32	NA	4.6	Roosens et al. (2010)
France	2004	91	0.12	2.8	0.060	1.9	0.37	0.72	0.065	0.21	5.8	(0.98[e])	Antignac et al. (2009)
Greece	2007	61	0.010	0.16	NA	0.090	0.11	0.51	0.020	0.030	1.2	(1.1[d])	Kalantzi et al. (2011)
Faroe Islands	1994	57	NA	1.3	NA	0.33	0.51	1.0	1.4[f]	NA	0.77	4.0	Fängström et al. (2005)
Spain	2003	217	0.060	2.5	0.16	2.4	1.4	0.83	0.10	0.30	1.1	11.4 (8.5)	Gómara et al. (2007)[c]
Spain	2003	174	ND	2.3	ND	0.35	ND	2.1	1.5	ND	<0.70	9.6	Vizcaino et al. (2011)
New Zealand	2001	23	NA	3.2	NA	0.80	0.62	1.0	0.080	0.23	NA	(6.1)	Harrad and Porter (2007)
Australia	2006	84[b]	–	8.4[g]	–	2.5[g]	2.0[g]	3.3[g]	–	–	–	16[g]	Toms et al. (2009)
Korea	2001	62	0.30[f]	5.2	0.15	2.6	1.1	2.7	0.27	1.9	NA	16	Lee et al. (2007)
China (north)	2006	115	0.040	0.83	ND	0.67	0.13	0.32	0.040	0.41	ND	7.1 (2.9)	Zhu et al. (2009)
China	2007	12[b]	29[g]	21[g]	NA	23[g]	15[g]	33[g]	42[g]	47[g]	403[g]	613[g]	Jin et al. (2009)
China (south)	–	21	0.39	1.0	NA	0.36	0.15	1.4	0.10	0.31	NQ	(4.4)	Bi et al. (2006)
Japan	–	156	0.14	0.74	0.071	0.17	0.22	0.59	0.11	0.16	6.9	9.5	Takasuga et al. (2004)
Japan	2007	72	0.080	0.32	0.010	0.040	0.14	0.66	0.050	0.015	0.90	3.6	Uemura et al. (2010)
Canada	2003	110	NA	8.1[h]	NA	1.4[h]	1.1[h]	1.35[h]	NA	NA	NA	(13.4[h])	Sandanger et al. (2007)
US	1999	416	0.40	15	ND	4.0	2.5	2.2	ND	ND	NA	27	Castorina et al. (2011)
US	2000	7[b]	NA	34	0.70	11	5.9	7.3	0.95	NA	NA	61	Sjödin et al. (2004)
US	2001	168	0.013	0.11	0.013	0.032	0.013	0.025	NA	NA	NA	0.22	Turyk et al. (2010)
US	2003	2040	1.1	19	ND	5.0[h]	3.6	4.8	ND	ND	NA	33.5[i]	Sjödin et al. (2008)
US	2006	20	0.44	8.8	ND	1.4	1.2	5.8	1.8[f]	ND	1.4	17	Lunder et al. (2010)
US	2007	24	1.3	17	<ND	2.4	3.0	7.0	ND	ND	ND	39	Johnson et al. (2010)

[a] Sum of all congeners analysed in each study. Between brackets, sum of tri-hepta BDE congeners ($\sum PBDEs_{3\_7}$).
[b] Pooled samples.
[c] Medians of the whole population were not available. They have been calculated from weighting of the medians of the different groups reported.
[d] Sum of BDEs 47, 99, 100, 153 and 154.
[e] Sum of all congeners except BDE-209.
[f] The reported value coeluted with another compound.
[g] Mean instead of median.
[h] Geometric mean instead of median.
[i] Sum of all geometric means of the congeners reported in the study. NA: Not analysed; ND: Not detected.