# Aspect human exposure to emerging and legacy flame retardants in the UK and Vietnam

by

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# Abstract

Analytical methods based on gas chromatography in combination with electron capture negative ion/electron ionisation mass spectrometry were developed and validated for the separation and determination of legacy and emerging flame retardants (EFRs) in a wide range of samples including indoor air, dust, diet and human milk. A broad suite of EFRs and legacy flame retardants (FRs) including polybrominated diphenyl ethers (PBDEs) and hexabromocyclododecane (HBCDDs) were determined in indoor air and dust taken from offices and homes in Birmingham, UK. Comparison with previous data, suggests that temporal trends in contamination with EFRs and legacy FRs reflect changes in production and use of such compounds as a result of bans and restrictions on the use of legacy FRs. Specifically, concentrations of some EFRs are increasing, while those of PBDEs and HBCDDs are decreasing in both indoor air and dust. In contrast, there are indications that concentrations of ∑PBDEs are increasing in UK diet samples, indicating a gradual shift over time of PBDEs from the indoor to the outdoor environment and thus our food supply. Using *inter alia* a simple, single compartment, steady state pharmacokinetic model, human exposure to FRs via air inhalation, dust ingestion and diet was estimated for different age groups (adults, toddlers and nursing infants) and the relative importance of each exposure route to overall exposure assessed under different exposure scenarios. Dust ingestion was the main exposure pathway for toddlers to all target FRs except DBE-DBCH (for which dietary exposure was the major exposure pathway), EH-TBB and BTBPE (for which dust and diet are broadly equally important). By comparison, for adults, dust ingestion appears to constitute the major exposure pathway to BDE-209, BTBPE, BEH-TEBP, and DBDPE, with dietary exposure the predominant pathway to HBCDDs, tri-hexa BDEs, DBE-DBCH and EH-TBB. Concentrations of target EFRs were detected for the first time in UK human milk samples ($n=35$). No significant differences were found between the levels of individual EFRs in human milk group 1 (collected in 2010) and group 2 (collected from 2014-2015) ($p > 0.05$). No obvious time trends were found between the two human milk groups for ΣPBDEs and HBCDDs. The most abundant EFR was found to be β-DBE-DBCH with a mean concentration of 2.5 ng/g lw which is comparable to the levels of the most abundant legacy FRs i.e. BDE 47, 153 and α-HBCDD (2.8, 1.7 and 2.1 ng/g lw, respectively). Human exposure to EFRs and HBCDDs via diet was estimated for a population impacted by a rudimentary e-waste processing area in Vietnam. Concentrations of all target FRs in e-waste-impacted samples exceed significantly ($p < 0.05$) those detected in the controls, suggesting e-waste processing activities exert a substantial impact on local environmental contamination and human dietary exposure. The estimated dietary intakes of EFRs in this study were 170 and 420 ng/kg bw/day, for adults and children, respectively; while daily ingestion of HBCDDs were an estimated 480 and 1500 ng/kg bw/day for adults and children, respectively.

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# ABBREVIATIONS

APPI Atmospheric pressure photoionisation

BAF Bioaccumulation factor

BEH-TEBP Bis(2-ethylhexyl) tetrabromophthalate

BFR Brominated flame retardant

BTBPE 1,2-bis (2,4,6-tribromophenoxy) ethane

Bw Body weight

DBDPE Decabromo diphenyl ethane

DBE-DBCH Tetrabromoethylcyclohexane

DDC-CO Dechlorane plus

Dw Dry weight

ECNI Electron capture negative ionisation

EH-TBB 2-ethylhexyl-2,3,4,5-tetrabromobenzoate

EF Enantomeric fraction

EFR Emerging flame retardant

EI Electron impact

GC Gas chromatography

HBB Hexabromobenzene

HBCDD Hexabromocyclododecane

HCDBCO Hexachlorocyclopentadienyldibromocyclooctane

HPLC High pressure liquid chromatography

IS Internal standard

Kow n-octanol/water partition coefficient

LOD Limit of detection.

LOQ Limit of quantification.

Lw Lipid weight

m/z Mass to charge ratio.

MS Mass spectrometry

PBBz pentabromobenzene

PBDE Polybrominated diphenyl ether

PBEB Pentabromoethylbenzene

PBT Pentabromotoluene

PK Pharmacokinetic.

POP Persistent organic pollutant

PUF Polyurethane foam

QA/QC Quality assurance/quality control

RDS Recovery determination standard

RSD Relative standard deviation

SD Standard deviation

S/N Signal to noise ratio

TBCT Tetrabromo-o-chlorotoluene

TBBPA-BDBPE Tetrabromobisphenol A-bis(2,3-dibromopropyl ether)

TBP-DBPE 2,3-dibromopropyl 2,4,6-tribromophenyl ether

TBX 1,2,4,5-Tetrabromo-3,6-dimethylbenzene

UNEP United Nations Environment Programme

USEPA United States Environmental Protection Agency

WHO World Health Organization

Ww Wet weight

# CHAPTER 1 Introduction

Halogenated flame retardants (HFRs) have found utility as flame retardants (FRs) incorporated within a wide range of consumer goods and materials. This is because halogens are very effective in capturing free radicals, and thus in preventing flame propagation (Alaee et al., 2003). Even though all four halogens are effective in eliminating free radicals, fluorinated compounds are very stable and decompose at much higher temperatures than most organic matter burns, and iodinated compounds are not stable and decompose at slightly elevated temperatures (Alaee et al., 2003). Therefore, organochlorine and organobromine compounds are favoured as flame retardants because of their stability during the lifetime of the product and their appropriate decomposing temperature (Alaee et al., 2003). Brominated flame retardants (BFRs) are one of the most-widely used groups of organic flame retardants because of their relatively low cost and high performance efficiency (Abdallah, 2009). As a result, there are more than 75 different organobromine compounds used as flame retardants to make sure that a variety of manufactured goods such as plastics, textiles, building materials, vehicles and electronic equipments meet fire safety regulations (Alaee et al., 2003; Ali et al., 2011a; Reistad et al., 2005). The most commonly used and studied "legacy" BFRs are PBDEs, HBCDDs and tetrabromobisphenol-A (TBBP-A). The Penta-BDE and Octa-BDE commercial mixtures were added to the Persistent Organic Pollutants list of the Stockholm Convention (2009) for global bans and the production and use of both commercial mixtures were phased out in Europe and North America in 2004 (La Guardia et al., 2006). The use of Deca-BDE in the EU has been banned in electrical and electronic applications since 1 July 2008 (European Court of Justice, 2008). HBCDDs were listed in the Stockholm Convention recently (UNEP, 2014). Following these bans and restrictions imposed on PBDEs and HBCDDs globally, there is potential for emerging flame retardants (EFRs) to replace these formulations. The estimated total production volume for EFRs is around 180,000 tons/year on the assumption that the production volumes have not changed much over the past few years

(Harju, et al., 2009). However, very limited information currently exists about the production volumes, physicochemical properties, analytical methods, environmental fate and behaviour, and toxicity of EFRs. In the absence of detailed evidence, concern exists that given their similar structures to “legacy” BFRs, they may show persistent, bioaccumulative, and toxic (PBT) characteristics and pose health risks to human and wildlife, especially for those produced in high volumes. Likewise, the similar use patterns and structures, means that the typical pathways of human exposure to EFRs are likely similar to those of the legacy BFRs, via a combination of diet, ingestion of dust, dermal uptake and inhalation (Harrad et al., 2010).

## 1.1 Environmental degradation, metabolism, and physicochemical properties

The physicochemical properties of some important EFRs, PBDEs and HBCDDs are shown in Table 1.1. Currently, although very limited information is available for physicochemical properties of EFRs, their structural similarity to “legacy” BFRs, suggests such properties will also be similar.

### 1.1.1 DBDPE

Because of its similar structure, the physicochemical properties of DBDPE are assumed to be similar to those of BDE 209, including: low vapour pressure, low water solubility, and high $K_{OW}$ (Hardy et al., 2002). Similar to BDE 209, DBDPE undergoes degradation to lower brominated congeners during sample preparation/analysis, although it appears to be less sensitive to thermal degradation than BDE 209 (Kierkegaard et al., 2009). Likewise, the inclusion of the ethane bridge between the aromatic rings makes it slightly more hydrophobic than BDE 209 (Covaci et al., 2011). DBDPE has been reported to have a high bioaccumulation factor (BAF), with log BAF values between 6.1 and 7.1 in fish that are one order of magnitude higher than those of BDE 209 (He et al., 2012). In contrast, DBDPE was found to bioaccumulate in rats at concentrations 3-5 orders of magnitude lower than BDE 209 after oral exposure to 100 mg/kg body weight(bw)/day of DBDPE or BDE 209 for 90 days (Wang et al., 2010). Preferential accumulation of BDE 209 in the liver rather than other tissues (kidney, adipose) has also been observed, however, adipose displayed the greatest accumulation of DBDPE (Wang et al., 2010). Biomagnification factor (BMF) values of 0.2-9.2 were found for DBDPE between trophic levels of the Lake Winnipeg (Canada) food web (Law et al., 2006b). At least seven unknown compounds were observed in DBDPE-exposed rats, indicating that DBDPE biotransformation occurred in rats (Wang et al., 2010). Two degradation products (nona-brominated congeners) were found to be present in the technical product (Saytex®8010) (Kierkegaard et al., 2004). When DBDPE dissolved in *n*-hexane was exposed to a daylight fluorescent lamp, DBDPE was degraded to the two nona-brominated congeners as well as a number of peaks tentatively identified as octa-brominated products (Kierkegaard et al., 2009). On the other hand, when high impact polystrene (HIPS) powder was spiked with BFRs and exposed to sunlight for 112 days, no degradation of DBDPE was found, while the photolytic half-life of BDE 209 was estimated at 31 days (Kajiwara et al., 2007).

### 1.1.2 BTBPE

For BTBPE, the derived BAF in juvenile rainbow trout was 2.3 ± 0.9 with a calculated half-life of 54.1 ± 8.5 days, suggesting that this chemical has a high potential for biomagnification in aquatic food webs (Tomy et al., 2007a). No metabolites were detected and results indicated that BTBPE is not a potent thyroid axis disruptor (Tomy et al., 2007a). Biomagnification between trophic levels of the Lake Winnipeg (Canada) food web resulted in a BMF of 0.1-2.5 for BTBPE (Law et al., 2006). However, BTBPE displayed poor gastrointestinal absorption in two oral ingestion studies of BTBPE in rats (Nomeir et al., 1993; Verreault et al., 2007).

Metabolites were excreted in the urine, bile and faeces, but at a very low level. Fecal metabolites were characterised as monohydroxylated, monohydroxylated with debromination, dihydroxylated/debrominated on a single aromatic ring, monohydroxylated on each aromatic ring with accompanying debromination, and cleavage on either side of the ether linkage to yield tribromophenol and tribromophenoxyethanol in rats (Verreault et al., 2007). In Hakk et al.'s study (2004), male Sprague-Dawley rats were orally exposed to a single oral dose of BTBPE (2.0 mg/kg bw) in conventional and bile-duct cannulated experiments and the great majority of BTBPE (>94%) was excreted in the faeces of both groups of rats at 72 h while a limited quantity of stable metabolites was excreted.

### 1.1.3 EH-TBB and BEH-TEBP

In laboratory photodegradation experiments, photodegradation of EH-TBB and BEH-TEBP via debromination reactions was reported to occur more slowly than photodegradation of decaBDE or nonaBDEs (Davis and Stapleton, 2009). In one *in vitro* study, EH-TBB was consistently metabolised to 2,3,4,5-tetrabromobenzoic acid (TBBA) via cleavage of the 2-ethylhexyl chain without requiring any added cofactors in experiments with liver and intestinal subcellular fractions and in purified porcine carboxylesterase, while BEH-TEBP was only metabolised to mono(2-ethylhexyl) tetrabromophthalate (TBMEHP) in purified porcine carboxylesterase (Roberts et al., 2012). Bearr et al. (2012) studied *in vitro* biotransformation of EH-TBB and BEH-TEBP in hepatic subcellular fractions (i.e. S9, microsomes and cytosol) of the fat-head minnow, common carp, mouse and snapping turtle. Metabolic loss of BEH-TEBP was measured for all species, with EH-TBB loss observed for all species except snapping turtle. The same study reported that EH-TBB was consistently metabolised to a greater extent than BEH-TEBP across all species tested (Bearr et al., 2012).

### 1.1.4 TBBPA-BDBPE

TBBPA-BDBPE exhibits the same overall persistence ($P_{OV}$) and long-range transport potential (LRTP) values as the POP-PBDEs - i.e. those listed as persistent organic pollutants in the Stockholm Convention in May 2009 (Kuramochi et al., 2014). Studies of the metabolism of this substance in rats (Fischer 344) showed TBBPA-BDBPE is poorly absorbed from the gastrointestinal tract and not metabolically transformed. On the other hand, elimination from the body took a relatively long time (13.9 h) and most of the retained compound accumulated in the liver (Knudsen et al., 2007). TBBPA-BDBPE is susceptible to hydrolysis at the same level as DDT with an experimental half-life of < 0.02 h at 273 K (MeOH/DMF, 5/95 ratio) with sodium methoxide as a strong nucleophile. The elimination product TBBPA bis-(bromopropenyl ether) might be more prevalent in sediment in a similar manner as DDE is for DDT (Rahm et al., 2005). If present in the atmosphere, TBBPA-BDBPE will likely be bound to particles because of its low volatility. Consequently, its atmospheric transport behaviour will be determined by particle transport (Harju et al., 2009).

### 1.1.5 DBE-DBCH

In Muir and Howard's (2006) review of screening and categorisation studies of chemicals in commerce, with high predicted bioconcentration potential, low biodegradation rates and long range atmospheric transport potential, DBE-DBCH was one of the 30 chemicals included on their list. With a $\log K_{OW}$ value of 5.25 (i.e. within the "ideal" >3−6 range), and a reported bioconcentration factor (BCF) of 2,153, the DBE-DBCH isomers have been identified as having high persistence and environmental bioaccumulation potential (Howard and Muir, 2010). DBE-DBCH was found to be bioavailable to *Eisenia fetida* and accumulated in earthworms (Nyholm et al., 2010a). Following its application to soil, degradation of DBE-DBCH was evident over 360 days; moreover, the soil-air partition coefficient of DBE-DBCH increased over time before levelling off, suggesting that the volatility of DBE-DBCH decreases with time as it becomes more strongly bound to soil (Wong et al., 2012). The α- and β-DBE-DBCH isomers undergo thermal rearrangement to γ- and δ-DBE-DBCH at temperatures above 120-125 °C, which could be expected to occur at the high temperatures used in manufacturing processes (Riddell et al., 2009). In *in vitro* enzyme-mediated biotransformation assays based on rat liver microsomes, concentrations of both α- and β-DBE-DBCH isomers were equally depleted by about 40% and metabolism via hydroxylation (but not debromination) occurred *in vitro* for α- and β-DBE-DBCH (Chu et al., 2012).

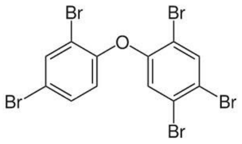
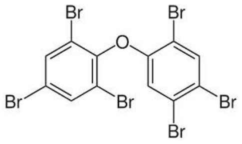
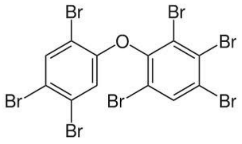
### 1.1.6 DDC-CO

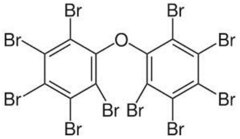
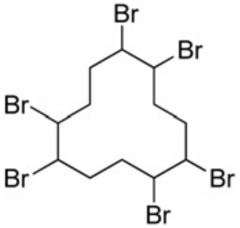
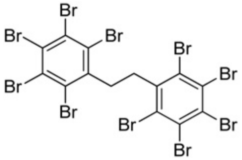
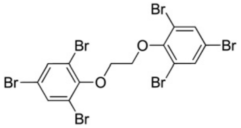
DDC-CO is an odourless, white, free-flowing powder that is thermally stable up to 285 °C; its vapour pressure is moderate (0.8 Pa at 200 °C) and $K_{OW}$ is very high (Log $K_{OW}$ = 9.3), with the latter making it extremely lipophilic (Xian et al., 2011). DDC-CO has been identified by the European Commission as a possible replacement for DBDPE (Chen et al., 2014). The DDC-CO technical product consists of the *syn*- and *anti*- isomers in a ratio of about 1:3 (Sverko et al., 2011). Given its physicochemical properties (high lipophilicity, chemical stability (exemplified by its long photodegradation> 24 years)), bioaccumulation in fish and lack of biodegradation, DDC-CO has characteristics typical of persistent organic pollutants (de la Torre et al., 2011). DDC-CO has potential to be bioaccumulated and biomagnified in fish regardless of its high molecular weight and very high log $K_{OW}$ value and to be susceptible to long-range atmospheric transport (Feo et al., 2012). DDC-CO was reported to be bioavailable to the oligochaete, *L. variegatus*, from sediment (*syn*-DDC-CO (biota-sediment accumulation factors (BSAFs) = 0.34-0.48 g OC/g lipid) > *anti*-DDC-CO (BSAFs= 0.21-0.39 g OC/g lipid)) (Li et al., 2014), and to bioaccumulate in a marine food web (with BMFs of 0.14 and 0.11 for *syn*-DDC-CO and *anti*-DDC-CO, respectively in black-headed gulls) (Peng et al., 2014). In Li et al.'s study (2013), accumulation of both *syn*-DDC-CO and *anti*-DDC-CO was reported in Sprague-Dawley rats. Specifically, DDC-CO was preferentially accumulated in the liver rather than in muscle, no significant stereoselectivity of *anti*-DDC-CO or *syn*-DDC-CO in tissues was observed in the low DDC-CO exposure groups, while the ratio of *syn*-DDC-CO to *anti*-DDC-CO increased significantly in all tissues in the high DP exposure groups. Furthermore, Tomy et al. (2008a) also found the BMF value (determined in whole fish minus liver) of the *syn*-isomer (5.2) in juvenile rainbow trout exceeded that of the *anti*-isomer (1.9), suggesting that the *syn*- isomer is more bioavailable. However, a different pattern of bioaccumulation was found in the Lake Winnipeg food web; with *anti*-DDC-CO dominant in higher trophic level (TL) organisms like walleye and goldeye, while *syn*-DDC-CO dominated the lower TL organisms like zooplankton and mussels (Tomy et al., 2007b).

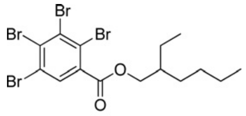
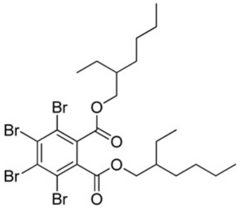
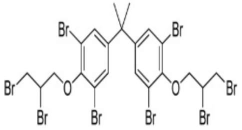
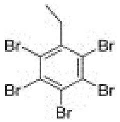
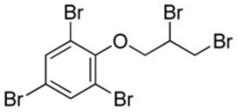
### 1.1.7 Other EFRs

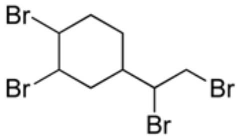
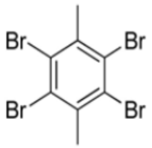
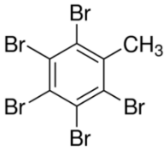
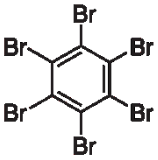
TBX, TBCT, PBT, and HBB were identified as POP-like chemicals on the basis of their $P_{OV}$ and LRTP values (Kuramochi et al., 2014). In another modelling study, PBT, HBB, and TBX were highlighted as capable of travelling extremely long distances (>8000 km) and were thereby classified as POP candidates based on $P_{OV}$, LRTP, and bioaccumulation criteria (Liagkouridis et al., 2015). Another study identified PBEB as potentially environmentally persistent (Gouteux et al., 2008). Log BAF values ranged from 2.04–4.77, 2.72–4.09 and 3.31–5.54 for PBT, PBEB, and HBB, respectively in various aquatic species, demonstrating their highly bioaccumulative properties (Wu et al., 2011). In Wu et al.'s study (2010), HBB was reported to be biomagnified in the food web, whereas PBT was found to be diluted. HBB was also found to be bioavailable to and accumulate in *Eisenia fetida* (Nyholm et al., 2010a). However, BMF values of HBB for the earthworm *Eisenia andrei* were <0.17 which suggested minimal biomagnification from soil (Belfroid et al., 1995). A study of the transformation of HBB in soil, showed it degraded more rapidly (with a half-life < 40 days) in aerobic soil than under anoxic conditions (in which a half-life >100 days was observed) (Nyholm et al., 2010b). As reported above for DBE-DBCH, following its application to soil, degradation of TBP-DBPE was evident over 360 days; moreover, the soil-air partition coefficient of TBP-DBPE increased over time before levelling off, suggesting that the volatility of TBP-DBPE decreases with time as it becomes more strongly bound to soil (Wong et al., 2012). PBEB is included in the OSPAR list of chemicals, being ranked as persistent, liable to bioaccumulate and toxic (OSPAR, 2001) and classified as a low production volume (LPV) chemical in the EU (ESIS, 2010).

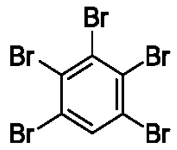
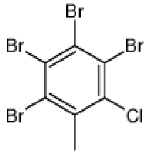
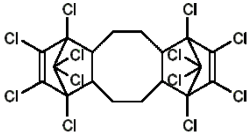
## Table 1.1 Physicochemical properties of some important EFRs, PBDEs and HBCDDs

| Compound | Acronym | Chemical structure | Molecular formula | Molecular weight | Melting point(°C) | Boiling point(°C) | Vapour pressure (Pa) (25 °C) | Water solubility (g/L) (25 °C) | Log $K_{OW}$ | Log $K_{OA}$ | Half-life in air (hours) | Half-life in water (hours) | Half-life in soil (hours) | Production volume | Type of BFR |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 2,2’,4,4’,5-Pentabromodiphenyl ether | BDE 99 |  | $C_{12}H_5Br_5O$ | 564.69 | 90.5-94.5 | 434.2 | 1.32E-07 [h] | 9E-06 [h] | 7.32 [h] | 11.31 [r] | 456 [n] | 19992 [n] | 19992 [n] | HPV [j] | Additive |
| 2,2’,4,4’,5,6’-Hexabromodiphenyl ether | BDE 154 |  | $C_{12}H_4Br_6O$ | 643.58 | N/A | 453.2 | 2.85E-08 [h] | 1E-06 [h] | 7.82 [h] | 11.92 [r] | N/A | N/A | N/A | HPV [j] | Additive |
| 2,2’,3,4,4’,5’,6-Heptabromodiphenyl ether | BDE 183 |  | $C_{12}H_3Br_7O$ | 722.48 | N/A | 490.7 | 3.51E-09 [h] | 2E-06 [h] | 8.27 [h] | 11.96 [r] | 1536 [m] | N/A | N/A | HPV [k] | Additive |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Decabromodiphenyl ether | BDE 209 |  | $C_{12}Br_{10}O$ | 959.17 [h] | 300-310 [h] | decomposes at >320 | 4.63E-06(21°C) [h] | <1E-07 [h] | 8.70 [i] | 18.42 [t] ,<br>14.98 [s] | 7632 [m] | N/A | N/A | HPV | Additive |
| Hexabromocyclodecane | HBCDD |  | $C_{12}H_6Br_6$ | 641.7 [c] | N/A | decomposes at >190 [c] | 1.04E-07 [c] | N/A | 7.92 [c] | 11.8 [o] | 40.8 [m] | 1.05E-15 (pH=7) [m] | N/A | HPV | Additive [c] |
| Decabromodiphenyl ethane | DBDPE |  | $C_{14}H_4Br_{10}$ | 971.2[a] | 334-337[b],<br>344-349[d],<br>348-353[e],<br>351-355[e] | 676±50[a] | 6.0E-15 [a]<br>1.0E-06 [e] | 2.10E-07 [a]<br>7.2E-04 [e] | 11.1 [a] | 18.8 [l]<br>19.34 [o] | 53.6 [l] | 4320 [l] | 8640 [l] | N/A | Additive [g] |
| 1,2-Bis(2,4,6-tribromophenoxy)ethane | BTBPE |  | $C_{14}H_8Br_6O_2$ | 687.6 [a] | N/A | 566.4±50.0[a] | 3.88E-10[a] | 1.90E-05 [a] | 7.88±0.86 [a] | 15.0 [l] | 8.6 [l] | 4320 [l] | 8640 [l] | LPV [g] | Additive [g] |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 2-Ethylhexyl-2,3,4,5-tetrabromobenzoate | EH-TBB (TBB) |  | $C_{15}H_{18}Br_4O_2$ | 549.9[c] | N/A | N/A | 3.71E-07 [e] | 1.14E-05 [f] | 7.73 [c]<br>8.75 [f] | 12.34 [q] | N/A | N/A | N/A | N/A | Additive [g] |
| Bis(2-ethylhexyl) tetrabromophthalate | BEH-TEBP (TBPH) |  | $C_{24}H_{34}Br_4O_4$ | 706.1 [a] | N/A | 584.8±45.0[a] | 1.55E-11 [a] | 1.60E-06 [a] | 10.08±0.94 [a] | 17.7 [l]<br>16.86 [o] | 5.9 [l] | 1440 [l] | 2880 [l] | LPV [g] | Additive [g] |
| Tetrabromobisphenol A-bis(2,3-dibromopropyl ether) | TBBPA-BDBPE (TBBPA-DBPE) |  | $C_{21}H_{20}Br_8O_2$ | 943.6 [a] | 90-105 [b],<br>90-100 [c] | 676.5±55.0[a] | 1.60E-07 [a] | 1.60E-07 [a] | 10.42±0.7 [a] | 21.1 [l] | 12.2 [l] | 4320 [l] | 8640 [l] | LPV [g] | Additive [g] |
| Pentabromoethylbenzene | PBEB |  | $C_8H_5Br_5$ | 500.7 [a] | 138 [b] | 413.3±40.0[a] | 3.2E-04 [a]<br>4.67E-05[e] | 3.50E-04 [a] | 6.40±0.62 [a] | 9.9 [l] | 111.6 [l] | 4320 [l] | 8640 [l] | LPV [g] | Additive [g] |
| 2,3-Dibromopropyl 2,4,6-tribromophenyl ether | TBP-DBPE (DPTE) |  | $C_9H_7Br_5O$ | 530.6 [c] | N/A | N/A | 1.86E-05 [c] | N/A | 5.82 [c] | N/A | N/A | N/A | N/A | N/A | Additive [c] |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Tetrabromoethylcyclohexane | DBE-DBCH (TBECH) |  | $C_8H_{12}Br_4$ | 427.8 [c] | N/A | N/A | 2.97E-03 [c]<br>1.05E-04[e] | 6.92E-05[e] | 4.82 [c],<br>5.24 [e],<br>5.25 [p] | 8.01 [p] | 52.8 [m] | 756864 (pH=7) [m] | N/A | N/A | Additive [g] |
| Benzene, 1,2,4,5-tetrabromo- 3,6-dimethyl | TBX (p-TBX) |  | $C_8H_6Br_4$ | 421.75 [c] | N/A | N/A | 5.80E-03 [c] | N/A | 6.2 [c] | 8.81[t] | N/A | N/A | N/A | N/A | Additive [c] |
| Pentabromotoluene | PBT |  | C7H3Br5 | 486.6 [g] | 280-282 [g],<br>288-289 [g] | 394.4±37 [g] | 1.22E-03 [g] | 7.80-E04 [g] | 5.87±0.62 [g],<br>5.43 [g],<br>6.99 [t],<br>6.26 [c] | 9.66 [u] | N/A | N/A | N/A | LPV [g] | Additive [c] |
| Hexabromobenzene | HBB |  | C6Br6 | 551.5 [g] | 327 [g],<br>326 [g] | 417.5±40[g] | 1.14E-04 [g]<br>3.17E-04 [g] | 7.70E-04[g],<br>1.10E-07[g] | 5.85±0.67 [g],<br>6.07 [g] | 10.26 [u] | 1992 [n] | 7584 [n] | 7584 [n] | N/A | Additive [c] |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pentabromobenzene | PBBz |  | C6HBr5 | 472.59 | N/A | N/A | N/A | N/A | 6.44[t] | 9.10[t] | N/A | N/A | N/A | N/A | N/A |
| Tetrabromo-o-chlorotoluene | TBCT<br>(TBoCT) |  | C7H3Br4Cl | 442.17[c] | N/A | N/A | 1.72E-03[c] | N/A | 6.29[c] | 8.82[t] | N/A | N/A | N/A | N/A | Additive[c] |
| Dechlorane plus | DDC-CO<br>(DP) |  | C18H12Cl12 | 653.7[v] | 206[w],<br>350 °C with decomposition[v] | N/A | 1.37E-11[c],<br>4.71E-08[w] | 4.0E-11[w] | 9.3[v],<br>10.12[c],<br>11.27[p] | 13.1[t] | N/A | N/A | N/A | N/A | Additive[c] |

a) Data from SciFinder originating from calculated properties (ACD/labs Software V9.04); b) Data from SciFinder data base originating from experimentally determined properties; c) Data from Bergman et al., (2012); d) Experimental data from Li et al. (2004); e) Experimental data from the Environment Agency Dungey, S and Akintoye (2007); e) Data from Syracuse Research Corporation. f) Data from US. EPA (2008a); (g) Information from Covaci et al., (2011); (h) Data from US. EPA (2008b); i) Data from Sifleet (2009); j) Data from Penta-BDE( Alaee et al. (2003)); k) Data from from octa-BDE (Alaee et al. (2003)); l) Data from NPCA (2008); m): Data from Nyholm (2009); n) Data from Wegmann et al. (2009); o) Data from Ruan et al. (2009); p) Data from Howard and Muir (2010); q): Data from Stapleton et al. (2008); r) Data from Harner and Shoeib (2002); s) Data from Cetin and Odabasi (2008); t) Data from HENRYWIN v3.20 (EPIWIN 4) u) Stenzel et al. (2013); v) Data from Xian et al. (2011); w) Data from Feo et al. (2012); HPV: high production volume (above 1000 tons/year); LPV: low production volume (below 1000 tons/year); N/A: not available.

### 1.1.8 Relationships between physicochemical properties and human exposure

The presence of EFRs in dust, air and biota is governed by several factors such as: type of flame retardant (reactive or additive), production volume, and physicochemical properties such as vapour pressure, $K_{OW}$, water solubility etc.

#### 1.1.8.1 Influence of production volume and types of EFR on human exposure

After the phase out of "legacy" BFRs, there is increasing demand for EFRs. However, there is very limited information about their production volumes. Dodson et al., (2012) found concentrations of Firemaster 550 (FM550) components (EH-TBB and BEH-TEBP) and DBDPE in 16 house dust samples from the USA were higher in 2011 than 2006, consistent with increased production and use of these EFRs. In a similar vein, Tian et al. (2011) reported DBDPE to exhibit the highest atmospheric concentrations other than PBDEs out of a group of EFRs monitored at e-waste and rural sites in China. The authors attributed this to the large production volume of DBDPE in China. These results indicate that the production volume affects environmental concentrations of EFRs.

BFRs are mainly divided into two subgroups depending on their mode of incorporation into polymers: reactive and additive. Reactive flame retardants, such as TBBP-A, are chemically bound to the plastics. Conversely, additive flame retardants, which include HBCDDs, PBDEs, DBDPE and BTBPE are simply blended with the polymers and are thus more easily released from the treated products. This is illustrated by the markedly higher concentrations of HBCDDs in indoor air, outdoor air and indoor dust compared to TBBP-A in spite of the higher production volume of TBBP-A compared to HBCDD (Abdallah et al., 2008). All the EFRs listed in Table 1.1 (except for PBBz which is unknown) are additive flame retardants; therefore, this makes them more prone to leach out of products leading to a potential for high levels in environmental and biotic samples.

#### 1.1.8.2 Influence of physicochemical properties on human exposure pathways

#### 1.1.8.3 Vapour pressure

Chemicals like PBEB, TBP-DBPE, TBX, PBT, HBB, TBCT and DBE-DBCH have relatively high vapour pressures which equates to likely existence in the gas phase in air samples in preference to the particulate phase. PBEB was found in a large number of air samples in many countries such as Denmark, Norway, Sweden (Nordic Council of Ministers, 2011), the US (Salamova and Hites 2011), and China (Tian et al. 2011). DBE-DBCH was the most frequently detected compound in indoor air in Sweden, with DBE-DBCH and PBT more frequently detected in indoor air than dust (Newton et al., 2015). Additionally, PBT and DDC-CO were detected in all, and TBP-DBPE, HBB, EH-TBB detected in >75% of air samples in Canada's Western Sub-Arctic (Yu et al., 2015). Inhalation – particularly of indoor air - will thus likely be an important route of human exposure to such chemicals.

#### 1.1.8.4 $K_{OW}$

BMF was found strongly, linearly related to log $K_{OW}$ for PBDEs and DDC-CO, with BMF increasing up to log $K_{OW}$ values of ~8 ($r^2 = 0.92$, $p < 0.00001$), and then markedly decreasing as $K_{OW}$ increased further (She et al., 2013). Burreau et al. (2004) also found that tri- to hepta-BDEs biomagnify with maximum biomagnification for penta-BDEs (log $K_{OW}$ = 6.46-6.97), whereas bioaccumulation of PBDEs with six or more bromine atoms appeared negatively correlated with the degree of bromination; octa-, nona- and deca-BDEs were bioavailable and detected in fish muscle but were not biomagnified (Burreau et al., 2004). Likewise, in Voorspoels et al. 's study (2007) of a rodent-buzzard food chain, biomagnification only increased with $K_{OW}$ for BDEs 47 to 154, with BDE 183 less biomagnified in buzzard than expected based on its $K_{OW}$. These observations are consistent with a bioaccumulation model that predicted that accumulation of organic chemicals in biota increases with increasing log

$K_{OW}$ up to log $K_{OW}$ = 7 but decreases at higher log $K_{OW}$ values (Thomann, 1989). This would also account for the observation that DBDPE bioaccumulated in rats to concentrations 3-5 orders of magnitude lower than BDE 209 after oral exposure to 100 mg/kg bw/day of DBDPE or BDE 209 for 90 days (log $K_{OW}$ of BDE 209 = 8.70 (Sifleet, 2009); log $K_{OW}$ of DBDPE = 11.10 (data from Scifinder)) (Wang et al., 2010). However, in stark contrast, He et al. (2012) found that DBDPE bioaccumulated in fish at concentrations one order of magnitude higher than those of BDE 209, even though DBDPE concentrations were lower than BDE 209 in both water and sediment samples to which the fish were exposed. This could be partly attributed to piscine metabolic debromination of BDE 209 and lack of metabolism for DBDPE. Moreover, the marked differences between the studies of Wang et al. (2010) and He et al. (2012) may reflect different bioavailability and biotransformation behaviour for DBDPE and BDE 209 in fish compared to rats. In summary, without considering biotransformation of the chemicals, compounds possessing log $K_{OW}$ values between around 7 to 8 may have the greatest potential for bioaccumulation. Based on this simple criterion, BTBPE, EH-TBB, PBEB, PBT and PBBz are potentially the most likely of the EFRs in Table 1.1 to display high bioaccumulative potential, and thus human exposure to these contaminants via the diet is likely to be substantial.

Perhaps more reliable are the evaluation criteria of EFSA (2012), who state that chemicals with log $K_{OW}$ between 3 and 11 and a metabolic rate constant ($k_m$) >1 day$^{-1}$ are likely to display high potential for bioaccumulation. Therefore, EFRs having a high potential for bioaccumulation according to these two heuristic rules are indicated in the box in Figure 1.1, namely: BTBPE, EH-TBB, DBE-DBCH, PBEB, PBT, HBB, TBX and TBCO. These EFRs exhibit potential to distribute in the sediment and biotic samples and dietary intake may be their main exposure pathway for humans.

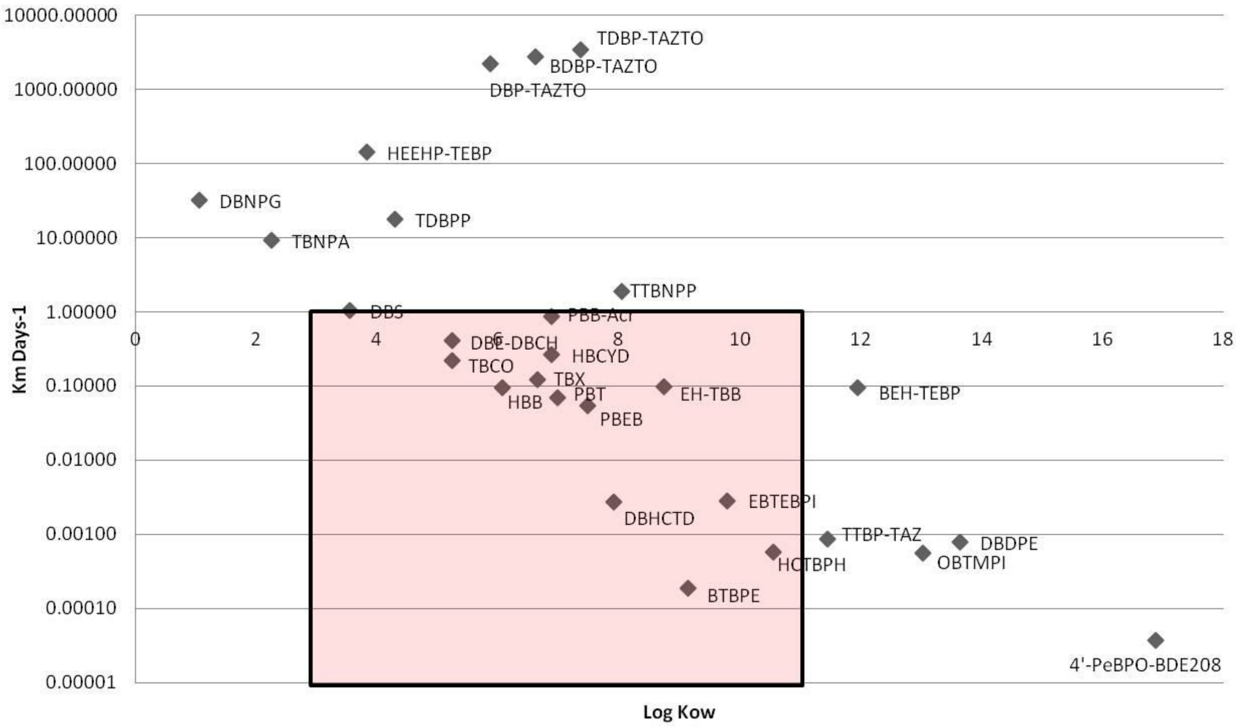


Figure 1.1 Potential for bioaccumulation (EFSA 2012)

#### 1.1.8.5 Water solubility

While all of the EFRs in Table 1.1 have very low water solubility, BTBPE and EH-TBB show higher water solubility and lower vapour pressure compared to other EFRs and may thus be found in water to a limited extent. Coupled with their bioaccumulative potential, this means that BTBPE and EH-TBB are likely to be prevalent in aquatic foodstuffs consumed by humans.

## 1.2 Applications and use

Application, uses and production volumes of some important EFRs are shown in Table 1.2. As replacements for "legacy" FRs, these EFRs were produced and used widely worldwide in electrical and electronic appliances, building insulation foam, textiles, construction materials, adhesives, polyurethane foam and so on. Despite the wide variety of potential applications, information about EFR production volumes is scarce. Compared to the estimated total consumption of Penta-BDEs (7,500 tons), Octa-BDEs (3,790 tons), Deca-BDEs (56,100 tons) in 2001 (BSEF, 2006) and annual worldwide production of HBCDDs (~28,000 tons) (Yang, 2014), reported global production volumes of DBDPE and BTBPE were comparable to those of Deca-BDEs and HBCDDs while worldwide production of BEH-TEBP, PBT, DDC-CO, and TBBPA-BDBPE was comparable to those of Penta-BDEs and Octa-BDEs. Production volume information for other EFRs is either not available, or reveals production to be low. More detailed, up-to-date information on production of EFRs is needed to test the hypothesis that their production has increased following restriction of "legacy" FRs.

## 1.3 Toxicology and health effects

### 1.3.1 DBDPE

DBDPE does not display acute toxicity in laboratory animals, with single-dose and long-term (90 days) oral administration of DBDPE in rats yielding a lethal dose (LD50) > 5000 mg/kg bw and an acute dermal LD50 > 2000 mg/kg bw for rabbits (Harju et al., 2009). However, the DBDPE concentrations in dust were positively associated with triiodothyronine (T3) levels in the exposed men, indicating exposure to DBDPE in indoor dust may be leading to endocrine disruption (adverse health effects which human and wildlife have suffered from exposed to chemicals that interact with the endocrine system (Kavlock et al., 1996)) in males (Johnson et al., 2013). This epidemiological finding was consistent with the laboratory study of Wang et al. (2010), which found a significant increase in the T3 level in male rats following DBDPE treatment, suggesting that DBDPE can alter thyroid hormone homeostasis. DBDPE-induced hepatotoxicity in rats was also indicated by serum clinical chemistry data for AST, ALP, Cr, and TBA (Wang et al., 2010). In aquatic organisms, DBDPE was reported to be acutely toxic to *Daphnia magna*, reduced the hatching rates of exposed zebra-fish eggs and raised significantly the mortality of hatched larvae (Nakari and Huhtala, 2009). Moreover, in rainbow trout and brown trout hepatocytes, DBDPE increased vitellogenin synthesis (indicating DBDPE to be estrogenic), inhibited CYP1-dependent monooxygenase activity, and increased the activity of UGT (Nakari and Huhtala, 2009). Additionally, DBDPE (at doses of 0.1 and 0.2 μM) induced AhR-regulated CYP1A4 and CYP1A5 mRNA in primary culture of chicken embryonic hepatocytes (Egloff et al., 2011).

### 1.3.2 BTBPE

Studies using *Salmonella typhimurium* strains TA98, TA100, TA 1535, TA1537 and TA1538 and *Saccharomyces cerevisiae* strain D4, both in the presence and absence of metabolic activation, revealed BTBPE to display a lack of mutagenicity (Harju et al., 2009). Harju et al. (2009) also reported the lethal concentration to be > 36.7 g/m$^3$ in rats following BTBPE inhalation for period of 4 hours, with the only relevant effects observed being behavioural modification, gastrointestinal lesions, and dermatitis. Dermal application to rabbits established the lethal dose to be > 10 g/kg (Harju et al., 2009). Acute toxicity studies showed the oral LD50 of BTBPE to be >10 g/kg b.w. for rats and dogs (Matthews, 1984, as cited by Nomeir et al., 1993). No obvious effect was seen for rats exposed to BTBPE in the diet at a concentration of 500 mg/kg, corresponding to 35 mg/kg b.w. for 14 days (Nomeir et al., 1993). Egloff et al. (2011) investigated the toxicity of BTBPE in chicken embryos. The embryonic pipping success was not affected at any of the injected doses (from 0.1 to 10 μg/g egg). The changes in the relative messenger RNA (mRNA) abundance of 11 genes involved in xenobiotic biotransformation, lipid metabolism and thyroid hormone homeostasis were investigated in the liver of pipped embryos, and in primary cultured chicken embryo hepatocytes exposed to various BTBPE concentrations. BTBPE significantly upregulated the expression of cytochrome P-450 (CYP) 1A4 and 1A5 mRNA levels *in vitro* (at ≥ 0.03 μM) and *in vivo* (at 3 μg/g egg). A dose-dependent decrease in type III iodothyronine 5‟-deiodinase mRNA was observed in embryonic livers, whereas type I iodothyronine deiodinase was upregulated in chicken embryo hepatocytes (Egloff et al., 2011). Moreover, it has been reported that BTBPE may cause antiestrogenic effects (Ezechiáš et al., 2012).

### 1.3.3 EH-TBB and BEH-TEBP

The Firemaster 550 formulation which contains both EH-TBB and BEH-TEBP, was shown to be an endocrine disruptor and an obesogen at environmentally relevant levels in rats (Patisaul et al., 2013) and caused DNA damage to fathead minnows (Bearr et al., 2010). EH-TBB and BEH-TEBP were reported to show the endocrine disrupting potentials using the yeast YES/YAS reporter assay and the mammalian H295R steroidogenesis assay (Saunders et al., 2013). In one study investigating the effects of EH-TBB and BEH-TEBP on steroidogenesis in a porcine primary testicular cell model, it was shown that EH-TBB exposure appeared to favour synthesis of cortisol and aldosterone, while BEH-TEBP exposures selectively produced more sex hormones (Mankidy et al., 2014). Concentrations of BEH-TEBP in indoor dust were positively associated with total T3 in exposed men, suggesting that exposure to contaminants in indoor dust may be leading to endocrine disruption in men (Johnson et al., 2013). Noyes et al. (2015) studied a new morphological and behavioural testing platform with embryonic zebrafish to characterise the developmental toxicity of some flame retardants. Their results indicated zebrafish neurodevelopment was sensitive to EH-TBB and BEH-TEBP.

### 1.3.4 TBBPA-BDBPE

The $LD_{50}$ of TBBPA-BDBPE was found to be > 20 g/kg for oral and dermal administration in mice and no abnormal gross symptoms or death were observed, suggesting that TBBPA-BDBPE is not acutely toxic (WHO, 1995). In mutagenicity tests performed with *Salmonella typhimurium* strains TA100 and TA1535, TBBPA-BDBPE caused positive responses both in the presence and absence of metabolic activation. However, its mutagenicity in the absence of metabolic activation was found to be higher than in its presence, suggesting any metabolites to be less mutagenic (Haneke, 2002). No endocrine effect of TBBPA-BDBPE was found in tests based on aromatase enzymes (CYP19 and CYP17) in H295R adrenal cortical cells (Cantón et al., 2005). In addition, no effects of TBBPA-BDBPE on the aryl hydrocarbon receptor, the androgen receptor, progesterone receptor and estrogen receptor were found, although TBBPA-BDBPE has a high potency of estradiol sulfotransferase inhibition and can compete with thyroxine for binding to the plasma transport protein transthyretin (Hamers, 2006).

### 1.3.5 DBE-DBCH

Liver and gonad somatic indices were not affected in juvenile brown trout following dietary exposure to three concentrations of β-DBE-DBCH (2.02 (low), 14.7 (medium) and 118.4 (high) pmoles/g fat at a feed ration of 1 % of their b.w.) (Gemmill et al., 2011). There were also no significant differences in circulating plasma levels of 17 β-estradiol, testosterone and 11-ketotestosterone between the treatment groups and no evidence of isomerisation of the β-isomer to other isoforms *in vivo* (Gemmill et al., 2011). In a follow-up study using the same dose levels, a significant reduction in total plasma thyroxine in the high dose group and a significant increase in mean thyroid epithelial cell height in the three different dose groups were found, while these differences disappeared at the end of the depuration phase, with the growth of the fish unaffected (Park et al., 2011). Larsson et al. (2006) found that DBE-DBCH bound to and activated the human androgen receptor (AR), and can compete with dihydrotestosterone (DHT) in a receptor-binding assay. Furthermore, Khalaf et al. (2009) reported that the γ- and δ-DBE-DBCH activated the human androgen receptor (AR) at concentrations that are comparable to those of DHT. While the γ- and δ-isomers were found to be more potent activators of AR than α- and β-DBE-DBCH, all diastereomers activated the AR in the human prostate cell line LNCaP, indicating DBE-DBCH to be a potential endocrine disrupter, and comparably increased expression of prostate specific antigen (PSA). Moreover, DBE-DBCH was reported to be mutagenic in the absence of metabolic activation in a L1578Y tk+/tk-mouse lymphoma-cell forward-mutation assay (McGregor et al., 1991). Furthermore, DBE-DBCH diastereomers bound to the zebrafish androgen receptor *in vitro* and *in vivo*, indicating that DBE-DBCH has androgenic properties (Pradhan et al., 2013). In the same study, DBE-DBCH displayed a negative effect on zebrafish hatching and induced morphological abnormalities and juvenile mortality (Pradhan et al., 2013).

### 1.3.6 DDC-CO

The developmental and reproductive toxicity of DDC-CO in rats was investigated, with no effects observed on in-life parameters or clinical or anatomic pathology, resulting in a no-observable-effect level (NOEL) in both repeat dose toxicity and developmental or reproductive toxicity phases of the study of 5000 mg/kg (Brock et al., 2010). DDC-CO has been shown to have very low toxicity to fish (no acute toxicity with regard to bluegill sunfish—*Lepomis macrochirus*—was seen at the highest test concentration of 100 mg/L) (US EPA, 2009). No adverse effects of DDC-CO on embryonic viability or pathways associated with the genes assessed are predicted at current environmental exposure levels (Crump et al., 2011). However, a significant dose-related decrease of liver and ovary weights was observed in rabbits compared to control animals following exposure to DDC-CO through the skin (US EPA, 2009). Furthermore, DDC-CO is reported to induce hepatic oxidative damage and perturbations of metabolism and signal transduction in rats and cause significant alteration of gene expression involved in carbohydrate, lipid, nucleotide, and energy metabolism, as well as signal transduction processes (Wu et al., 2012).

### 1.3.7 Other EFRs

PBT and HBB are both reported as able to bind and activate the Ah receptor (Brown et al., 2004). There were no evident signs of PBT toxicity when rats were exposed orally to PBT at doses ranging from 0.05 to 500 mg/kg for 91 days, except some histological changes in the thyroid gland, liver and kidneys (Harju et al., 2009). There was also no observed mutagenic activity as assessed by *S. typhimurium* assays, either in the presence or in the absence of metabolic activation (Harju et al., 2009). HBB shows very low toxicity to rats, quail and chickens. The lowest toxic dose reported following oral exposure to HBB in rats, quail and chickens was 150 mg/kg, 1.5 g/kg/ 15 days and 52.5 g/kg/ 12 weeks, respectively, and dietary exposure to HBB gave rise to effects on the liver, enzyme inhibition and induction of, or changes in, blood or tissue levels of rats, quail and chickens (Harju et al., 2009). Larsson et al. (2006) reported that HBB is not an agonist or antagonist of the androgen receptor as shown by tests in human liver carcinoma cells. Conversely, an increase in the porphyrin level in rats following HBB exposure suggested porphyrogenic activity (Szymańska and Piotrowski, 2000).

PBEB has been tested for acute toxicity in rabbits with $LD_{50}$ values > 8 g/kg reported (Harju et al., 2009). No mutagenic activity was observed for this compound according to *S. typhimurium* assays (Harju et al., 2009). TBP-DBPE was demonstrated to be a potent AR antagonist in humans, zebrafish, and chickens, with observed alterations in LAT gene transcription suggesting that this compound should be considered as a potential neurotoxic and endocrine disrupting compound (Asnake et al., 2015; Pradhan et al., 2015).

No recent relevant toxicity data appear to be available for TBX, PBBz and TBCT.

## Table 1.2 Application, uses and production volumes of some important EFRs

| Compounds | Application and uses | Product (Company) | Production/import volume |
|---|---|---|---|
| DBDPE | ABS, polycarbonate/ABS, HIPS/polyphenylene oxide and textiles [a] | Saytex® 8010 (Albemarle Corp.) [a]<br>Firemaster® 2100 (Chemtura Corp.) [a] | ~1000 and 5000 tons (European imports, primarily to Germany) in 2001 [a]<br>12,000 tons (production in China) in 2006 [a] |
| BTBPE | ABS, HIPS, thermoplastics, thermoset resins, polycarbonate and coatings [a] | FF-680 (Chemtura Corp.) [a] | 450-4500 tons/year in the USA after 1998 [a]<br>~16,710 tons (worldwide) in 2001 [a] |
| EH-TBB(TBB) | PUF applications [a] | Firemaster 550 (Chemtura Corp.) [a]<br>Fire retardant BZ-54 (Chemtura Corp.) [a] | N/A |
| BEH-TEBP(TBPH) | PUF applications, PVC and neoprene, wire and cable insulation, film and sheeting, carpet backing, coated fabrics, wall coverings and adhesives [a] | Firemaster 550 (Chemtura Corp.) [a]<br>Fire retardant BZ-54(Chemtura Corp.) [a]<br>DP-45(Chemtura Corp.) [a] | 450 to 4500 tons/year from 1990 to 2006 [a] |
| TBBPA-BDBPE (TBBPA-DBPE) | plastic products, such as pipes, water barriers, kitchen hoods and electronics [a] | now ICL Industrial Products, Israel, Albemarle Corp., the Great Lakes Chemical Corporation/Chemtura, [a]<br>JiangSu HaoHua Fine Chemical Co., Ltd and Weidong International Group, Ltd. in China [a] | 4000 tons in China in 2006 [a]<br>< 4500 tons in the USA in 2006 [a]<br>3000 tons/year (JiangSu HaoHua Fine Chemical Co., Ltd) in China [a] |
| PBEB | thermoset polyester resins (circuit boards, textiles, adhesives, wire and cable coatings, polyurethane foam) [a], unsaturated polyesters, styrene butadiene copolymers, textiles [b] | Albemarle Corp. [a]<br>FR-105 (now ICL Industrial Products) [a] | N/A |
| TBP-DBPE(DPTE) | N/A | Bromkal 73-5PE (Chemische Fabrik Kalk in Germany) [c] | N/A |

|  |  |  |  |
|---|---|---|---|
| DBE-DBCH(TBECH) | expandable PS beads for house insulation, extrudedpolystyrene, for adhesives in fabric, electrical cable coatings, high impact plastic in appliances and some construction materials [a] | Saytex BCL-462 Albemarle Corp. (contains equal amounts of the two diastereomers, α-and β-TBECH) [a] | 4.5 to 230 tons/year in the US from1986 to 2002 [a] |
| TBX(p-TBX) | N/A | N/A | N/A |
| PBT | unsaturated polyesters, polyethylene, polypropylene, polystyrene, SBR-latex, textile, rubbers, ABS (acrylonitrile butadiene styrene terpolymer) [a] | Flammex 5-BT (Berk Ltd., U.K.) [a]<br>FR-105 (ICL Industrial Products, Israel and Chemtura, US) [a] | ~1000 and 5000 tons/ year [a]<br>600 tons/year at Shou Guang Longfa Chemical Co. Ltd in China [a] |
| HBB | paper, woods, textiles, electronic and plastic goods [a] | FR-B (Japanese Nippoh Chemicals Corp) [a]<br>Dayang Chemicals in China [a]<br>AFR 1001, HBB (flame retardant), HBB-S, NSC 113975 [d] | 350 tons in 2001 in Japan [a]<br>600 tons/year (Shou Guang Longfa Chemical Co. Ltd.) in China [a] |
| PBBz | N/A | N/A | N/A |
| TBCT(TBoCT) | N/A | N/A | N/A |
| DDC-CO(DP) | nylons, polybutylene terephthalate (PBT), polypropylene (PP), ABS, epoxy resins, electric wire and cable, and synthetic elastomers [e]<br>plastic connectors in televisions and computer monitors, and furniture [f]<br>plastic roofing materials and other polymeric systems for fire prevention [g] | DP,DP-25,DP-35, and DP-515 (Hooker Chemical Corporation, now called Occidental Chemical Corporation (Oxychem), in Buffalo, USA) [e]<br>Anpon Electrochemical Co., Ltd in Jiangsu province in China [e] | 5000 tons/ year (worldwide) [e]<br>300-1,000 tons/ year in China [g] |

a) Covaci, A., Harrad, S., Abdallah, M.A.E., Ali, N., Law, R.J., Herzke, D., de Wit, C.A., 2011. Novel brominated flame retardants: A review of their analysis, environmental fate and behaviour. Environ. Int. 37, 532–556.; b) Wu, J.-P., Guan, Y.-T., Zhang, Y., Luo, X.-J., Zhi, H., Chen, S.-J., Mai, B.-X., 2011. Several current-use, non-PBDE brominated flame retardants are highly bioaccumulative: Evidence from field determined bioaccumulation factors. Environ. Int. 37, 210–215.; c) De Wit, C., Kierkegaard, A., Ricklund, N., Sellström, U., 2011. Emerging Brominated Flame Retardants in the Environment Brominated Flame Retardants, in: Eljarrat, E., Barceló, D. (Eds.), . Springer Berlin / Heidelberg, pp. 241–286.; d) Brominated Flame Retardants (BFRs) in Food. EFSA Journal 2012;10(10):2908. [125 pp.]. Available online: www.efsa.europa.eu/efsajournal; e) Xian, Q., Siddique, S., Li, T., Feng, Y., Takser, L., Zhu, J., 2011. Sources and environmental behavior of dechlorane plus — A review. Environ. Int. 37, 1273–1284.; f) Sverko, E., Tomy, G.T., Reiner, E.J., Li, Y.F., McCarry, B.E., Arnot, J.A., Law, R.J., Hites, R.A., 2011. Dechlorane plus and related compounds in the environment: a review. Environ. Sci. Technol. 45, 5088–98.; g) Feo, M.L., Barón, E., Eljarrat, E., Barceló, D., 2012. Dechlorane Plus and related compounds in aquatic and terrestrial biota: a review. Anal. Bioanal. Chem. 404, 2625–2637.

## 1.4 Concentrations of EFRs in air and dust

Multiple studies have reported the concentrations, fate and behaviour of EFRs in both indoor and outdoor environments, as shown in Table 1.3. EH-TBB, BEH-TEBP, DBDPE, BTBPE, HBB, TBX, PBBz, PBEB, PBT, TBP-DBPE, as well as *syn*- and *anti*-DDC-CO were detectable in European Arctic air (Möller et al., 2011a, 2011b; Salamova et al., 2014) or Canada's Western Sub-Arctic (Yu et al., 2015) suggesting that these compounds can undergo long-range atmospheric transport. This is supported by the detection of PBT, PBEB, HBB, EH-TBB, BEH-TEBP, BTBPE, TBP-DBPE, and DDC-COs in various animals from remote areas such as the Norwegian Arctic and Greenland (Mckinney et al., 2011a; Verreault et al., 2007; Vorkamp et al., 2015). The concentrations of some EFRs, especially HBB, TBP-DBPE, PBT, EH-TBB, BEH-TEBP, DBDPE and DDC-COs were similar to or higher than those of PBDEs in the Arctic, indicating a trend away from PBDEs towards EFRs (Möller et al., 2011a, 2011b; Salamova et al., 2014).

Very little is known about the levels of EFRs in indoor air. DBDPE has been determined in indoor air in a recycling facility at a concentration of 0.6 ng/m$^3$ in Sweden (Kierkegaard et al., 2004) which exceeds concentrations reported for one Swedish household (22.9 pg/m$^3$) (Karlsson et al., 2007) and those detected more recently in Swedish offices, apartments, stores, and schools (<90-250 pg/m$^3$) (Newton et al., 2015). BTBPE was also measured at high concentrations (<0.6–67 ng/m$^3$) in indoor air from various electronic dismantling sites in Sweden (Pettersson-Julander et al., 2004; Sjödin et al., 2001), but was not detected in air from Swedish offices, apartments, stores, and schools (Newton et al., 2015) nor in households and primary schools in Norway (Cequier et al., 2014).

The more volatile compounds such as DBE-DBCH, PBT, and PBEB, similar to BDE 28, 47, 99, and 100, were more frequently detected in indoor air samples (Cequier et al., 2014;

Newton et al., 2015), and DBE-DBCH was the most abundant EFR found in indoor air in Sweden (Newton et al., 2015) and Norway (Cequier et al., 2014).

Less volatile EFRs such as BEH-TEBP, DDC-CO, and DBDPE were found much more frequently than more volatile EFRs like DBE-DBCH, PBT, and PBEB in indoor dust than air (Cequier et al., 2014; Newton et al., 2015; Qi et al., 2014; Shoeib et al., 2012) (Table 1.3 and 1.4). Levels of DBDPE, BTBPE and PBDEs determined in floor and furniture dust from the living room and bedroom of five households in Sweden were 47 ng/g (<0.455-121), 4.8 ng/g (2.5-8.2) and 692.6 ng/g (174.4-1722.5), respectively (Karlsson et al. 2007). The same study reported PBDEs in the blood plasma of householders at a mean concentration of 33.7 ng/g lw; with EFRs not found above detection limits in the same individuals (<1.31 ng/g lw for BTBPE and <1.03 ng/g lw for DBDPE) (Karlsson et al. 2007). In North America, Stapleton et al. (2008) reported DBDPE, BTBPE, BEH-TEBP, and EH-TBB in domestic dust collected from 19 homes from Boston, USA. In living area, bedroom, and vacuum bag dust, geometric mean concentrations (ranges) of these were: DBDPE: 138 ng/g (<10-11,070), 153 ng/g (<10-3240), and 39.4 ng/g (<10-262); BTBPE 48 ng/g (4.7-654), 48 ng/g (1.6-789), and 18 ng/g (2.5-219); BEH-TEBP 234 ng/g (3.0-10,630), 105 ng/g (1.5-763), and 66 ng/g (24.3-111); and EH-TBB 322 ng/g (<6.6-15,030), 90 ng/g (<11-378), and 92 ng/g (35.7-669), respectively. In a study monitoring a wide range of EFRs in indoor dust in Norwegian households and schools, the most abundant compounds were DBDPE and BEH-TEBP in both living rooms and classrooms (Cequier et al., 2014). Especially in house dust samples, DBDPE and BEH-TEBP were found to be the main congeners in various countries such as the USA (Dodson et al., 2012), China (Qi et al., 2014), Sweden (Sahlström et al., 2015), Germany (Fromme et al., 2014), Belgium and the UK (Ali et al. 2011a).

Dust-air partitioning coefficients ($K_{DA}$) (expressed as the quotient of matched dust and indoor air samples ($m^3/g$)) for compounds were found to be strongly correlated with $K_{OA}$ values for three EFRs: DBE-DBCH, PBT, HBB, and BDE 47 ($R^2 = 0.9965$, $P < 0.005$) (Newton, 2015). Cequier et al. (2014) reported similar highly significant linear correlations for 20 FRs in the living rooms sampled including DBE-DBCH, PBT, and HBB. This correlation suggests that thermodynamic equilibrium between dust and air is reached for these compounds.

## Table 1.3a Summary of median concentrations of EFRs in air and dust (range values in parentheses)

| **Matrix** | DBDPE | BTBPE | EH-TBB | BEH-TEBP | TBBPA-BDBPE | DBE-DBCH | Region | References |
|---|---|---|---|---|---|---|---|---|
| Ambient air (pg/m$^3$) |  | 6.7 (2.8-70) |  |  |  |  | USA | (Hoh et al., 2005) |
|  | 1842 (402-3578) | 25.7(3.83-67.4) |  |  | 371(131-1240) |  | China | (Shi et al., 2009) |
|  |  | 0.4-1.8 $^a$ |  | 0.5-8 $^a$ |  |  | USA | (Ma et al., 2013) |
|  |  | nd-0.06 $^b$ |  |  |  |  | Greenland | (Möller et al., 2011b) |
|  | 23 $^a$ | 0.73 $^a$ |  |  |  |  | China | (Qiu et al., 2010) |
|  |  | 0.044 (0.024-0.222) | 0.143(0.024-1.21) | 0.353(0.028-5.55) |  |  | Canada, Western sub-arctic | (Yu et al., 2015) |
|  | 0.27 (0.04-2.2) | 0.03 (0.01-0.09) | 2.2(0.17-58) | 1.8(0.27-14) |  |  | Longyearbyen, Svalbard | (Salamova et al., 2014) |
|  | 1.0-22 | 0.5-1.2 |  |  |  |  | USA, near Great Lakes | (Venier and Hites, 2008) |
|  | 0.15(<0.12-0.33) | <0.077(<0.077-0.26) | 0.13(<0.029-0.47) |  |  | 0.46(0.18-2.4) | Sweden | (Newton et al., 2015) |
|  | 2.95(<5.90-216) | 1.26(<0.06-90.5) | 0.03(<0.06-1.19) | 0.35(<0.69-64.2) |  |  | East Africa | (Arinaitwe et al., 2014) |
|  | nd-2240 | 4.49-398 |  |  |  |  | China, e-waste | (Tian et al., 2011b) |
|  | 3.97-1370 | nd-28.4 |  |  |  |  | China, rural area | (Tian et al., 2011b) |
| Indoor air (pg/m$^3$) | <90(<90-250) |  |  | <35(<35-150) |  | 55(7.0-130) | Sweden | (Newton et al., 2015) |
|  |  | 20 $^a$(5.6-67) ng/m$^3$ |  |  |  |  | Sweden, e-waste | (Sjödin et al., 2000) |
|  | 0.6 ng/m$^3$ |  |  |  |  |  | Sweden, e-waste | (Kierkegaard et al., 2004) |
|  | 8.30(na-20.6) |  |  | nd(na-6.32) |  | 46.6(na-399) | Norway, classroom | (Cequier et al., 2014) |
|  | 0.01–1.2 ng/m$^3$ | <0.6–39 ng/m$^3$ |  |  |  |  | Sweden, e-waste | (Pettersson-Julander et al., 2004) |
| House air (pg/m$^3$) | nd(na-963) |  |  | nd(na-24.2) |  | 77.9(na-4120) | Norway, living room | (Cequier et al., 2014) |
|  | nd(nd-22.9) |  |  |  |  |  | Sweden | (Karlsson et al., 2007) |
| Indoor dust (ng/g) | 12(<0.41-2200) | 17(<0.76-150) | 9.1(<2.5-65) | 140(<33-1500) |  | 1.2(<0.073-3.8) | Sweden | (Newton et al., 2015) |
|  | 280(nd-16000) | 2.5(0.21-220) | 0.83(nd-6300) | 29(nd-1600) |  |  | China | (Qi et al., 2014) |
|  | 156(na-360) | 6.55(na-53.0) | 3.32(na-5.72) | 103(na-151) |  | 2.10(na-10.0) | Norway, classroom | (Cequier et al., 2014) |
| Office dust (ng/g) | 750-16200 | 6.5-11700 | 4.0-66.4 | 42.7-928 |  |  | China | (Cao et al., 2014) |
|  | 721(170-1846) | 19(2-384) | 7(<2-31) | 64(16-265) | 306(<20-2211) |  | Belgium | (Ali et al., 2011a) |
|  | 99(nd-860) | nd(nd-40) |  |  |  |  | UK | (Harrad et al., 2008a) |
| House dust (ng/g) | 14(5-850) | 3.15(<0.2-397) | 0.03(<0.2-4.5) | 3.5(<0.2-141) |  |  | Pakistan | ( Ali et al., 2012b) |
|  | 153(55-2126) | 2(<0.5-1019) | 1(<2-436) | 13(<2-5004) | 78(<20-1286) |  | Belgium | (Ali et al., 2011a) |
|  | 23(<2-1430) | 2(<2-175) | 2(<2-2285) | 12(<2-640) |  |  | New Zealand | (Ali et al., 2012a) |
|  | 201(<10-11070) | 30(1.6-789) | 133(<6.6-15030) | 142(1.5-10630) |  |  | USA | (Stapleton et al., 2008) |
|  | 2733(100-47000) | 6.47(nd-211) |  |  |  |  | China | (Wang et al., 2010) |
|  | 24(nd-3400) | 5.3(nd-1900) |  |  |  |  | UK | (Harrad et al., 2008a) |
|  | 140(18-2800) | 12(3-130) | 100(45-5900) | 260(<2-3800) | 7(<10-560) | <2(<2-25) α-DBE-DBCH;<br><2(<2-26) β-DBE-DBCH | USA | (Dodson et al., 2012) |
|  | 146(47-1570) | <10(<10-34) | <3.0(<3.0-13.6) | 343(25-2274) |  |  | Germany | (Fromme et al., 2014) |
|  | 150(43-1500) | 6.3(1.1-36) | 2.6(<0.29-280) | 61(<10-340) |  | 3.3(<0.6-19) α-DBE-DBCH;<br>3.4(0.77-16) β-DBE-DBCH | Sweden | (Sahlström et al., 2015) |
|  |  | 30(1.8-610) | 120(<0.30-18000) | 99(10-6400) |  |  | Canada | (Shoeib et al., 2012) |
|  | 147(na-4460) | 3.76(na-41.9) | 2.54(na-245) | 78.5(na-809) |  | 1.72(na-172) | Norway | (Cequier et al., 2014) |

a) Average concentration; b) Gaseous phase; c) Total DDC-CO concentration; d) Passive air sampling; e) Active air sampling; na: not available; nd: not detectable.

## Table 1.3b Summary of median concentrations of EFRs in air and dust (range values in parentheses) (Continued)

| **Matrix** | PBEB | TBP-DBPE | TBX | PBT | HBB | PBBz | TBCT | *Syn*-DDC-CO | *Anti*-DDC-CO | Region | References |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Ambient air ($pg/m^3$) | | | | 0.001-0.02 $^{b}$ | 0.04-0.66 $^{b}$ | | | | | Greenland | (Möller et al., 2011b) |
| | | | | | 1.7 $^{a}$ (0.3-6.5) | | | 3.5 $^{a}$ | | China | (Qiu et al., 2010) |
| | nd-0.01 | | | nd-0.02 | 0.02-0.09 | | | | | Canada | (Gouteux et al., 2008) |
| | 0.011(0.002-0.078) | 0.034(0.006-0.201) | | 0.055 (0.007-0.47) | 0.015(0.003-0.104) | | | 0.050(0.003-0.722) | 0.046(0.005-1.04) | Canada, Western sub-arctic | (Yu et al., 2015) |
| | 0.03(0.01-0.24) | | 0.07(0.01-0.16) | | 0.12(0.01-1.7) | 0.04(0.01-1.1) | | 0.22(0.05-0.91) | 0.55(0.15-4.2) | Longyearbyen, Svalbard | (Salamova et al., 2014) |
| | | | | | | | | 0.8-20 $^{c}$ | | USA, near Great Lakes | (Venier and Hites, 2008) |
| | | | | | | | | <1-9.0 | <1-33.1 | Greenland | (Vorkamp et al., 2015) |
| | | | | 0.17(0.066-0.50) | <0.053(<0.053-0.26) | | | | | Sweden | (Newton et al., 2015) |
| | 0.03(<0.06-1.83) | | | 0.03(<0.06-22.2) | 0.05(<0.09-1.47) | | | 0.03(<0.05-4.39) | 0.03(<0.05-1.37) | East Africa | (Arinaitwe et al., 2014) |
| | 0.29-867 | | nd-158 | 0.19-125 | 4.47-559 | | | | | China, e-waste | (Tian et al., 2011b) |
| | 0.10-4.80 | | nd-3.43 | 0.21-3.57 | 0.42-13.9 | | | | | China, rural area | (Tian et al., 2011b) |
| | | | | | | | | 6.9 $^{d}$ /0.4 $^{e\ a}$ | | China, urban area | (Ma et al., 2011) |
| Indoor air ($pg/m^3$) | | | | 11(2.6-29) | 3.6(<5.1-64) | | | <36(<36-48) $^{c}$ | | Sweden | (Newton et al., 2015) |
| | nd(na-nd) | 0.841(na-10.6) | nd(na-2.92) | 2.57(na-4.14) | 4.03(na-6.52) | | | nd(na-nd) | nd(na-nd) | Norway, classroom | (Cequier et al., 2014) |
| House air ($pg/m^3$) | 0.531(na-30.6) | 0.789(na-132) | nd(na-2830) | 7.64(na-213) | 4.11(na-297) | | | nd(na-7.39) | nd(na-7.61) | Norway, living room | (Cequier et al., 2014) |
| Indoor dust (ng/g) | | | | 1.1(<0.066-5.5) | 3.1(<0.26-69) | | | 4.8(<0.19-4800) $^{c}$ | | Sweden | (Newton et al., 2015) |
| | nd(nd-4.8) | nd(nd-26) | | 0.60(nd-34) | 0.57(nd-260) | 0.14(nd-3.6) | | | | China | (Qi et al., 2014) |
| | nd(na-0.103) | 0.178(na-0.707) | nd(na-nd) | 0.232(na-1.06) | 0.901(na-5.27) | | | 0.921(na-3.13) | 2.63(na-9.25) | Norway, classroom | (Cequier et al., 2014) |
| Office dust (ng/g) | 0.15(nd-2.05) | | | 1.52(0.22-12.0) | 21.0(3.22-658) | | | | | China | (Wang et al., 2010) |
| House dust (ng/g) | | | | | <2(<2-13) | | | | | USA | (Dodson et al., 2012) |
| | | | | 2.8(<0.20-100) | 8.9(<0.32-170) | 0.99(<0.29-11) | | | | Sweden | (Sahlström et al., 2015) |
| | <0.07-4.1 | 1.50(<0.30-1200) | | 0.30(<0.02-12) | 3.7(<0.02-130) | | <0.02-7.1 | 2.8(<0.70-170) | 4.0(<0.70-170) | Canada | (Shoeib et al., 2012) |
| | | | | | | | | 14(2.3-5683) $^{c}$ | | Canada | (Zhu et al., 2007) |
| | | | | | | | | 212(nd-8810) | 325(nd-12200) | South China, e-waste | (Wang et al., 2011) |
| | | | | | | | | 2.98(nd-21.6) | 12.4(1.83-62.1) | South China, Guangzhou | (Wang et al., 2011) |
| | | | | | | | | 0.971(nd-7.97) | 2.49(nd-20.7) | South China, rural area | (Wang et al., 2011) |
| | nd(na-8.00) | 0.505(na-21.4) | nd(na-88.8) | 0.633(na-16.1) | 0.671(na-8.94) | | | 1.45(na-311) | 4.16(na-590) | Norway | (Cequier et al., 2014) |

a) Average concentration; b) Gaseous phase; c) Total DDC-CO concentration; d) Passive air sampling; e) Active air sampling; na: not available; nd: not detectable.

## 1.5 Concentrations of EFRs in biota and humans

There is very little information on the occurrence of EFRs in biota and only a few EFRs such as BTBPE and DBDPE have been reported in biotic samples. Current information about concentrations of EFRs in biota are summarised in Table 1.4. BTBPE and DBDPE were detected in a variety of biota samples including meat, fish, shellfish, offal, eggs and birds (Fernandes et al., 2010; Gauthier et al., 2008; Guerra et al., 2012; Labunska et al., 2015; Law et al., 2006a; Li et al., 2015; Munschy et al., 2011; Shi et al., 2009). A selection of UK and Irish food samples were analysed for HBB, DBDPE and BTBPE. HBB and DBDPE were not detected in any foods, whilst BTBPE occurred in some UK food samples such as meat (0.05 - 1.76 ng/g lw), fish (0.26 - 3.33 ng/g lw), eggs (0.29 ng/g lw) and offal (0.75 - 0.81 ng/g lw) (Fernandes et al., 2010). The highest BTBPE concentrations were found in chicken eggs (37.2-264 ng/g lw) from an e-waste area in South China (Zheng et al., 2012), with DBDPE also found at high levels in chicken eggs, birds' offal, muscle and eggs from an e-waste area and the Pearl River Delta, South China (Shi et al., 2009; Sun et al., 2012, 2014; Zheng et al., 2012). Meanwhile, in food samples collected from e-waste impacted locations in Eastern China, EH-TBB was the dominant compound (Labunska et al., 2015). Interestingly, the levels of EH-TBB and BEH-TEBP in avian livers exceeded those in corresponding muscle samples in the same study (Labunska et al., 2015). However, in Sweden, EFRs were only detected in fish samples in a market basket study, with DBE-DBCH the predominant compound in these fish samples (Sahlström et al., 2015).

## Table 1.4 Summary of concentrations of EFRs in biota (ng/g lw) (range of values in parentheses)

| Samples | BTBPE | DBDPE | EH-TBB | BEH-TEBP | DBE-DBCH | HBB | PBEB | Location | References |
|---|---|---|---|---|---|---|---|---|---|
| **Meat** | | | | | | | | | |
| Beef | 0.56 | nd | | | | nd | | UK | (Fernandes et al., 2010) |
| Pork | 0.55 | nd | | | | nd | | | |
| Lamb | 0.05 | nd | | | | nd | | | |
| Turkey | 1.76 | nd | | | | nd | | | |
| Chicken | nd | nd | 2.66 | 1.78 | | nd | nd | Shanghai and Nanjing City, China [a] | (Labunska et al., 2015) |
| Duck | 1.87 | nd | 2.74 | nd | | nd | 1.16 | | |
| Pork | 2.69 | nd | 2.14 | 1.37 | | nd | nd | | |
| Chicken | 1.46 | nd | 24.7 | 8.97 | | 0.41 | nd | E-waste area, South China [a] | (Labunska et al., 2015) |
| Duck | 4.57 | nd | 24.2 | 7.23 | | 0.56 | 0.87 | | |
| Pork | 5.4 | nd | 38.2 | 12.4 | | 0.41 | nd | | |
| Meat [c] | | 100 | | | | 0.8 | 0.1 | Shandong Province, North China [a] | (Li et al., 2015) |
| **Fish and shellfish** | | | | | | | | | |
| Salmon | 0.26 | nd | | | | nd | | UK | (Fernandes et al., 2010) |
| Mackerel | 0.3 | nd | | | | nd | | | |
| Herring | 0.25 | nd | | | | nd | | | |
| Haddock | 0.83 | nd | | | | nd | | | |
| Lemon Sole | 3.33 | nd | | | | nd | | | |
| Whitebait | 0.77 | nd | | | | nd | | | |
| Fish | 2.1 | nd | 4 | 1.9 | | nd | nd | Shanghai and Nanjing City, China [a] | (Labunska et al., 2015) |
| Shrimp | nd | 9.19 | 11.7 | 9.32 | | 0.97 | nd | | |
| Fish | 1.46 | nd | 24.7 | 8.97 | | 0.41 | nd | E-waste area, South China [a] | (Labunska et al., 2015) |
| Shrimp | 5.4 | nd | 38.2 | 12.4 | | 0.41 | nd | | |
| Fish | nd-3.72 | nd-3.30 | | | | | | Lake Winnipeg [a] | (Law et al., 2006a) |
| Mussel | 1.29 (0.83-2.28) | nd | | | | | | | |
| Zooplankton | 0.37 (nd-0.69) | nd | | | | | | | |

|  |  |  |  |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|
| Fish [c] | 1.1-3.6[b] |  | Nd | nd | 114 | 23 | 8.7 | Sweden | (Sahlström et al., 2015) |
| Juvenile common sole | 0.08-0.31 | 0.28-1.13 |  |  |  | 0.5-1.4 |  | Nursery zones situated along the French coast [a] | (Munschy et al., 2011) |
| Fish | nd-0.15 | nd |  |  |  |  |  | E-waste area, South China | (Shi et al., 2009) |
| Fish |  | nd-230 |  |  |  |  |  | Dongjiang River, South China | (He et al., 2012) |
| Fish and seafood |  | 121 |  |  |  | 3.2 | nd | Shandong Province, North China [a] | (Li et al., 2015) |
| **Eggs** |  |  |  |  |  |  |  |  |  |
| Free range organic eggs | 0.29 | nd |  |  |  | nd |  | UK | (Fernandes et al., 2010) |
| Chicken eggs | 37.2-264 | 5.97-37.9 |  |  |  | 7.32-25.7 | 0.63-0.77 | E-waste area, South China | (Zheng et al., 2012) |
| Chicken eggs | nd | nd | 1.73 | nd |  | nd | nd | Shanghai and Nanjing City, China [a] | (Labunska et al.. 2015) |
| Chicken eggs | 2.93 | nd | 4.80 | 1.16 |  | nd | nd | E-waste area, South China [a] | (Labunska et al.. 2015) |
| Duck eggs | nd | nd | 1.21 | nd |  | nd | nd | Shanghai and Nanjing City, China [a] | (Labunska et al.. 2015) |
| Duck eggs | 2.11 | nd | 4.03 | 1.11 |  | nd | nd | E-waste area, South China [a] | (Labunska et al.. 2015) |
| **Offal** |  |  |  |  |  |  |  |  |  |
| Pork liver | 0.81 | nd |  |  |  | nd |  | UK | (Fernandes et al., 2010) |
| Chicken liver | 0.75 | nd |  |  |  | nd |  |  |  |
| Chicken liver | 3.38 | nd | 5 | 2.61 |  | nd | 2.66 | Shanghai and Nanjing City, China [a] | (Labunska et al.. 2015) |
| Duck liver | 3.27 | nd | 8.2 | 1.69 |  | nd | nd |  |  |
| Chicken liver | 15 | nd | 35 | 10.6 |  | nd | 2.3 | E-waste area, South China [a] | (Labunska et al.. 2015) |
| Duck liver | 11.7 | nd | 38.4 | 13.7 |  | nd | 3.37 |  |  |
| Juvenile common sole liver | 0.17-4.1 | nd-1.33 |  |  |  | 0.1-5.3 |  | Nursery zones situated along the French coast [a] | (Munschy et al., 2011) |
| Beluga blubber |  |  |  |  | 1.1-9.3 |  |  | Canadian Arctic | (Tomy et al., 2008b) |

| | | | | | | |
|---|---|---|---|---|---|---|
| Fish liver | nd-0.041 | nd | | | E-waste area, South China | (Shi et al., 2009) |
| Bird liver | 0.27-2.41 | 13.7-54.6 | | | | |
| Bird kidney | 0.12-0.89 | 24.5-124 | | | | |
| Bird muscle | 0.07-0.39 | 9.6-16.3 | | | | |
| **Birds and birds' eggs** | | | | | | |
| Peregrine eggs | 3.3-13 | nd-8.2 | nd-4.5 | | Canada (Great Lakes Basin, New Brunswick) and Central Spain | (Guerra et al, 2012) |
| terrestrial birds eggs | nd-1.7 | 6.1-609 | | | Pearl River Delta, South China | (Sun et al.,2014) |
| terrestrial birds | | nd-130 | | | Pearl River Delta, South China | (Sun et al.,2012) |
| Herring gull eggs [c] | 60-1820 | 1300-288000 | | 40-3440 | Colonies in the five Laurentian Great Lakes | (Gauthier et al., 2009) |

a) average concentrations; b) mLOD – mLOQ; c) the units are pg/g ww.

Very few studies have been conducted to investigate EFRs in human matrices. In Sahlström et al.'s study (2015) of two Swedish pooled human milk samples, only BTBPE, α-DBE-DBCH and β-DBE-DBCH were found with mean concentrations at 1.0-3.4, 4.0 and 0.50-1.7 pg/g ww, respectively. EFRs were also detected in 20 matched serum samples from Swedish first-time mothers and their toddlers. Specifically, α-DBE-DBCH and β-DBE-DBCH were found in serum from one toddler (1.8 and 1.3 ng/g lw, respectively), α-DBE-DBCH in one maternal serum sample (0.7 ng/g lw), with *syn*-DDC-CO and *anti*-DDC-CO present in serum from one toddler (85 and 63 ng/g lw, respectively) and one mother (49 and 39 ng/g lw, respectively) (Sahlström et al., 2014). In Canada, Zhou et al. (2014) examined several EFRs including EH-TBB, BEH-TEBP, BTBPE, and DBDPE in paired human maternal serum (n = 102) and breast milk (n = 105) samples collected in 2008-2009 in the Sherbrooke region. EH-TBB had detection frequencies greater than 55% in both serum and milk samples and the median concentrations in serum and milk were 1.6 and 0.41 ng/g lw, while BEH-TEBP, BTBPE, and DBDPE were less frequently detected in both human matrices (the detection frequencies in serum and milk were 16.7% and 32.4% for BEH-TEBP, 3.9% and 0.0% for BTBPE and 5.9% and 8.6% for DBDPE) (Zhou et al., 2014). Human hair samples from urban, e-waste, and rural areas in south China were collected and analysed for EFRs, with the levels of BTBPE, HBB and PBBz found to be in the order: occupationally exposed workers > residents in e-waste recycling area > residents in urban and rural areas (Zheng et al., 2011). For DBDPE, similar concentrations were observed in residents from urban (median of 17.7 ng/g) and e-waste-impacted (median of 17.8 ng/g) areas, as well as workers involved in e-waste handling (median of 24.2 ng/g). The lowest DBDPE levels in human hair were found in residents from a rural area (Zheng et al., 2011).

## 1.6 Relative importance of different exposure pathways for EFRs

Because of the resemblance between the chemical structures and physicochemical properties of some EFRs and "legacy" PBDEs such as BDE 209 and DBDPE, it can be hypothesised that the human exposure pathways to these compounds and their relative significance will be broadly similar. Owing to the current paucity of data relating to human exposure to EFRs, this section cross-refers to exposure to legacy BFRs where necessary.

Human exposure to BFRs can be divided into non-occupational and occupational exposure. It has been reported that the main pathways of non-occupational human exposure to BFRs are likely to be a combination of diet, ingestion of indoor dust and inhalation of indoor air (Abdallah et al., 2008; Roosens et al., 2009a, 2009b). The relative significance of these different exposure pathways for BFRs will vary between individuals, chemicals, and countries etc. For toddlers (1–5 yr), children (6–11 yr), teenagers (12–19 yr), and adults (≥20 yr), exposure to PBDEs via dust ingestion is considered the main pathway of human exposure in the US, followed by dietary exposure; nevertheless, diet (in the form of human milk) is the major source of PBDE exposure for breast-fed infants (<1 yr) (Johnson-Restrepo and Kannan, 2009). International variations in exposure patterns are illustrated by the fact that in Germany (Fromme et al., 2009), and Belgium (Roosens et al., 2009b) the intake of ΣPBDEs in adults is influenced mainly by diet; whereas in the US (Johnson-Restrepo and Kannan, 2009; Lorber, 2008) and Canada (Jones-Otazo et al., 2005) ingestion of house dust is the major pathway of human exposure to ΣPBDEs in adults; moreover, in the UK, both diet and dust make substantial contributions to human exposure to PBDEs (Harrad et al., 2006). For UK individuals, dust ingestion makes an important - sometimes an overwhelming - contribution to exposure to BDE 209 as a result of the high levels of BDE 209 in UK dust that are substantially higher than in Canada and the US (Harrad et al., 2008b). In contrast, persistent and bioaccumulative BFRs, such as BDE 47, make their way to human populations mainly via food intake; while human exposure to non-persistent BFRs, such as TBBP-A, and other phenolic BFRs, occurs primarily via inhalation (Sjödin et al., 2003). However, Abdallah et al. (2008) reported that dietary intake and dust ingestion both constitute important pathways of exposure to TBBP-A and inhalation appears a minor exposure pathway to TBBP-A. For occupationally-exposed workers, ingestion and inhalation of dust particles and vapour are thought to constitute major exposure pathways (Covaci et al., 2011).

### 1.6.1 Dietary exposure

Table 1.5 summarises the dietary intake of BFRs and its relative contribution to overall exposure in different countries. Of note, is the fact that in some countries, dietary intake is deemed an important exposure pathway to PBDEs, TBBP-A and HBCDDs for adults (Abdallah et al., 2008; Fromme et al., 2009; Harrad et al., 2004; Jones-Otazo et al., 2005; Roosens et al., 2009b; Sahlström et al., 2015).

Harrad et al. (2006) reported the median value of dietary exposure to PBDEs (sum of BDE 28, 47, 49, 66, 85, 99, 100, 153 and 154) to be 90.5 ng/person/d in UK, contributing 96.8% of overall exposure, which is in line with estimates for Germany (Fromme et al., 2009), Spain (Domingo et al., 2008), the US (Johnson-Restrepo and Kannan, 2009) and Canada (Jones-Otazo et al., 2005), but rather higher than that reported for Belgium (Roosens et al., 2009b). In Germany, dietary intakes of PBDEs (sum of BDE 47, 99, 100, 153, 154, 183) were 107.9 ng/person/d (1.42 ng/kg bw/d, assuming a body weight of 76 kg) and 58.1 ng/person/d (0.88 ng/kg bw/d, assuming a body weight of 66 kg) for male and female population, separately, responsible for 97% (average intake) of the total intake for an adult population (Fromme et al., 2009). The dietary intake of PBDEs for Spanish male adults was reported to be 75.4 ng/person/d (1.1 ng/kg bw/d, assuming a body weight of 70 kg) (Domingo et al. 2008). Dietary intakes for adults of Σtri-hepta BDEs (sum of BDE 28, 47, 99, 100, 153, 154, and 183) in Belgium were 5.9-22.0 ng/person/d (median 10.3), accounting for 96% of total exposure (Roosens et al. 2009b). The median daily dietary intakes of PBDEs for mothers and toddlers in Sweden were 20.88 and 9.96 ng/person/d, respectively, contributing 65.6% and 34.9% of overall exposure, respectively (Sahlström et al., 2015). The dietary intake of PBDEs by US and Canadian adults has been reported to be 1.1 ng/kg bw/d and 49.7 ng/person/d, respectively (Johnson-Restrepo and Kannan 2009; Jones-Otazo et al, 2005). Even though this intake in the US is consistent with the intakes found in other countries and the dietary intake in Canada is half of that in the UK; dietary intake was not the major source of PBDEs exposure for the American population and the contribution of dietary exposure in Canada is only one third of that in the UK (Johnson-Restrepo and Kannan 2009; Jones-Otazo et al, 2005; Harrad et al. 2004, 2006). This is because of the significantly higher PBDE levels found in US and Canadian indoor dust compared to elsewhere (Lorber, 2008; Jones-Otazo et al, 2005). PBDEs enter infants primarily via human milk intake; Johnson-Restrepo and Kannan (2009) reported the average estimated daily intake of PBDEs was highest for breastfed infants (86.4 ng kg/bw/d), and originated primarily (91%) from the consumption of breast milk. Jones-Otazo et al. (2005) also found the main exposure pathway for infants was via human milk (1800 ng/person/d), accounting for 91.7% of exposure. Average dietary intakes of HBCDDs and TBBP-A for UK adults were 413.0 and 2.8 ng/person/d, contributing 5.2% and 59.6% of the overall human exposure, respectively (Abdallah et al. 2008).

The median daily dietary intakes of EFRs for mothers and toddlers determined in one Swedish market basket study were 6.83 and 3.86 ng/person/d, respectively, which are lower than for

## Table 1.5 Summary of dietary intakes of BFRs in different countries and their contributions to overall human exposure to some BFRs

| Country, Population group and BFR | Method | Dietary intake (ng/person/d) | | Contribution to overall exposure (%) | | References |
|---|---|---|---|---|---|---|
| **UK** | | | | | | |
| PBDEs, adults [a] | Duplicate diet study | 90.5 | | 96.8 | | (Harrad et al. 2006) |
| PBDEs, toddlers [a] | | 51.6 | | 80.8 | | |
| HBCDD, adults | Single food product samples | 413.0 | | 75.2 | | (UK Food Standards Agency, 2006) |
| TBBP-A, adults | | 2.8 | | 59.6 | | (de Winter-Sorkina,2003) |
| **Germany** | | | | | | |
| PBDEs, female [b] | Duplicate diet study | 107.9 | | 97.0 | | (Fromme et al., 2009) |
| PBDEs, male [b] | | 58.1 | | 97.0 | | |
| **Belgium** | | | | | | |
| PBDEs, adults [c] | Duplicate diet study | 10.3 | | 96.0 | | (Roosens et al., 2009b) |
| **Spanish** | | | | | | |
| PBDEs, adults [d] | Market basket study | 75.4 | | - | | (Domingo et al., 2008). |
| **US** | | | | | | |
| PBDEs, adults [e] | Market basket study | 71.5 | | 56.0 | | (Johnson-Restrepo and Kannan, 2009) |
| PBDEs, teenagers [e] | | 67.6 | | 38.0 | | |
| PBDEs, children [e] | | 55.7 | | 36.9 | | |
| PBDEs, toddlers [e] | | 44.2 | | 20.8 | | |
| PBDEs, infants [e] | | 432.0 | | 91.0 | | |
| PBDEs, adults | - | 91 | | 16.9 | | (Lorber, 2008) |
| **Canada** | | | | | | |
| PBDEs, average adults [f] | Market basket study | Meat, dairy,eggs | 24.7 | 15.9 | 32.1 | (Jones-Otazo et al. 2005) |
| | | Fish and shellfish | 5.6 | 3.6 | | |
| | | Plant products | 15.8 | 10.2 | | |
| | | Fats, nuts, oils | 2.9 | 1.9 | | |
| | | Sugars and sweets | 0.7 | 0.5 | | |
| PBDEs, elevated indoor sources [f] | | Meat, dairy,eggs | 24.7 | 1.1 | 1.7 | |
| | | Fish and shellfish | 5.6 | 0.3 | | |

|  |  |  |  |  |  |  |
|---|---|---|---|---|---|---|
|  |  | Plant products | 15.8 | 0.2 |  |  |
|  |  | Fats, nuts, oils | 2.9 | 0.1 |  |  |
|  |  | Sugars and sweets | 0.7 | ~0 |  |  |
| PBDEs, fish eater [f] |  | Meat, dairy,eggs | 24.7 | 10.9 | 53.4 |  |
|  |  | Fish and shellfish | 76.9 | 33.9 |  |  |
|  |  | Plant products | 15.8 | 7.0 |  |  |
|  |  | Fats, nuts, oils | 2.9 | 1.3 |  |  |
|  |  | Sugars and sweets | 0.7 | 0.3 |  |  |
| PBDEs, occupational [f] |  | Meat, dairy,eggs | 24.7 | 8.8 | 17.6 |  |
|  |  | Fish and shellfish | 5.6 | 2.0 |  |  |
|  |  | Plant products | 15.8 | 5.6 |  |  |
|  |  | Fats, nuts, oils | 2.9 | 1.0 |  |  |
|  |  | Sugars and sweets | 0.7 | 0.2 |  |  |
| PBDEs, infants [f] |  | 1800 |  | 91.7 |  |  |
| **Sweden** |  |  |  |  |  |  |
| HBCDDs, Mothers | Market basket study | 11.0 |  | 76.4 [h] |  | (Sahlström et al., 2015) |
| HBCDDs, Toddlers |  | 5.0 |  | 42.0 [h] |  |  |
| PBDEs, Mothers [g] |  | 20.88 |  | 65.6 [h] |  |  |
| PBDEs, Toddlers [g] |  | 9.96 |  | 30.9 [h] |  |  |
| EFRs, Mothers [g] |  | 6.83 |  | 47.7 [h] |  |  |
| EFRs, Toddlers [g] |  | 3.26 |  | 17.7 [h] |  |  |

a) ΣPBDE: sum of PBDE 28, 47, 49, 66, 85, 99, 100, 153 and 154; b) ΣPBDE: sum of PBDE 47, 99, 100, 153, 154, and 183; c) ΣPBDE: sum of PBDE 28, 47, 99, 100, 153, 154, and 183; d) ΣPBDE: sum of PBDE 47, 99, 100, 153, 154, and 183; e) ΣPBDE: sum of PBDE 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209; f) Fatty foods: sum of PBDE15, 17, 28, 47, 66, 71, 75, 77, 85, 99, 100, 119, 126, 138, 153, 154, 183, and 190; non-fatty foods (fruit, vegetables, and grain): arithmetic mean concentrations of BDE 47, 99, 100, 153, and 154 and BDE 15, 17, 28, 47, 66, 71, 75, 77, 85, 99, 100, 119, 126, 138, 153, 154, 183 and 190; whole milk: sum of PBDE 28, 47, 99, 100, 153, 154, and 183; g) Dietary intake calculated based on fish consumption only; PBDEs: sum of PBDE28, 47, 99, 100, 153, 196, 197, 203, 206, 207, 208 and 209; EFRs: sum of α-DBE-DBCH and β-DBE-DBCH, TBX, BATE, PBBz, TBCT, PBT, PBEB, TBP-DBPE, DBDPE, BTBPE, EH-TBB and BEH-TEBP; h) overall exposure only includes dust ingestion and dietary intake.

PBDEs and HBCDDs (Sahlström et al., 2015). The DBDPE intake for infants consuming formula, cereal, and puree was quantified in one study in 2013 (Liu et al., 2014). Between the age of 1 and 12 months the daily median intake varied from 3.1 to 5.3 ng/day in the US and 2.2 to 3.4 ng/day in China. No other data on dietary exposure to EFRs have been reported so far.

### 1.6.2 Exposure via dust ingestion

Dust ingestion plays a key role in toddler (1–5 yr) exposure to PBDEs and has been hypothesised to account for higher exposure of toddlers than adults as a result of markedly greater dust ingestion by toddlers arising from frequent hand-to-mouth contact behaviour and crawling (Lorber, 2008). For EFRs, as for PBDEs, toddlers were estimated to be more exposed than adults (Ali et al., 2011a; Ali et al., 2012a, 2012b; Cequier et al., 2014; Qi et al., 2014; Sahlström et al., 2015).

Table 1.6 summarises estimates of human exposure to BFRs via dust ingestion in different countries and its relative contribution to overall human exposure.

**Table 1.6 Summary of estimates of human exposure to selected BFRs via dust ingestion in different countries and their contributions to overall human exposure**

| Population and compounds | High dust ingestion [a] (ng/person/d) | Mean dust ingestion [b] (ng/person/d) | Contribution to overall exposure (%) (mean dust ingestion) | References |
|---|---|---|---|---|
| **UK** | | | | |
| PBDEs, adults [c] | 21.5 | 0.9 | 1.0 | (Harrad et al., 2006) |
| PBDEs, toddlers [c] | 43.1 | 11.8 | 18.6 | |
| **Germany** | | | | |
| PBDEs, adults [d] | 7.7 | 2.0 | 2.7 | (Fromme et al., 2009) |
| **Belgium** | | | | |
| PBDEs, adults [e] | 0.31 | 0.76 | 4.0 | (Roosens et al., 2009b) |
| **US** | | | | |
| PBDEs, adults [f] | N/A | 84.5 | 45.0 | (Johnson-Restrepo and Kannan, 2009) |
| PBDEs, teenagers [f] | N/A | 83.2 | 46.8 | |
| PBDEs, children [f] | N/A | 75.4 | 49.9 | |
| PBDEs, toddlers [f] | N/A | 152 | 71.7 | |
| PBDEs, infants [f] | N/A | 33.5 | 7.7 | |

|  |  |  |  |  |
|---|---|---|---|---|
| PBDEs, adults | 357.3 | N/A | 66.1[g] | (Lorber, 2008) |
| **Canada** |  |  |  |  |
| PBDEs, average adults [h] | N/A | 95.8 | 61.7 | (Jones-Otazo et al., 2005) |
| PBDEs, elevated indoor sources [h] | N/A | 1983 | 90.5 |  |
| PBDEs, fish eater [h] | N/A | 95.8 | 42.3 |  |
| PBDEs, occupational [h] | N/A | 215.7 | 76.5 |  |
| PBDEs, infants [h] | N/A | 162.6 | 8.3 |  |
| **Belgium, adults** |  |  |  |  |
| BTBPE | 0.7 | 0.28 | NA | (Ali et al., 2011a) |
| DBDPE | 14 | 5.6 | NA |  |
| TBB (EH-TBB) | 0.7 | 0.28 | NA |  |
| TBPH (BEH-TEBH) | 1.4 | 0.56 | NA |  |
| TBBPA-DBPE (TBBPA-BDBPE) | 5.6 | 2.24 | NA |  |
| ΣEFRs | 22.4 | 8.96 | NA |  |
| **Belgium, toddlers[n]** |  |  |  |  |
| BTBPE | 0.65 | 0.16 | NA | (Ali et al., 2011a) |
| DBDPE | 24.7 | 6.18 | NA |  |
| EH-TBB | 1.04 | 0.26 | NA |  |
| BEH-TEBP | 5.2 | 1.3 | NA |  |
| TBBPA-BDBPE | 14.56 | 3.64 | NA |  |
| ΣEFRs | 46.15 | 11.51 | NA |  |
| **New Zealand, adults** |  |  |  |  |
| BTBPE | <0.7 | <0.7 | NA | (Ali et al. 2012a) |
| DBDPE | <0.7 | 0.7 | NA |  |
| EH-TBB | <0.7 | <0.7 | NA |  |
| BEH-TEBP | <0.7 | 0.7 | NA |  |
| ΣEFRs | NA | 0.14 | NA |  |
| **New Zealand, toddlers** |  |  |  |  |
| BTBPE | <0.7 | 0.7 | NA | (Ali et al. 2012a) |
| DBDPE | 0.48 | 1.92 | NA |  |
| TBB (EH-TBB) | 0.12 | 0.48 | NA |  |
| TBPH (BEH-TEBH) | 0.60 | 1.28 | NA |  |
| ΣEFRs | 1.2 | 4.38 | NA |  |
| **Pakistan, adults** |  |  |  |  |
| ΣEFRs [k] | 0.7 | 1.4 | NA | (Ali et al., 2012b) |
| **Pakistan, toddlers** |  |  |  |  |
| ΣEFRs [k] | 1.32 | 5.4 | NA | (Ali et al., 2012b) |
| **Norway, Woman (ng/kg bw/day)** |  |  |  |  |
| ΣEFRs [l,o] | 0.45 |  |  | (Cequier et al., 2014) |
| **Norway, Children (ng/kg bw/day)** |  |  |  |  |
| ΣEFRs [l,o] | 1.71 |  |  | (Cequier et al., 2014) |
| **Sweden, Adults [p]** |  |  |  |  |
| ΣEFRs [m] | 7.48 |  |  | (Sahlström et al., 2015) |
| PBDEs | 10.9 |  |  |  |
| HBCDDs | 3.4 |  |  |  |
| **Sweden, Toddlers [p]** |  |  |  |  |
| ΣEFRs [m] | 15.16 |  |  | (Sahlström et al., 2015) |
| PBDEs | 22.3 |  |  |  |
| HBCDDs | 6.9 |  |  |  |
| **China, infants [j]** |  |  |  | (Qi et al., 2014) |
| ΣEFRs [i] | 12.5 |  |  |  |

| | | |
|---|---|---|
| DBDPE [l] | 4.9 | |
| **China, Toddlers**[j] | | |
| ΣEFRs [l] | 115.2 | |
| DBDPE [l] | 44.8 | |
| **China, Children**[j] | | |
| ΣNBFRs [l] | 28.71 | |
| DBDPE [l] | 11.02 | |
| **China, Teenagers**[j] | | |
| ΣEFRs [l] | 31.72 | |
| DBDPE [l] | 12.48 | |
| **China, Adults**[j] | | |
| ΣEFRs [l] | 31.85 | |
| DBDPE [l] | 12.35 | |

a) Mean dust ingestion rate for adults = 20 mg/d; for toddlers = 50 mg/d; b) High dust ingestion rate for adults = 50 mg/d; for toddlers = 200 mg/d; c) ΣPBDE: sum of PBDE 28, 47, 49, 66, 85, 99, 100, 153 and 154; b) ΣPBDE: sum of PBDE 47, 99, 100, 153, 154, and 183; d) Average intake scenario based on median concentrations and high intake scenario based on 90th percentiles as not otherwise stated; ΣPBDE: Sum of BDE 47, 99, 100, 153, 154, and 183; e) Total exposure calculated based on dust ingestion and food intake; ΣPBDE: Sum of BDE 28, 47, 99, 100, 153, 154, and 183; f) ΣPBDE: sum of PBDE 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209. g) Calculated from high dust ingestion; h) ΣPBDE: sum of PBDE 17, 28, 47, 66, 71, 85, 99, 100, 153 in the residential scenarios, and 154 and BDE 17, 28, 47, 66, 77, 85, 99, 100, 126, 153, 154, and 183 in the occupational scenarios; i) ΣEFRs: sum of PBBz, PBT, PBEB, DPTE, HBB, EH-TBB, BTBPE, BEH-TEBP, and DBDPE; j) Dust ingestion rates were 0.02, 0.1, 0.05, 0.05, 0.05 g/day for infants, toddlers, children, teenagers and adults, respectively; k) ΣEFRs: sum of EH-TBB, BTBPE, BEH-TEBP, and DBDPE; l) ΣEFRs: sum of DBE-DBCH, TBP-AE, PBB, PBT, TBP-DBPE,PBEB, HBB, EH-TBB, BTBPE, BEH-TEBP, and DBDPE, *syn*-DDC-CO, *anti*-DDC-CO; m) Calculated based on dust ingestion rate of 30 mg/day and 60 mg/day for adults and toddlers, respectively; ΣEFRs: sum of DBE-DBCH, TBCT, PBBz, BATE, TBX, PBT, TBP-DBPE, PBEB, HBB, EH-TBB, BTBPE, BEH-TEBP, and DBDPE; n) the samples of home and office dust they used were from Belgium, but the samples of classroom dust were from the UK; o) Calculated based on dust ingestion rate of 50 mg/day and 100 mg/day for adults and toddlers, respectively; p) Calculated based on dust ingestion rate of 30 mg/day and 60 mg/day for adults and toddlers, respectively.

### 1.6.3 Exposure via dermal absorption of BFRs in dust

There are few results reporting human exposure to BFRs, especially for EFRs via dermal absorption from dust. One study suggests that only a small fraction (3%) of the dust is absorbed through the skin (Lorber, 2008). But taking into account the high concentration detected in dust, exposure via dermal absorption from dust may be an important pathway of human exposure – in addition to dermal uptake via direct contact with materials containing BFRs. Abdallah et al. (2015a) investigated human dermal absorption of eight mono- to deca-brominated diphenyl ethers (PBDEs) using EPISKIN human skin equivalent tissue and found the proportion of the applied PBDE dose that accumulated in the skin increased with increasing bromine substitution from BDE 1 (~18%) to BDE 153 (~37%), this proportion dropped steeply from BDE 183 (~13%) to BDE 209 (8%). Additionally, the percutaneous penetration of HBCDD and TBBP-A through two commercially available 3D-human skin equivalents (3D-HSE) models was studied by Abdallah et al. (2015b), and the absorbed dose was found to be low (less than 7%) and was

**Table 1.7 Summary of exposure via dermal absorption of BFRs in different countries and its contribution to overall human exposure**

| Population and compounds | Dermal absorption (ng/person/d) | Contribution to overall exposure (%) (mean dust ingestion $^{e}$) | References |
|---|---|---|---|
| **USA** | | | |
| PBDEs, adults (≥20 yr) $^{a}$ | 22.1 | 11.8 | (Johnson-Restrepo and Kannan, 2009) |
| PBDEs, teenagers (12–19 yr) $^{a}$ | 17.7 | 9.9 | |
| PBDEs, children (6–11 yr) $^{a}$ | 13.3 | 8.8 | |
| PBDEs, toddlers (1–5 yr) $^{a}$ | 11.2 | 5.3 | |
| PBDEs, infants (<1 yr) $^{a}$ | 3.85 | 0.9 | |
| PBDEs, adults $^{b}$ | 85.9 | 16 | (Lorber, 2008) |
| **China** | | | |
| PBDEs, infants (3-12months) $^{c}$ | 0.35 | N/A | (Chen et al., 2009) |
| PBDEs, toddlers (1-3 yr) $^{c}$ | 0.46 | N/A | |
| PBDEs, children (3-5 yr) $^{c}$ | 0.74 | N/A | |
| PBDEs, children (5-9 yr) $^{c}$ | 0.81 | N/A | |
| PBDEs, teenagers (9-14 yr) $^{c}$ | 1.35 | N/A | |
| **Norway, children**(ng/kg bw/day) | | | |
| ΣEFRs $^{d}$ | 0.25 | | (Cequier et al., 2014) |
| PBDEs | 0.14 | | |
| **Norway, women** (ng/kg bw/day) | | | |
| ΣEFRs $^{d}$ | 0.109 | | (Cequier et al., 2014) |
| PBDEs | 0.091 | | |

a) ΣPBDE: sum of PBDE 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209; b) ΣPBDE: sum of PBDE 28, 47, 99, 100, 138, 153, 154, 183, and 209; c) ΣPBDE: sum of PBDE 28, 47, 66, 99, 100, 138, 153, 154, 183, 196, 197, 203, 206, 207, 208 and 209. d) EFRs: sum of PBBz, PBT, PBEB, DPTE, HBB, EH-TBB, BTBPE, BEH-TEBP, and DBDPE; e) In this scenario, the human exposure via dust was calculated by mean dust ingestion rates which are 20 mg/d for adults and 50 mg/d for toddlers, respectively.

significantly correlated with log $K_{OW}$ of the tested BFR. Table 1.7 summarises estimates of dermal absorption exposures to BFRs from different countries and its relevant contribution to overall human exposure. For the USA, Johnson-Restrepo and Kannan (2009) calculated the daily exposure doses for PBDEs from dermal absorption for infants (<1 yr), toddlers (1–5 yr), children (6–11 yr), teenagers (12–19 yr), and adults (≥20 yr) to be 0.77, 0.7, 0.46, 0.34 and 0.34 ng/kg bw/d, respectively. These estimates are lower than those from diet or dust ingestion but comparable to those from air inhalation. The estimated dermal absorption dose of PBDEs for adults in Johnson-Restrepo and Kannan's (2009) study was approximately 4-fold lower than the estimate of Lorber (2008); this was mainly due to the differences in dust PBDE concentrations (1910 ng/g dw in study versus 8154 ng/g dw in Lorber, 2008) (Johnson-Restrepo and Kannan 2009; Lorber 2008). In China, Chen et al. (2009) estimated that daily exposures to PBDEs for children from 3 months to 14 years via dermal contact with toys varied from 30.5 to 43.3 pg/kg bw/d. This is approximately one order of magnitude lower than the exposure from dermal absorption of dust or air inhalation and three orders of magnitude lower than the exposure from human milk (78.6 ng/kg bw/d) for infants in the USA (Johnson-Restrepo and Kannan 2009). However, it should be noted that the BFR concentrations in toys are much lower than in flame-retarded materials. Qi et al. (2014) estimated dermal exposure to ΣEFRs via this pathway to be 0.25 and 0.109 ng/kg bw/d for women and children in China, respectively, a figure comparable to that obtained for PBDEs .

### 1.6.4 Exposure via inhalation

The presence of BFRs in indoor air is a non-negligible exposure pathway for humans. Harrad et al. (2004, 2006) estimated that the median human intake of ∑PBDE via inhalation was 2.1 ng/person (contributing 2.3% of overall exposure) using the median concentrations of ∑PBDE detected in air from outdoor, workplace, and domestic environments and found a significant positive correlation ($p < 0.001$) between PBDE concentrations and both the number of electrical appliances and polyurethane foam-containing chairs (Harrad et al. 2004). HBCDDs and TBBP-A were also found in indoor air from UK homes (n = 33; median concentrations ΣHBCDDs =180 pg/m$^3$; TBBP-A = 15 pg/m$^3$), offices (n = 25; 170; 11), public microenvironments (n = 4; 900; 27) and outdoor air (n= 5; 37; 1) (Abdallah et al. 2008). Table 1.7 summarises exposure via air inhalation of BFRs in different countries and its relative contribution to overall human exposure. Compared to dietary intake and dust ingestion, inhalation is a minor exposure pathway for PBDEs, HBCDDs and TBBP-A in the UK, the USA, Germany, and Canada (Harrad et al. 2006; Abdallah et al. 2008; Fromme et al. 2009; Johnson-Restrepo and Kannan 2009; Abdallah et al. 2008). Similar to “legacy” FRs, Cequier et al. (2014) reported human exposure to EFRs via air inhalation which is 1-2 orders of magnitude lower than the human exposure to EFRs via dust ingestion (Cequier et al., 2014), while for the more volatile compounds (e.g., DBE-DBCH), the main source of exposure was air (~80%) (Cequier et al., 2014).

### 1.6.5 Occupational exposure

High levels of some EFRs were found in air and dust samples near EFR manufacturing sites or e-waste processing areas and the workers may be subject to high occupational exposures through air inhalation, dust ingestion and dermal absorption. In one study, the concentrations of BTBPE and DBDPE were 14.6-232 ng/g dw (mean: 107 ng/g dw) and <2.50-139 ng/g dw (mean: 107 ng/g dw) respectively in dust collected from an e-waste area in China; while TBBPA-BDBPE was not detectable (Shi et al. 2009). High BTBPE levels of 70 and 19 pg/m$^3$ were found in two outdoor air samples collected near a BTBPE producing facility in the US (150 km east and 480 km southeast of the facility) (Hoh et al. 2005). BTBPE was also found in indoor air at electronics

### Table 1.8 Summary of exposure to BFRs via inhalation in different countries and its contribution to overall human exposure

| Population and compounds | Air inhalation (ng/person/d) | Contribution to overall exposure (%) | References |
|---|---|---|---|
| **UK** | | | |
| PBDEs, adults [a] | 2.1 | 2.3 | Harrad et al. (2006) |
| PBDEs, toddlers (0.5-2 yr) [a] | 0.7 | 0.6 | |
| HBCDDs, adults | 5.0 | 0.9 | Abdallah et al. (2008) |
| HBCDDs, toddlers (0.5-2 yr) | 1.0 | 0.2 | |
| TBBP-A, adults | 0.3 | 6.4 | |
| TBBP-A, toddlers (0.5-2 yr) | 0.1 | 2.0 | |
| **Germany** | | | |
| PBDEs, adults [b] | 0.17 | 0.24 | Fromme et al. (2009) |
| **USA** | | | |
| PBDEs, adults (≥20 yr) [c] | 9.1 | 4.8 | Johnson-Restrepo and Kannan (2009) |
| PBDEs, teenagers (12–19 yr) [c] | 9.36 | 5.3 | |
| PBDEs, children (6–11 yr) [c] | 6.67 | 4.4 | |
| PBDEs, toddlers (1–5 yr) [c] | 4.64 | 2.2 | |
| PBDEs, infants (<1 yr) [c] | 3.0 | 0.7 | |
| **Canada** | | | |
| PBDEs, average adults [d] | 9.7 | 6.2 | |
| PBDEs, elevated indoor sources [d] | 158.7 | 7.2 | |
| PBDEs, fish eater [d] | 9.7 | 4.3 | |
| PBDEs, occupational [d] | 16.3 | 5.8 | |
| PBDEs, infants [d] | 0.8 | ~0 | |
| **Norway, children**(pg/kg bw/day) | | | |
| ΣEFRs [e] | 27 | | (Cequier et al., 2014) |
| PBDEs | 55 | | |
| **Norway, women** (pg/kg bw/day) | | | |
| ΣEFRs [e] | 12 | | (Cequier et al., 2014) |
| PBDEs | 19 | | |

a) ΣPBDE: sum of PBDE 28, 47, 49, 66, 85, 99, 100, 153 and 154; b) ΣPBDE: Sum of BDE 47, 99, 100, 153, 154, and 183; c) ΣPBDE: sum of PBDE 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209; d) ΣPBDE: sum of PBDE 17, 28, 47, 66, 71, 85, 99, 100, 153 in the residential scenarios, and 154 and BDE 17, 28, 47, 66, 77, 85, 99, 100, 126, 153, 154, and 183 in the occupational scenarios; e) ΣEFRs: sum of PBBz, PBT, PBEB, DPTE, HBB, EH-TBB, BTBPE, BEH-TEBP, and DBDPE.

recycling plants, with the mean BTBPE levels being 20,000 and 23 000-32 000 pg/m$^3$ in the air of dismantling hall and shredder, separately. Such high concentrations of EFRs in air and dust samples illustrate the potential for high occupational exposure to these chemicals for workers in such facilities (Sjödin et al., 2001). By comparison, indoor air in an office containing computers only, contained BTBPE at 5.8 pg/m$^3$, ca. 4000 times lower than in recycling plants. The much higher levels observed in such recycling plants are hypothesised to occur because bromine containing additives to plastic materials are emitted to the indoor work environment from particulate matter generated via the dismantling and shredding of BFR-containing plastics (Sjödin et al., 2001).

## 1.7 Objectives of this study

EFRs are of environmental and human health concern due to their potential toxicity, both in humans and animals. Current understanding of the potential environmental impacts of EFRs reveals several research gaps with respect to our knowledge of their environmental presence, human exposure and body burdens. The overriding hypothesis of the work reported in this thesis is that human exposure to EFRs is substantial, and is increasing following restrictions on the use of "legacy" FRs. To test this hypothesis and address some of the research gaps, the aims of the current study are to:

1- Develop and validate analytical methodology for determination of EFRs in a range of matrices relevant to human exposure.

2- Determine concentrations of EFRs in foodstuffs from a location impacted by rudimentary e-waste processing in Bui Dau, Vietnam and estimate human exposure to EFRs via dietary intake in this area.

3- Characterise concentrations of EFRs and "legacy" FRs in indoor air and dust in the UK.

4- Elucidate the time trends in concentrations of EFRs and "legacy" FRs in indoor air and dust samples which may reflect changes in production and use of such compounds.

5- Estimate human exposure to EFRs and “legacy” FRs via air inhalation, dust ingestion and dietary intake in the UK, assess the relative importance of each exposure route to the overall exposure of adults and toddlers using different exposure scenarios.

6- Determine concentrations of EFRs and “legacy” FRs in human milk from a small number of UK mothers and study the relationship between external and internal exposure to the studied FRs using a simple, one-compartment pharmacokinetic model.

# CHAPTER 2 Sampling and Analytical Methodology

## 2.1 Synopsis

In order to test the hypotheses outlined at the end of the previous chapter, different types of samples were collected, including indoor air, indoor dust, diet, soil and human milk. A description of sampling methods and locations employed for each of these sample matrices is given in Table 2.1. Analytical methodology for determination of emerging and legacy FRs consisting of extraction, cleaning up and instrumental analysis has been developed and optimised in diverse biotic and abiotic samples. The reliability of the developed and applied analytical methods has been tested in different matrices, along with quality assurance (QA)/quality control (QC) employed to validate the data in this thesis.

**Table 2.1 Overview of sampling methods and locations for different sample types**

| Sample Types | Collection Method | Location |
|---|---|---|
| Indoor dust | Nylon sock | Birmingham, UK |
| Air | Passive PUF disk | Birmingham, UK |
| Market basket diet [a] | Market basket method from different markets | Birmingham, UK |
| Human milk | Obtained from adult healthy volunteers via Birmingham Women's Hospital Milk Bank (Abdallah and Harrad, 2014) | Birmingham, UK |
| Human milk | Supplied by Imperial College, London. Obtained from two hospitals in Southampton and London, respectively | Southampton and London, UK |

| | | |
|---|---|---|
| Soil samples | Surface soil collected at the same time from the backyards where the poultry were raised | Bui Dau (Cam Xa, Hung Yen province), Vietnam |
| Diet samples | Poultry and fish purchased from local residents and market | Bui Dau (Cam Xa, Hung Yen province), Vietnam |

a) market basket diet: this method is to collect a number of the most commonly bought food based on the actual average consumption of different categories of food (Frederiksen et al., 2009).

## 2.2 Chemicals and reagents

Solvents used were all of HPLC analytical grade and purchased from Fisher Scientific (Loughborough, UK). Standards of BDE 28, 47, 99, 100, 153, 154, 183 and 209, α-, β- DBE-DBCH, TBX, PBBz, PBT, TBP-DBPE, PBEB, HBB, BTBPE, DBDPE, EH-TBB, BEH-TEBP, and labelled internal standards (IS) $^{13}$C-BDE 209, $^{13}$C-BTBPE, $^{13}$C-BEH-TEBP and $^{13}$C-HBCDD were purchased from Wellington Laboratories (Guelph, ON, Canada). DDC-CO was purchased from Cambridge Isotope Laboratories Inc. (Tewksbury, MA, USA). TBCT was obtained from Accustandard (New Haven, CT, USA). TBBPA-BDBPE was purchased from Dr. Ehrenstorfer (Essex, UK). HBCDDs were obtained from Sigma-Aldrich Company Ltd. (Dorset, UK). BDE 77 and 128 (IS) were obtained from AccuStandard Inc. (New Haven, CT, USA). Indoor dust SRM 2585 was purchased from the US National Institute of Standards and Technology (NIST, Gaithersburg, MD, USA).

## 2.3 Sampling

### 2.3.1 Air sampling

#### 2.3.1.1 Air sampling method

Air samples were collected in offices (n=20) and houses (n=15) using passive air sampling techniques in Birmingham, UK from February to May 2015. Each passive sampler was deployed for around 45 days around a height of the human breathing zone. The passive air sampler configuration used followed the method reported by Abdallah and Harrad (2010) and comprised polyurethane foam disks (140 mm diameter, 12 mm thickness, 360.6 $cm^2$ surface area, 0.07 g $cm^{-3}$ density, PACS, Leicester, UK) and a glass fibre filter (GFF, 12.5 cm diameter, 1 μm pore size, Whatman, UK) fully sheltered by two different size stainless steel housings (Figure 2.1). PUF disks were pre-cleaned with DCM, acetone, and hexane in turn using ASE extraction and GFFs were preconditioned by heating at 450°C for 5 h. "Field blanks" comprising PUF disks and GFFs analysed as air samples were examined to check that the levels of target compounds in the disks and GFFs were acceptably low. Shelters were cleaned carefully and wrapped with hexane

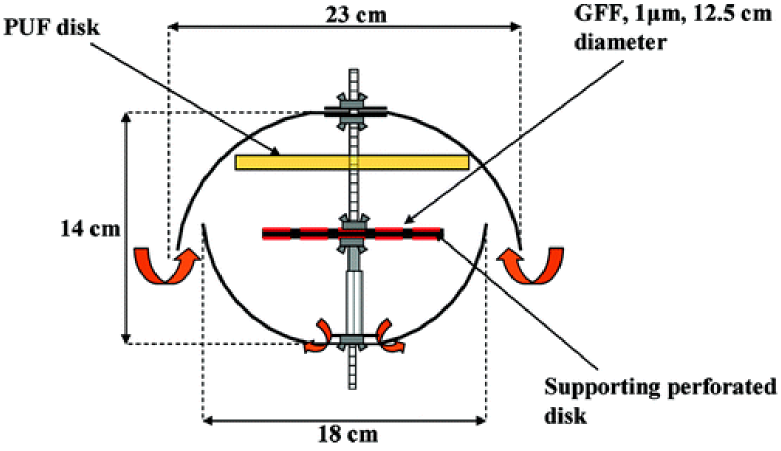


**Figure 2.1 Diagram of passive air sampler configuration**

rinsed aluminium foil and sealed in a polyethylene zip bag before and after deployment. Harvested disks were stored at -20°C until analysis.

#### 2.3.1.2 Passive air sampling rates

The sampling rate for BDE 28 (1.74 $m^3$/day) was derived from the study of Hazrati and Harrad (2007). Sampling rates ($m^3$/day) for HBCDDs and PBDEs except for BDE 28 in this study were derived from the values of sampling rates for PUF using the same passive air sampler configuration (Abdallah and Harrad, 2010). We analysed PUF disks and GFFs together, while in Abdallah's study (2010) the sampling rates were calculated separately for gas and particle phases. Therefore, in this study we calculated the sampling rates for the combination of gas and particle phases using the equation below:

$$\mathrm{S} = S_{Gas} * P_{Gas} + S_{Particle} * P_{Particle} \ldots\ldots\ldots\ldots\ldots (2.1)$$

where $S_{Gas}$ : sampling rates for gas phase from Abdallah's study (2010); $S_{Particle}$ : sampling rates for particle phase from Abdallah's study (2010); $P_{Gas}$ : the percentage of target compounds in gas phase of gas and particle phases from Abdallah's study (2010); $P_{Particle}$ : the percentage of target compounds in particle phase of gas and particle phases from Abdallah's study (2010).

Sampling rates ($m^3$/day) for HBCDDs and PBDEs except for BDE 28 in this study derived from equation 2.1 are shown in Table 2.2.

To our knowledge, no EFR-specific PAS sampling rates exist – i.e. sampling rates derived via a bespoke calibration exercise. As a result of this, in this study, sampling rates for EFRs were derived via two different methods. The first method assumed a uniform sampling rate for all EFRs targeted in this study that was the average value obtained for PBDEs in two previous

### Table 2.2 Passive sampling rates ($m^3$/day) of PBDEs and HBCDDs in indoor air [a]

| | Sampling rates (gas) | Sampling rates (particle) | Gas (pg/day) | Particle (pg/day) | Gas (%) | Particle (%) | Sampling rates ($m^3$/day) (this study) |
|---|---|---|---|---|---|---|---|
| BDE 47 | 1.509 | 0.448 | 63.8 | 2.3 | 97% | 3% | 1.472 |
| BDE 99 | 1.075 | 0.491 | 27.2 | 4.5 | 86% | 14% | 0.992 |
| BDE 100 | 1.136 | 0.494 | 4.7 | 1.1 | 81% | 19% | 1.014 |
| BDE 153 | 0.844 | 0.542 | 2.8 | 3.1 | 47% | 53% | 0.685 |
| BDE 154 | 0.807 | 0.537 | 0.4 | 0.3 | 57% | 43% | 0.691 |
| α-HBCDD | 0.852 | 0.561 | 23 | 4.1 | 85% | 15% | 0.807 |
| β-HBCDD | 0.848 | 0.567 | 6 | 2.1 | 74% | 26% | 0.775 |
| γ-HBCDD | 0.837 | 0.579 | 49.4 | 20.5 | 71% | 29% | 0.761 |

a) The sampling rates values of PBDEs and HBCDDs cited in this table and used for calculation of sampling rates in this study were from Abdallah and Harrad's study (2010).

### Table 2.3 Passive sampling rates ($m^3$/day) of the individual studied EFRs in indoor air

| **PBDEs** | **sampling rate of PBDEs in the literature[a]** | **sampling rate of PBDEs in the literature[b]** | **overall average sampling rate** |
|---|---|---|---|
| BDE 28 | - | - | |
| BDE 47 | 1.472 | - | |
| BDE 100 | 0.992 | - | |
| BDE 99 | 1.014 | - | |
| BDE 154 | 0.685 | - | |
| BDE 153 | 0.691 | - | |
| BDE 183 | 0.550 | - | |
| BDE 209 | 0.567 | - | |
| Average sampling rate | 0.853 | 2.5 | 1.677 |

a) Abdallah, M.A.-E. & Harrad, S., 2010. Modification and calibration of a passive air sampler for monitoring vapor and particulate phase brominated flame retardants in indoor air: application to car interiors. *Environmental science & technology*, 44(8), pp.3059–65; b) Wilford, B.H., Harner, T., Zhu, J., Shoeib, M., Jones, K.C., 2004. Passive Sampling Survey of Polybrominated Diphenyl Ether Flame Retardants in Indoor and Outdoor Air in Ottawa, Canada: Implications for Sources and Exposure. Environ. Sci. Technol. 38, 5312–5318.

studies (Table 2.3). The second approach is outlined in Table 2.4, whereby the sampling rates for individual EFRs were assumed equivalent to literature reported sampling rates for PBDEs of similar $K_{OA}$ and $K_{OW}$ values. To illustrate, the sampling rate for BTBPE (log $K_{OA}$ = 15.67) was assumed equivalent to that for BDE 183 (log $K_{OA}$ = 14.55 and 11.96). These approaches are broadly consistent with that used by Drage et al. (2016) to derive PAS sampling rates for EFRs for an outdoor air sampling campaign. In that earlier study, for most EFRs (those expected to be mostly in the vapour phase) the authors assumed the same rate as BDE 47 and 99 (3.92 $m^3$/day), while for DBDPE they applied the same rate as BDE 209 (2.26 $m^3$/day). The 5th percentile, 95th percentile, mean, median, minimum, maximum and geometric mean values of human exposure to EFRs via inhalation using the two different sampling rates in Table 2.3 and 2.4 were compared by independent t-test. No big differences were found between the data using these two different sampling rates for both toddlers and adults ($p > 0.05$). This suggests the effect from of the sampling rates for EFRs was minor, therefore in this study we used the sampling rates calculated from the first approach (1.677 $m^3$/day) for all EFRs.

### Table 2.4 Passive sampling rates ($m^3$/day) of the individual studied EFRs in indoor air

| EFRs | number of Br | Log $K_{OA}$ | Log $K_{OW}$ | PBDEs | number of Br | log $K_{OA}$ | Log $K_{OW}$ | sampling rate of PBDEs in the literature[j] ($m^3$/day) | | | sampling rate ($m^3$/day) (this study) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | PUF | filter | PUF and filter | |
| DBE-DBCH (TBECH) | 4 | 8.01[h] | 4.82[c], 5.24[h], 4.41[a], 5.24[b] | BDE 28 | 3 | 9.40[b] , 9.5[g] | 5.94[d], 6.47[a], 5.88[b] | | | | 1.74[k] |
| TBX | | 8.81[b] | 6.2[c],6.06[a], 6.65[b] | | | | | | | | |
| TBCT(TBoCT) | | 8.82[b] | 6.74,6.29[c] | | | | | | | | |
| TBP-DBPE(DPTE) | 5 | 8.01[b] | 5.25[b] | | | | | | | | |
| PBBz | | 9.10[b] | 6.44[b] | | | | | | | | |
| PBT | | 9.66[i] | 5.87[f],5.43[f],6.25[c], 6.42[a],6.99[b] | | | | | | | | |
| PBEB | 5 | 9.97[b] | 6.76[c], 6.78[a],7.48[b] | BDE 47 | 4 | 10.69[b] , 10.53[g] | 6.81[d], 7.16[a], 6.77[b] | 1.509 | 0.448 | 1.472 | 1.472 |
| HBB | 6 | 10.26[i] | 5.85[f],6.07[i], | | | | | | | | |
| EH-TBB (TBB) | 4 | 12.34[b] | 7.73[c], 8.28[a],8.75[b] | BDE 153 | 6 | 11.82[g] | 7.90[d], 8.73[a] | 0.844 | 0.542 | 0.691 | 0.691 |
| | | | | BDE 154 | 6 | 13.27[b] , 11.92[g] | 7.82[d], 8.75[a], 8.55[b] | 0.807 | 0.537 | 0.685 | 0.685 |
| BTBPE | 6 | 15.67[b] | 7.88[f], 8.31[c], 9.39[a], 9.15[b] | BDE 183 | 7 | 14.55[b] , 11.96[g] | 8.27[d], 9.55[a], 9.44[b] | 0.558 | 0.546 | 0.550 | 0.550 |
| DBDPE | 10 | 19.22[b] | 11.1[c],12.27[a], 13.64[b] | BDE 209 | 10 | 18.42[b] , 14.98[e] | 12.04[a], 12.11[b] | | 0.567 | 0.567 | 0.567 |
| BEH-TEBP (TBPH) | | 16.86[b] | 9.34[c], 10.92[a], 11.95[b] | | | | | | | | |
| TBBPA-BDBPE | | 20.30[b] | 12.99[c],11.52, 12.22 | | | | | | | | |
| DDC-CO(DP) | -- | 13.1[b] | 10.12[c], 11.27[h] | | | | | | | | |

a)Sparc On-Line Calculator 4.6

b)HENRYWIN v3.20 (EPIWIN 4.)

c)Bergman, Å., Rydén, A., Law, R.J., de Boer, J., Covaci, A., Alaee, M., Birnbaum, L., Petreas, M., Rose, M., Sakai, S., Van den Eede, N., van der Veen, I., 2012. A novel abbreviation standard for organobromine, organochlorine and organophosphorus flame retardants and some characteristics of the chemicals. Environ. Int. 49, 57–82.

d)Braekevelt, E., Tittlemier, S. a., Tomy, G.T., 2003. Direct measurement of octanol-water partition coefficients of some environmentally relevant brominated diphenyl ether congeners. Chemosphere 51, 563–567.

e)Cetin, B., Odabasi, M., 2008. Atmospheric concentrations and phase partitioning of polybrominated diphenyl ethers (PBDEs) in Izmir, Turkey. Chemosphere 71, 1067–1078.

f)Covaci, A., Harrad, S., Abdallah, M.A.E., Ali, N., Law, R.J., Herzke, D., de Wit, C.A., 2011. Novel brominated flame retardants: A review of their analysis, environmental fate and behaviour. Environ. Int. 37, 532–556.

g)Harner, T., Shoeib, M., 2002. Measurements of octanol-air partition coefficients (KOA) for polybrominated diphenyl ethers (PBDEs): Predicting partitioning in the environment. J. Chem. Eng. Data 47, 228–232.

h)Howard, P.H., Muir, D.C.G., 2010. Identifying new persistent and bioaccumulative organics among chemicals in commerce. Environ. Sci. Technol. 44, 2277–85.

i)Stenzel, A., Goss, K.-U., Endo, S., 2013. Determination of polyparameter linear free energy relationship (pp-LFER) substance descriptors for established and alternative flame retardants. Environ. Sci. Technol. 47, 1399–406.

j)Abdallah, M.A.-E. & Harrad, S., 2010. Modification and calibration of a passive air sampler for monitoring vapor and particulate phase brominated flame retardants in indoor air: application to car interiors. Environ. Sci. Technol. 44(8), pp.3059–65.

k)Hazrati, S., Harrad, S., 2007. Calibration of polyurethane foam (PUF) disk passive air samplers for quantitative measurement of polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs): Factors influencing sampling rates. Chemosphere 67, 448–455.

### 2.3.2 Dust sampling

Dust samples were collected in offices (n=42) and houses (n=30) using a Black and Decker 780 W mini vacuum cleaner and nylon sample socks (25 µm pore size) from June 2013 to May 2015. Both living room and bedroom dust samples were collected in 15 houses. Samples were collected under normal office and house use conditions to reflect actual human exposure. In each location, one $m^2$ of carpet was vacuumed for 2 min and 4 $m^2$ of bare floors were vacuumed for 4 min to take the dust samples. After sampling, samples were sealed in a plastic bag and stored at -20 °C. All dust samples were sieved through a pre-cleaned 500 µm mesh sieve, homogenised thoroughly, transferred to clean glass vials and then stored at -20 °C prior to analysis.

### 2.3.3 Diet sampling

In this study we only focus on meat-related food samples because FRs such as PBDEs are lipophilic and persistent organic compounds which accumulate in lipid-rich tissues (Frederiksen et al., 2009).

#### 2.3.3.1 Market basket diet sampling in Birmingham, UK

Food samples were purchased from two supermarkets representing national chains and one local indoor market in Birmingham, UK from May to June 2015. The collected foods were composited into 14 of the food groups (90 samples total) that make up our preliminary market basket study (Table 2.5). Equal weights of each of the 3 samples of each food group from each market were homogenized and combined into 30 composite samples. All food samples were homogenised, freeze dried and stored at -20 °C prior to analysis. The water content of each sample was determined gravimetrically to permit calculation of concentrations on a wet weight (ww) basis. Concentrations of FRs (ng/g ww) in each sample were multiplied by the consumption rates for various food groups to calculate an estimate of dietary intake.

Table 2.5 Food groups included in the market basket study

| Food types | Food groups | Food name | Number of samples analysed |
|---|---|---|---|
| Meat | Group 1 | beef | 3 |
| | Group 2 | chicken | 3 |
| | Group 3 | pork | 3 |
| | Group 4 | lamb | 3 |
| Fish | Group 5 | salmon | 3 |
| | Group 6 | tuna | 2 |
| | Group 7 | trout | 2 |
| | Group 8 | mackerel | 2 |
| Egg and dairy product | Group 9 | cheese | 2 |
| | Group 10 | chicken egg | 1 |
| Offal | Group 11 | chicken liver | 1 |
| | Group 12 | pork liver | 1 |
| | Group 13 | beef liver | 2 |
| | Group 14 | lamb liver | 2 |

#### 2.3.3.2 Diet sampling in Vietnam

Samples of selected human foodstuffs were collected in northern Vietnam from an e-waste processing area in Bui Dau (Cam Xa, Hung Yen province). This area is a rural location with approximately 200 households. The main supplies of livestock products and fish for the local people in Bui Dau are from neighbouring communities (Tue et al., 2010), and the livestock and fish raised in farm yards in Bui Dau are intended mainly for consumption by the families themselves with any small surpluses sold commercially. E-waste treatment activities such as: dismantling of electrical wires and metals, shredding of plastics into pellets, manual recycling of TVs, printers, printed circuit boards and other computer components started in this village at the beginning of the current decade. Most businesses are family-based and e-waste is recycled in the backyard of the house where livestock are also raised. Locations of the sampling sites are presented in Figure 2.2. Questionnaires were given to the inhabitants of the sites sampled to collect information about the location, types of the facilities, sampling site description, dietary habits and sales of the food. Fresh hens' eggs (n=18) were collected from chicken farm owners in six sampling sites (site 1- site 6) shown in Figure 2.2, with five chickens purchased from five farm owners in five locations (site 1- site 5) to obtain samples of chicken muscle (n=5), chicken liver (n=5), and chicken skin (n=5) in January 2014. One control egg sample and one control chicken muscle sample were purchased from Thanh Hoa province, which is situated approximately 175 km distant from the e-waste recycling sites. Furthermore, chicken egg, chicken muscle, chicken liver, and chicken skin were purchased as control samples in Tsukuba, Japan. River fish samples (tilapia, n=5) and pond fish samples (rohu, n=3 and tilapia, n=2) were collected from the river (site 7) and the fish pond (site 8) located close to Bui Dau, with 2 pork samples purchased from the small market in Bui Dau village. Control samples of pork and fish were purchased in Hanoi City.

Heat treatment of eggs was conducted in a large pan by boiling gently for 8 minutes at 100 ˚C. This was conducted to facilitate easy transportation of samples for analysis. Eggs were removed from the heat and cooled down to room temperature before egg yolks were separated from the egg white and wrapped in pre-cleaned aluminium foil. All samples were stored at -20 ˚C until shipping to the analytical laboratory on ice.

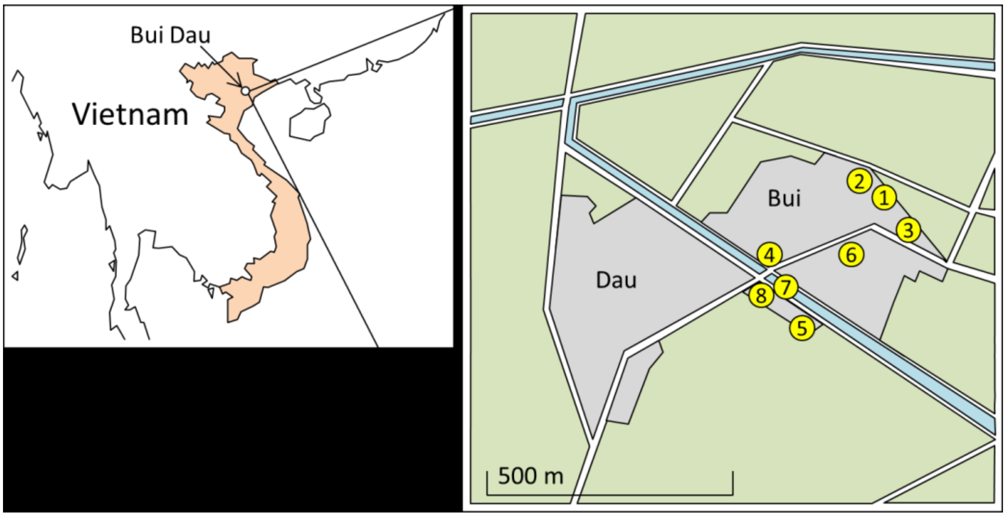


**Figure 2.2 Sampling sites in an e-waste processing region, Bui Dau, Cam Xa, Hung Yen province, Vietnam**

### 2.3.4 Soil sampling in Vietnam

Five soil samples were collected from the same backyards from which the chicken samples were collected (site 1- site 5 - Figure 2.2). Three subsamples of soil were collected from each location, homogenised, and stored at -20 ºC until shipping on ice to the analytical laboratory.

### 2.3.5 Human milk sampling

Archived human milk samples (n=25, each comprising ~50 mL) were obtained from the milk bank of Birmingham Women's Hospital in 2010 after the research proposal and experimental design were approved by a local research ethics committee (REC reference number: 9/H1211/57) according to the NHS guidelines (Abdallah and Harrad, 2014). Detailed sampling collection procedures are provided elsewhere (Abdallah and Harrad, 2014), but in summary, milk samples were transferred on ice from the milk bank in 100 mL clean polypropylene containers and freeze-dried prior to storage at -20 ºC until analysis.

Another batch of human milk samples were collected from two hospitals in Southampton and London, respectively, between August 2014 and May 2015 by researchers at Imperial College

London. After collection, samples were kept in clean screw-capped plastic containers and transferred from Imperial College London to Birmingham in special ice boxes then stored at -20 °C until the time of analysis.

### 2.3.6 Lipid determination

The lipid content in diet and human milk samples was determined gravimetrically on separate aliquots of the freeze-dried samples (typically 1g) following a standard procedure (The European Standard EN 1528-2, 1996).

## 2.4 Extraction

### 2.4.1 Comparison of extraction methods: ASE vs. Vor-Usoni

A mixture of n-hexane (Hex)/acetone (Ac) (3:1, v/v) was proven to be a reliable solvent for both EFRs and PBDEs in a previous study (Van den Eede et al., 2012) and was thus employed as the extraction solvent in this study. Two extraction methods (accelerated solvent extraction (ASE) and vortex and ultrasonication extraction (Vor-Usoni)) were assessed by performing spiking experiments on sorbent material ($Na_2SO_4$) using three concentration levels, $Q_{low}$ (50 ng of each, 75 ng DBDPE), $Q_{medium}$ (100 ng of each, 150 ng DBDPE) and $Q_{high}$ (200 ng of each, 300 ng DBDPE). Each of these spiked samples were extracted in triplicate on three separate days (n=9).

#### 2.4.1.1 ASE

An aliquot of each sample of spiked sorbent material (~100 mg) was accurately weighed. The extraction cells were filled from bottom to top with: pre-cleaned hydromatrix, spiked sample, and hydromatrix. Spiked samples were further treated with internal standards (ISs - $^{13}$C-BDE 209, $^{13}$C-BTBPE, $^{13}$C-BEH-TEBP, BDE 77, BDE 128). Extraction was performed using an ASE 350 (temperature 100 °C, pressure 1500 psi, heating time 5 min, static time 4 min, purge time 90 s, flush volume 60%, 3 static cycles, solvent Hex-Ac (3:1, v/v)).

#### 2.4.1.2 Vor-Usoni

An aliquot of each spiked sample (~100 mg) was accurately weighed into glass tubes and spiked with the same IS described in the previous section. Hex-Ac (2 mL, 3:1, v/v) was added and the tube vortexed for 2 mins, prior to ultrasonication for 5 mins. The supernatant was removed, fresh solvent introduced and the extraction process repeated three times. After extraction, spiked sample extracts were combined, centrifuged, transferred into clean glass tubes and then evaporated to dryness and redissolved in 1 mL hexane.

#### 2.4.1.3 Extraction efficiency comparison results: ASE vs. Vor-Usoni

Figure 2.3 compares the recoveries obtained for the two different extraction methods. It is evident that recoveries were generally higher for ASE extraction than Vor-Usoni. The recoveries of all target analytes for ASE extraction exceeded 90% except for DBDPE for which the recovery was ~70%. Moreover, no significant differences in recoveries were observed for the three spike levels. Relatively low variability was seen for the low-level spiked samples for both ASE and Vor-Usoni (RSD < 15%). Recoveries for ASE were less consistent for PBT, HBB, HCDBCO, BEH-TEBP (15% < RSD <17%) in the medium-level spiking experiments with 2 compounds (TBP-DBPE and DBDPE) displaying moderate variability (15% < RSD <18%) for the high-level spiked samples. By comparison, Vor-Usoni displayed moderate variability (15% < RSD < 22%) in high-level spike experiments, for all target compounds except EH-TBB and DBE-DBCH.

Overall, this comparison of the two different methods indicates that the ASE method is more robust for extracting EFRs, and this method was thus chosen as our extraction method for air, dust, diet, and human milk samples.

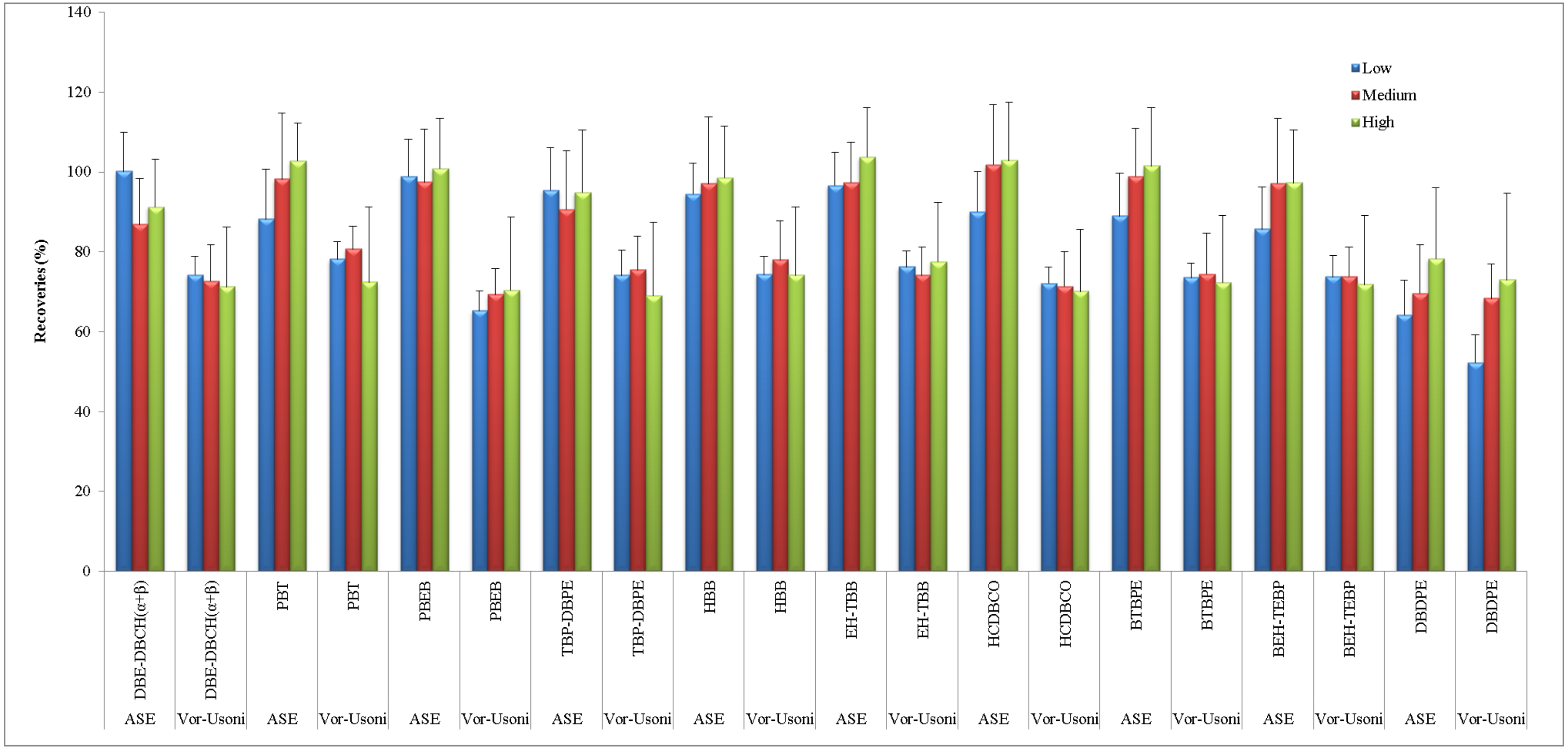


**Figure 2.3 Spiking experiment results on sorbent material. Each level was composed of three replicate measurements on three different days (vertical lines represent standard deviations)**

### 2.4.2 Air and dust sample extraction

Aliquots of dust samples (typically between 50 and 200 mg) were accurately weighed, and then extracted using ASE 350; air samples (combined PUF disks and GFF) were also extracted by ASE. The extraction conditions were the same as for the spiked samples described in 2.4.1.

### 2.4.3 Diet and human milk sample extraction

Aliquots of human milk or diet samples (~500 mg) were accurately weighed and extracted using ASE 350. Extraction cells were filled from the bottom to top with: pre-cleaned hydromatrix, 2 g florisil, 3 g alumina, samples, and then topped with hydromatrix. The other extraction conditions were the same as for the spiked samples as described in 2.4.1.

### 2.4.4 Extraction of diet and soil samples collected in Vietnam

Aliquots of samples (5 g) were extracted in National Institute for Environmental Studies (NIES), Japan using a rapid solvent extractor (SE100, Mitsubishi Chemical Analytech) at 35 ˚C for 40 min with 50% acetone in n-hexane at a flow rate of 2 mL/min, followed by secondary extraction at 80 °C for 40 min with toluene at a flow rate of 2 mL/min. The extracts were evaporated to incipient dryness and then diluted in 10 mL toluene.

## 2.5 Clean up

The effectiveness of three clean-up methods involving different combinations of chromatographic fractionation and acid cleaning, was evaluated. The three methods assessed were as follows.

***Method 1*** Following evaporation to 1 mL, sample extracts were fractionated on a column packed with 2 g florisil. Before the sample extract was applied, the column was washed and conditioned sequentially with 6 mL dichloromethane (DCM) and 6 mL hexane (Hex). The extracts were quantitatively transferred and fractionation achieved by eluting with 20 mL of Hex (Fraction 1-F1) and 35 mL of DCM (Fraction 2 -F2). F1 was evaporated to 1 mL and the solvent applied to 44% acidified silica cartridges (2 g acidified silica, prewashed with 6 mL Hex-DCM (1:1, v/v)) for a second clean-up. The extracts were eluted with 20 mL of Hex-DCM (1:1, v/v). The eluate was then combined with fraction 2 prior to evaporation to dryness under a gentle nitrogen flow and reconstitution in 200 µL of iso-octane containing PCB-129 (250 pg/uL) as a recovery determination standard. After analysis via gas chromatography coupled with electron capture negative ionization mass spectrometry (GC-NCI-MS), samples were evaporated carefully to dryness and resolubilized in 200 uL of methanol containing $d_{18}$-γ-HBCDD (25 pg/µL) as recovery determination standard for LC determination of HBCDDs.

***Method 2*** Following concentration of crude extracts to 1 mL, they were subjected to fractionation on a column packed with 2 g florisil washed and conditioned prior to use sequentially with 6 mL DCM and 6 mL Hex. Crude extracts were quantitatively transferred and fractionation achieved by eluting sequentially with 20 mL of Hex and then 15 mL Hex-DCM (1:1, v/v) (Fraction 1-F1) and 35 mL of DCM (Fraction 2 -F2). F1 was evaporated to 1 mL prior to application of the concentrate to 44% or 22% acidified silica cartridges (2 g acidified silica, prewashed with 6 mL Hex-DCM (1:1, v/v)) for further clean-up. Extracts were eluted through these acidified silica columns with 20 mL of Hex-DCM (1:1, v/v). The eluate was then combined with F2 and afterwards evaporated to near dryness under a gentle nitrogen flow and reconstituted in 200 μL of iso-octane containing PCB129 (250 pg/uL) as a recovery determination standard. After analysis via GC-NCI-MS, the samples were evaporated to dryness and resolubilized in 200 uL of methanol containing $d_{18}$-γ-HBCDD (25 pg/μL) as recovery determination standard for LC determination of HBCDDs.

***<u>Method 3</u>*** Crude extracts were evaporated to 0.5 mL using a Zymark Turbovap® II (Hopkinton, MA, USA) and then transferred to 10 mL glass tubes, followed by clean up with 3-6 mL concentrated sulfuric acid with vortexing (speed 4, 20 s) or gentle shaking 20 times. After vortexing/shaking, glass tubes were centrifuged at 2000 g for 5 mins, the supernatants collected and rinsed with 3 x 2 mL hexane. The pooled supernatants were transferred into clean glass tubes, the glass tubes were evaporated until dryness under a gentle stream of $N_2$, then reconstituted in 200 μL of iso-octane containing 250 pg/uL PCB129 used as a recovery determination standard. After analysis via GC-NCI-MS, the samples were evaporated until dryness and resolubilized in 200 uL of methanol containing $d_{18}$-γ-HBCDD (25 pg/μL) as recovery determination standard for LC determination of HBCDDs.

To evaluate the performance of these three methods, one procedural blank and spiked matrix ($Na_2SO_4$) with three concentration levels: $Q_{low}$ (50 ng of each, 75 ng DBDPE), $Q_{medium}$ (100 ng of each, 150 ng DBDPE) and $Q_{high}$ (200 ng of each, 300 ng DBDPE), were used to assess recoveries for each method. Each of these spiked samples were extracted in triplicate on three separate days (n=9). The recoveries for our target compounds obtained using different clean-up methods are shown in Table 2.6. Using method 1 and method 3 as clean-up procedures, relatively high and reproducible recoveries were achieved for all target compounds. By comparison, recoveries of EH-TBB were low (15 % and 40 %, respectively) in method 2 when using either 44% or 22% acidified silica for clean up. This contrasts with a previous report that satisfactory recoveries were obtained for EH-TBB when using an acid silica column (Ali et al. 2011b). This difference probably arises because of differences in the

**Table 2.6 Relative recoveries (SD) of FRs on different clean-up methods**

| | **Method 1 (%)** | | **Method 2 (%)** | | **Method 3 (%)** | |
|---|---|---|---|---|---|---|
| | **F1** | **F2** | **F1** | **F2** | **Vortexing** | **Shaking** |
| **α-DBE-DBCH** | 60(2) | | 58(4) | | 75(1) | 78(1) |
| **β -DBE-DBCH** | 61(3) | | 59(2) | | 80(3) | 81(1) |
| **TBX** | 78(5) | | 75(6) | | 83(5) | 82(3) |
| **PBBz** | 83(4) | | 82(4) | | 85(8) | 85(2) |
| **TBCT** | 85(2) | | 90(5) | | 85(9) | 90(6) |
| **BDE 28** | 81(3) | | 81(6) | | 88(2) | 91(2) |

| | | | | | | |
|---|---|---|---|---|---|---|
| **PBT** | 82(7) | | 87(7) | | 96(2) | 93(9) |
| **PBEB** | 95(9) | | 96(8) | | 98(4) | 95(2) |
| **TBP-DBPE** | 92(8) | | 92(9) | | 103(2) | 99(2) |
| **HBB** | 93(4) | | 87(10) | | 104(9) | 105(10) |
| **BDE 47** | 96(3) | | 98(2) | | 98(6) | 99(8) |
| **BDE 77** | 92(6) | | 99(11) | | 90(2) | 95(3) |
| **BDE 100** | 93(8) | | 92(2) | | 91(1) | 96(4) |
| **BDE 99** | 93(10) | | 93(5) | | 92(8) | 97(5) |
| **EH-TBB** | | 98(8) | 15(2)[a]/40(2)[b] | | 98(9) | 94(3) |
| **BDE 154** | 104(11) | | 103(3) | | 105(8) | 101(11) |
| **BDE 153** | 107(12) | | 101(4) | | 108(7) | 110(10) |
| **BDE 128** | 100(11) | | 105(2) | | 103(6) | 103(9) |
| **BDE 183** | 101(13) | | 104(10) | | 100(10) | 105(10) |
| **BTBPE** | 103(14) | | 105(11) | | 101(10) | 108(7) |
| **$^{13}$C-BTBPE** | 102(15) | | 103(9) | | 108(2) | 107(8) |
| **$^{13}$C-BEH-TEBP** | | 97(3) | | 98(2) | 99(6) | 95(9) |
| **BEH-TEBP** | | 97(3) | | 98(3) | 99(7) | 96(10) |
| **syn-DDC-CO** | 101(2) | | 105(9) | | 105(8) | 101(11) |
| **anti-DDC-CO** | 87(2) | | 90(3) | | 88(2) | 98(6) |
| **TBBPA-BDBPE** | 60(4) | | 57(2) | | 65(2) | 63(1) |
| **BDE 209** | 99(5) | | 79(7) | | 98(7) | 99(8) |
| **$^{13}$C-BDE 209** | 98(6) | | 88(6) | | 95(9) | 90(3) |
| **DBDPE** | 88(4) | | 90(8 | | 91(3) | 93(2) |
| **HBCDD** | 89(3) | | 90(9) | | 92(2) | 94(4) |

a) clean-up procedure using 44% acidified silica cartridges; b) clean-up procedure using 22% acidified silica cartridges.

precise procedures followed. In this study, 2 g acid silica was used (rather than 1 g in Ali et al. (2011b)) and silica was activated before adding acid. Such differences are important as EH-TBB and the structually-related BEH-TEBP may be destroyed as a consequence of long contact times with acid on acidified silica column. Interestingly, the recoveries of BEH-TEBP were high and reproducible when using method 3 incorporating a direct acid wash by gentle vortexing or shaking. This contrasts with previous studies that have shown BEH-TEBP to degrade on acidified silica (Ali et al., 2011b; Sahlström et al., 2012; Stapleton et al., 2008; Van den Eede et al., 2012). Our findings point to a crucial role of silica as a support to the acid in effecting degradation and are consistent with a recent report of <10% recovery of BEH-TEBP when acidified silica was used, but 114% recovery when washed with concentrated acid alone (Guo et al., 2014). The authors of this study suggested that the poor recoveries when using acidified silica may be attributed to longer contact times with acid and surface dependent reactions when using this reagent as opposed to neat acid. Overall, based on the data shown in Table 2.6, methods 1 and 3 both appear reliable clean up methods. However, method 3 used less solvent and was less time-consuming and was therefore selected as the clean-up method to be used in this study.

### 2.5.1 Air and dust sample clean up procedures

The crude extracts were evaporated to 0.5 mL using a Zymark Turbovap® II (Hopkinton, MA, USA) and then transferred to 10 mL glass tubes, followed by clean up by vortexing (20s) with 3-4 mL concentrated sulfuric acid. After vortexing, the glass tubes were centrifuged at 2000 g for 5 mins and the supernatants were collected and rinsed with 3 x 2 mL hexane. The pooled supernatants were transferred into clean glass tubes, the glass tubes were evaporated to incipient dryness under a gentle stream of $N_2$, before reconstitution in 200 µL (dust) and 50 µL (air) iso-octane containing 250 pg/uL PCB129 as recovery determination standard.

### 2.5.2 Diet and human milk samples clean up procedures

Crude extracts of diet and human milk samples were evaporated to 0.5 mL using a Zymark Turbovap® II (Hopkinton, MA, USA) and then transferred to 10 mL glass tubes, followed by clean up by shaking with 5-6 mL concentrated sulfuric acid (20 times). After shaking, the glass tubes were centrifuged at 2000 g for 3 mins, before the supernatants were collected and rinsed with 3 x 2 mL hexane. The pooled supernatants were transferred into clean glass tubes, evaporated to near dryness under a gentle stream of $N_2$, and reconstituted in 100 µL of iso-octane containing 250 pg/uL PCB129 as recovery determination standard.

## 2.6 Instrumental Analysis

### 2.6.1 GC-NCI-MS analysis for determination of PBDEs and EFRs

For analysis of EFRs and PBDEs, samples were injected into Thermo Trace 1310 GC coupled to a Thermo mass spectrometer (ISQ™ LT Single Quadrupole) operated in ECNI mode with a programmable-temperature vaporizer (PTV) injector. 2 µL of cleaned extract were injected on a TG-SQC column (15 m×0.25 mm×0.25 µm) using solvent vent injection. Injection was performed under a pressure of 0.19 bar for 1 min and purge flow to split vent of 50 mL/min. The GC temperature program was 50 °C, hold 0.50 min, ramp 20 °C/min to 240 °C, hold 5 min, ramp 5 °C /min to 270 °C, ramp 20 °C/min to 305 °C, hold 17 min. Helium was used as a carrier gas with a starting flow rate of 1.5 mL/min, hold 22 min, ramp 1 mL/min to 2.5 mL/min, hold 13 min. The electron multiplier voltage was 1460 V. Methane was used as moderating gas.

#### 2.6.1.1 Evaluation of the PTV vs the Split/Splitless injector

The split/splitless injector is one of the most common injection techniques for PBDE analysis. However, low transfer efficiency of analytes with high boiling points to the column and thermal degradation and discrimination of higher molecular weight PBDEs are encountered using the split/splitless injector (Kierkegaard et al., 2009; Stapleton, 2006). In the PTV injector, the transfer of compounds with high boiling points is enhanced via gradual heating of the injector during sample injection. By introducing the sample in the liquid state into a low temperature liner and subsequently raising the temperature of the liner to the normal temperature of a conventional hot injector, the risk of thermal degradation and discrimination is minimised (Kierkegaard et al., 2009). Therefore, in this study, peak areas of the two techniques were compared using the same concentration standards for PBDEs and EFRs (1 ng/uL of each). The PTV injection temperature was set at 92 °C, hold 0.04 min, ramp 11.7 °C /sec to 295 °C, hold 20 mins. The splitless mode temperature was set at 280 °C. Table 2.7 shows that the peak areas obtained when using the PTV were 1.2 to 2.8 times larger than those obtained using conventional split/splitless injection for most PBDEs and EFRs, with the peak area for DBDPE obtained using PTV being 6.3 times higher than that achieved in splitless mode (SL). This enhanced performance increased substantially with increasing number of bromines.

#### 2.6.1.2 Influence of GC-MS transfer line temperatures

In NCI mode, we examined the impact of GC-MS transfer line temperatures on peak area for our target compounds. We evaluated two sets of temperatures: NCI-1 = 230 and 280 °C, and NCI-2 = 300 and 320 °C. As shown in Table 2.7, while NCI-2 conditions gave slightly worse performance for lower molecular weight compounds, they yielded proportionally better performance for higher molecular weight compounds. Thus the NCI-2 temperatures were incorporated into our method.

#### 2.6.1.3 Evaluation of the three different GC injection port liners

GC injection port liners play an important role in the volatilisation and transfer of analytes onto the GC column and ultimately on detection limits. In this study, three different liners were investigated: PTV siltek metal thick liner (2 mm x 2.75 mm x 120 mm), PTV siltek metal thin liner (1 mm x 2.75 mm x 120 mm), and a baffled liner (2 mm x 2.75 mm x 120 mm). Table 2.7 shows that while the thin siltek liner performed well for all compounds except DDC-CO and TBBPA-BDBPE, the baffled liner showed excellent results for all target compounds especially for high boiling analytes such as BDE 209 and DBDPE. Consequently, the baffled liner was used in the GC method because of its superior performance.

### 2.6.2 LC-ESI-MS/MS analysis for determination of HBCDDs

With respect to the instrumental analysis of HBCDDs in this study, a method reported previously (Harrad et al., 2009a) was used with minor modifications. Briefly, analysis of three HBCDD diastereomers (α, β, and γ-HBCDDs) was achieved using a dual

**Table 2.7 Peak area ratios of PBDEs and EFRs injected on injection mode (PTV and Splitless)**

|  | NCI-2[a]/NCI-1[b] | PTV/SL | Thin liner/Thick liner | Baffled liner/Thin liner |
|---|---|---|---|---|
| **α-DBE-DBCH** | 0.88 | 1.0 | 2.2 | 1.7 |
| **β-DBE-DBCH** | 0.89 | 1.0 | 2.3 | 1.7 |
| **TBX** | 0.87 | 1.1 | 1.0 | 1.4 |
| **PBBz** | 0.89 | 1.1 | 1.1 | 1.0 |
| **TBCT** | 0.90 | 1.1 | 1.0 | 1.2 |
| **BDE 28** | 0.92 | 1.2 | 1.0 | 1.2 |
| **PBT** | 0.82 | 1.3 | 1.0 | 1.2 |
| **PBEB** | 0.96 | 1.5 | 1.0 | 1.3 |
| **PCB129** | 0.42 | 1.9 | 1.0 | 1.0 |
| **TBP-DBPE** | 0.92 | 1.8 | 1.3 | 1.3 |
| **HBB** | 0.93 | 1.9 | 1.2 | 1.1 |
| **BDE 47** | 0.88 | 1.7 | 1.2 | 1.1 |
| **BDE 77** | 0.89 | 1.5 | 1.4 | 1.1 |
| **BDE 100** | 0.86 | 1.5 | 1.3 | 1.0 |
| **BDE 99** | 0.83 | 2.4 | 1.3 | 1.0 |

| | | | | |
|---|---|---|---|---|
| **EH-TBB** | 0.76 | 2.1 | 1.5 | 1.1 |
| **BDE 154** | 0.83 | 2.2 | 1.3 | 1.1 |
| **BDE 153** | 0.86 | 2.2 | 1.3 | 1.1 |
| **BDE 128** | 0.89 | 1.5 | 1.7 | 1.4 |
| **BDE 183** | 0.91 | 1.9 | 1.3 | 1.2 |
| **BTBPE** | 1.3 | 2.7 | 2.1 | 1.4 |
| **$^{13}$C-BTBPE** | 1.2 | 2.5 | 2.1 | 1.3 |
| **$^{13}$C-BEH-TEBP** | 1.2 | 2.7 | 1.4 | 3.3 |
| **BEH-TEBP** | 1.2 | 2.8 | 1.5 | 5.2 |
| **syn-DDC-CO** | 1.3 | 2.6 | 0.8 | 1.0 |
| **anti-DDC-CO** | 1.3 | 2.6 | 0.9 | 1.1 |
| **TBBPA-BDBPE** | 1.4 | 2.8 | 0.2 | 0.6 |
| **BDE 209** | 1.2 | 2.6 | 15.3 | 2.6 |
| **$^{13}$C-BDE 209** | 1.2 | 2.5 | 16.2 | 2.8 |
| **DBDPE** | 1.1 | 6.3 | 3.2 | 7.1 |

a) Transfer line: 300 ℃ Ion source: 320 ℃; b) Transfer line: 230 ℃ Ion source: 280 ℃.

pump Shimadzu LC-20AB Prominence high pressure liquid chromatograph (Shimadzu, Kyoto, Japan) equipped with a Varian Pursuit XRS3 (Varian, Inc., Palo Alto, CA, USA) C18 reversed phase analytical column (150 mm × 2 mm i.d., 3 μm particle size). An SIL-20A autosampler and a DGU-20A3 vacuum degasser were used. The following mobile phase program was used: (a) 1:1 methanol/water and (b) methanol at a flow rate of 180 μL/min was applied; the mobile phase b starts at 50% before increasing linearly to 100% over 4 min, held for 5 min followed by a linear decrease to 88% over 1 min, and a rapid drop to 50%, held for 1 min. The post-elution was conducted by increasing the mobile phase b gradually to 100% over 1 min, held 6 min, and then finished to 50% for 4 min. Using this method, α-, β-, and $\gamma$-HBCDDs were separated at the retention times of 9.0, 10.6, and 11.2, respectively.

Mass spectrometric analysis was performed using a Sciex API 2000 triple quadrupole mass spectrometer (Applied Biosystems, Foster City, CA, USA) operated in electrospray negative ionisation mode ($ESI^-$). MS/MS detection operated in the multiple reaction monitoring (MRM) mode was applied for quantitative determination of the target compounds based on *m/z* 640.6/79, *m/z* 652.4/79 and *m/z* 657.7/79 for the native, $^{13}$C-HBCDDs and $d_{18}$-$\gamma$-HBCDD, respectively.

## 2.7 QA/QC and validation of methods

### 2.7.1 Analyte identification and quantification criteria

The specific retention time was confirmed for each studied compound by injecting pure individual standards of each analyte (1 ng on column) on GC-NCI-MS in our method. To evaluate the linearity of the MS response, full five-point calibrations were conducted for most of target compounds over a concentration range from 25 to 500 pg/uL, for BDE 209 over a concentration range from 50 to 1000 pg/uL, and for TBBPA-BDBPE over a concentration range spanning 1000 to 10000 pg/uL. Excellent linearity ($R^2 > 0.99$) was observed over the studied ranges for all of the studied compounds. Known amounts of $^{13}$C-labelled isomers were spiked into the samples prior to extraction as internal standards (ISs) for BTBPE, BEH-TEBP, BDE 209 and

HBCDDs, respectively. This is because the detected ions for native and $^{13}$C-labelled isomers are different for these compounds and thus there are no interferences between the fragmentation of native and $^{13}$C-labelled isomers. In contrast, BDE 77 and BDE 128 were used for quantification of our other target compounds as the level of BDEs 77 and 128 in the environment are extremely low and will not affect their response when used as internal standards.

ISs were also added to the five-point calibration plot solutions to calculate relative response factors (RRFs) for each target compound. The RRF is defined as the instrument response for a unit amount of target analyte relative to the instrument response obtained for the same amount of the internal standard (IS) and is calculated as in equation 2.2.

$$\mathrm{RRF} = \frac{A_{NAT}}{A_{IS}} \times \frac{C_{IS}}{C_{NAT}} \ldots\ldots\ldots\ldots\ldots (2.2)$$

Where $A_{NAT}$ is the peak area of the native compound (i.e. the $^{12}$C of the target compound); $A_{IS}$ is the peak area of the internal standard used for each analyte; $C_{NAT}$ is the level of the native compound; and $C_{IS}$ is the concentration of the internal standard. The relative standard deviation (RSD) of the RRFs calculated for studied compounds from the calibration curves did not exceed 5%.

A calibration standard was injected before and after each sample batch (around 10 samples). The average RRF for each target compound from these two injections was calculated. These must not exceed ± 25% of the average RRFs calculated for those standards in the initial five-point calibration curves and used for calculating the level of each target compound in different types of samples of this batch using equation 2.3.

$$\text{Concentration} = \frac{A_{NAT}}{A_{IS}} \times \frac{1}{RRF} \times \frac{M_{IS}}{SS} \dots\dots\dots\dots\dots (2.3)$$

Where $A_{IS}$ is the peak area of internal standard in each sample; $A_{NAT}$ is the peak area of target compound in each sample; RRF is the relative response factor for the target pollutant; $M_{IS}$ is mass of internal standard added to sample (pg or ng) and SS is the sample size ($m^3$ or g).

For a peak in the chromatogram for each sample to be quantified as a target pollutant in a sample, the following criteria needed to be met:

1. The signal to noise ratio (S/N) must exceed 10:1.

2. The isotope ratios must not exceed ± 20% of the average value for the 2 calibration standards injected before and after that sample batch.

3. The relative retention time (RRT) of the peak in the sample must not exceed ± 0.2% of the average value calculated for the same compound in the 2 calibration standards injected before and after that sample batch.

### 2.7.2 Recovery determination (syringe) standard (RDS)

The recoveries of IS in the sample preparation were calculated using the RDS added to the samples before GC-MS analysis. In this study, $d_{18}$-γ- HBCDD was used as RDS for α-, β-, and γ- HBCDDs, while PCB129 was used as RDS for IS for PBDEs and

EFRs. The recoveries of the IS in each sample were calculated as in equation 2.4.

$$\%\text{IS Recovery} = \left[\left(\frac{A_{IS}}{A_{RDS}}\right)_S \times \left(\frac{A_{RDS}}{A_{IS}}\right)_{STD} \times \left(\frac{C_{IS}}{C_{RDS}}\right)_{STD} \times \left(\frac{C_{RDS}}{C_{IS}}\right)_S\right] \times 100 \ldots\ldots\ldots\ldots\ldots (2.4)$$

where $(A_{IS}/A_{RDS})_S$ is the ratio of internal standard peak area to recovery determination standard peak area in the sample; $(A_{RDS}/A_{IS})_{STD}$ is the ratio of recovery determination standard peak area to internal standard peak area in the calibration standard (the average of values calculated using 2 calibration standards injected before and after this batch of samples); $(C_{IS}/C_{RDS})_{STD}$ is the ratio of concentration of internal standard to concentration of recovery determination standard in the calibration standard; and $(C_{RDS}/C_{IS})_S$ is the ratio of concentration of recovery determination standard to concentration of internal standard in the sample.

### 2.7.3 Analysis of Blanks and calculation of LODs and LOQs

Instrumental limits of detection (LOD) and method limits of quantification (LOQ) were calculated for each target compound based on a 3:1 and 10:1 signal to noise ratio column respectively which are shown in Table 2.8.

For air samples, none of the target compounds were detected in method blanks (n=4) consisting of a pre-cleaned PUF disk and a GFF. One method blank was prepared using the same analytical method for each batch of five samples. None of the target compounds were detected in method blanks for air, dust, soil, human milk and diet samples. While very low concentrations of HBCDDs and BDE 209 which didn't exceed 5 % of the lowest concentration in the samples were observed in field blanks.

**Table 2.8 LODs and LOQs of PBDEs, HBCDDs and EFRs**

| | | **Air** | **Dust** | **Diet and Human milk** |
|---|---|---|---|---|
| | **LOD pg/uL** | **LOQ (pg/m$^3$)** | **LOQ (ng/g)** | **LOQ (ng/g dry weight)** |
| **α-DBE-DBCH** | 0.070 | 1.6 | 0.2 | 0.04 |
| **β-DBE-DBCH** | 0.070 | 1.6 | 0.2 | 0.04 |
| **TBX** | 0.004 | 0.1 | 0.01 | 0.003 |
| **PBBz** | 0.004 | 0.1 | 0.01 | 0.003 |
| **TBCT** | 0.004 | 0.1 | 0.01 | 0.003 |
| **PBT** | 0.004 | 0.1 | 0.01 | 0.003 |
| **PBEB** | 0.004 | 0.1 | 0.01 | 0.003 |
| **TBP-DBPE** | 0.018 | 0.4 | 0.05 | 0.01 |
| **HBB** | 0.011 | 0.2 | 0.03 | 0.01 |
| **EH-TBB** | 0.004 | 0.1 | 0.01 | 0.003 |
| **BTBPE** | 0.046 | 1.0 | 0.13 | 0.03 |
| **BEH-TEBP** | 0.004 | 0.1 | 0.01 | 0.003 |
| ***syn*-DDC-CO** | 0.088 | 2.0 | 0.25 | 0.05 |
| ***anti*-DDC-CO** | 0.053 | 1.2 | 0.15 | 0.03 |
| **TBBPA-BDBPE** | 0.44 | 10 | 1.25 | 0.25 |
| **DBDPE** | 0.44 | 10 | 1.25 | 0.25 |
| **BDE 28** | 0.011 | 0.2 | 0.03 | 0.01 |

| | | | | |
|---|---|---|---|---|
| **BDE 47** | 0.014 | 0.3 | 0.04 | 0.01 |
| **BDE 100** | 0.004 | 0.1 | 0.01 | 0.003 |
| **BDE 99** | 0.004 | 0.1 | 0.01 | 0.003 |
| **BDE 154** | 0.021 | 0.5 | 0.06 | 0.01 |
| **BDE 153** | 0.018 | 0.4 | 0.05 | 0.01 |
| **BDE 183** | 0.046 | 1.0 | 0.13 | 0.03 |
| **BDE 209** | 0.12 | 2.8 | 0.35 | 0.07 |
| **α-HBCDD** | 0.056 | 1.3 | 0.16 | 0.03 |
| **β-HBCDD** | 0.049 | 1.1 | 0.14 | 0.03 |
| **γ-HBCDD** | 0.039 | 0.9 | 0.11 | 0.02 |

### 2.7.4 Accuracy and precision

As an initial evaluation of method accuracy and precision, 6 aliquots of SRM 2585 were analysed to validate the extraction and clean-up method developed for PBDEs, HBCDDs and EFRs in combination with GC-NCI-MS and LC-MS/MS. The values obtained are compared with certified/indicative/previously reported levels as appropriate in Table 2.9. Levels of PBDEs using NCI mode measured with the analytical method were comparable to the certified values.

With respect to EFRs, the concentrations of EH-TBB, BTBPE and BEH-TEBP obtained in this study were similar to those reported previously (Ali et al. 2011a; Van den Eede et al. 2012; Sahlström et al. 2012; and Cristale & Lacorte 2013). In contrast, Sahlström et al. (2012) reported BEH-TEBP levels much higher than those found in

**Table 2.9 Mean values and standard deviations (ng/g dust) of flame retardants measured in SRM 2585 (standard deviations in parentheses)**

| | This study N=6 (NCI source) | NIST certified/ indicative values | Ali et al. (2011b) N=2 (NCI source) | Sahlstrom et al. (2012) N=5 (NCI source) | Stapleton et al. (2008) N=3 (NCI source) | Cristale et al. (2013) N=4 (GC-EI-MS/MS) | Van den Eede et al. (2012) N=6 (NCI source) |
|---|---|---|---|---|---|---|---|
| **BDE 28** | 48.7(14) | 46.9 | | | | | |
| **BDE 47** | 458(114) | 497 | | | | | |
| **BDE 99** | 752(178) | 892 | | | | | |
| **BDE 100** | 103(50) | 145 | | | | | |
| **BDE 153** | 129(32) | 119 | | | | | |
| **BDE 154** | 108.4(55)[a] | 83.5 | | | | | |
| **BDE 183** | 52(14) | 43 | | | | | |
| **BDE 209** | 2329(175) | 2510 | | | | | |
| **EH-TBB** | 49(12) | -- | 40 | 36(2.4) | <30 | 35(6) | 26(2) |
| **BTBPE** | 63(7) | -- | 32 | 39(4.9) | <0.8 | 76(4) | 39(14) |
| **BEH-TEBP** | 863(175) | -- | 652 | 1300(94) | 145(16.7) | 857(73) | 574(49) |
| **α-HBCDD** | 21(2.0) | 19(3.7)[b] | -- | 25(5.6) | -- | -- | 19(9) |
| **β-HBCDD** | 4.2(0.3) | 4.3(1.1)[b] | -- | 5.7(0.2) | -- | -- | 4.2(1.4) |
| **γ-HBCDD** | 115(18) | 121(22)[b] | -- | 80(13) | -- | -- | 119(42) |
| **∑HBCDD** | 140(21) | -- | -- | 100(17) | 137(7.8) | -- | 141(45) |

SD: standard deviation; a) co-elution with BB-153, b) Keller, J. M.; Stapleton, H. M.;

Heltsley, R.; Peck, A.; Kucklick, J. R.; Schantz, M.; Wise, S. A. SRMs Available from NIST for the Analysis of Brominated Flame Retardants; Poster presented at BFR07, Amsterdam, 24–27 April, 2007.

our study. However, our value is consistent with most other studies. Finally, PBEB was detected in our study while it was not detected in any of the previous studies.

## 2.8 Statistical Analysis

Statistical analysis of the data in this study was conducted using both Excel (Microsoft Office 2010) and IBM SPSS Statistics 21.0 (Chicago, IL, U.S.A.). SPSS Statistics 21.0 was used to perform the statistical analyses for compounds with detection frequencies ≥40%. In the instances where analyte peaks were below LOQ and the detection frequencies (DF) are higher than 50%, the concentrations of target compounds are reported as LOQ/2, and if the detection frequencies are lower than 50%, the concentration for each compound is reported as LOQ*DF – e.g. where the detection frequency is 45%, the concentration of compounds below LOQ are assumed to be LOQ x 0.45. Statistical analysis (ANOVA and independent t-test) was performed on logtransformed concentrations on IBM SPSS Statistics 21.0 after concentrations in all data sets were revealed to be log-normally distributed using both the Kolmogorov-Smirnov test and visual inspection of the quantile-by-quantile graphic plot in SPSS and a p value <0.05 was regarded as indicating statistical significance.

This chapter contains some sections (2.7.1, 2.7.2 and 2.7.3) of verbatim text adapted from our lab’s protocol for analysis of semi-volatile persistent organic pollutants (prepared by: Prof. Stuart Harrad, Persistent Organic Pollutants Research Group, Public Health Building, School of Geography, Earth & Environmental Sciences, University of Birmingham).

# CHAPTER 3 EFRs and HBCDDs in food Samples from an e-waste processing area in Vietnam

## 3.1 Synopsis

More and more attention is being paid to environmental contamination arising from emissions occurring during end-of-life treatment of treated goods. Particular concern exists about situations where electrical and electronic waste (e-waste) is dismantled under rudimentary conditions. Numerous studies have shown elevated contamination of air, dust, soil, and sediments with PBDEs in such locations (Chen et al., 2009; Labunska et al., 2013; Liu et al., 2008) with a smaller number of recent studies reporting elevated human dietary exposure to local populations. In contrast, relatively few data exist about environmental contamination with EFRs in areas where such informal e-waste processing is conducted. In addition to PBDEs, HBCDDs have also been frequently detected in the environment and human milk sampled in the vicinity of rudimentary e-waste processing sites (Gao et al., 2011; Tue et al., 2013, 2010). However, studies of human exposure via consumption of food sourced from locations where rudimentary processing of e-waste is conducted are scarce.

The available data for PBDEs and HBCDDs suggest that fish, pigs, and free-range chickens reared in e-waste impacted locations accumulate these compounds – likely through uptake from their environment (e.g. soil and sediment) and food. Therefore, concentrations of EFRs and HBCDDs were investigated in such foodstuffs collected from a location impacted by rudimentary e-waste processing in Bui Dau, Vietnam. These data are compared with concentrations detected in samples of the same matrices sourced from non-e-waste impacted control locations in Vietnam and Japan, and are combined with information on their consumption by local inhabitants to estimate human exposure to these contaminants. Concentrations of EFRs and HBCDDs are also measured in soil samples from the same e-waste impacted locations, to investigate the impact of soil contamination on chicken tissues and eggs.

## 3.2 Detection of HFRs in food and soil samples

### 3.2.1 Concentrations in food

Table 3.1 shows concentrations of HFRs in food, co-located soil and co-located sediment samples (Someya et al., 2015). Of our target EFRs: PBBz, HBB, BEH-TEBP, BTBPE, DBDPE, *syn*-DDC-CO, *anti*-DDC-CO were detectable in chicken samples, with the same EFRs (except PBBz) found in soil samples. In contrast, only BTBPE was detected in river fish, only DBDPE was detectable in pork and no EFRs were detected in pond fish.

Most strikingly, concentrations of all target EFRs in e-waste-impacted samples in this study exceed substantially those detected in the corresponding controls. This suggests substantial impact of the e-waste processing activities on the environment in Bui Dau. These findings are consistent with previous studies of PBDEs and some EFRs in Taizhou, eastern China (Labunska et al., 2015, 2014). DDC-COs were the most frequently detected EFR (100% detection) in chicken samples (muscle, liver, egg, skin), followed by BTBPE and HBB with a detection frequency of 70% and 50%, respectively. PBBz was less frequently detected in chicken samples while BEH-TEBP

**Table 3.1 Average, median and range of concentrations of EFRs and HBCDDs (ng/g lw) in food and co-located soil and sediment samples (ng/g dw) from an e-waste processing (Bui Dau, Vietnam) and control sites.**

| | | Chicken muscle (n=5) | Chicken liver (n=5) | Chicken skin (n=5) | Chicken egg (n=15) | Soil (n=5) | River fish (n=5) | Pork (n=2) | Sediment[c] (n=8) | Control chicken muscle-1 (n=1)[a] | Control chicken egg-1 (n=3) [a] | Control chicken muscle-2 (n=1) [b] | Control chicken egg-2 (n=1) [b] | Control chicken liver (n=1) [b] | Control chicken skin (n=1) [b] | Control fish (n=1) [a] | Control pork (n=1) [a] |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Lipid%** | | 6.0 | 15 | 43 | 42 | -- | 4.0 | 18 | -- | 21 | 42 | 2.0 | 44 | 12 | 46 | 17 | 30 |
| **PBBz** | average | <1.5 | 2.0 | 1.3 | 0.92 | <0.15 | <0.36 | <0.36 | -- | <1.5 | <0.31 | <1.5 | <0.31 | <0.36 | <0.31 | <0.36 | <0.36 |
| | median | <1.5 | <0.36 | 0.42 | 0.96 | <0.15 | <0.36 | <0.36 | -- | <1.5 | <0.31 | <1.5 | <0.31 | <0.36 | <0.31 | <0.36 | <0.36 |
| | range | -- | <0.50-3.7 | <0.50-2.0 | 0.19-1.9 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- |
| **HBB** | average | 6.4 | 6.8 | 3.0 | 1.5 | 6.9 | <0.36 | <0.36 | 0.033 | <1.5 | <0.30 | <1.5 | <0.30 | <0.36 | <0.30 | <0.36 | <0.36 |
| | median | <1.5 | <0.36 | 0.49 | 1.4 | 1.3 | <0.36 | <0.36 | -- | <1.5 | <0.30 | <1.5 | <0.30 | <0.36 | <0.30 | <0.36 | <0.36 |
| | range | <1.5-10 | <0.36-11 | <0.30-5.8 | <1.3-2.0 | 0.15-21 | -- | -- | ND[d]-0.23 | -- | -- | -- | -- | -- | -- | -- | -- |

|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **BEH-TEBP** | average | <6.5 | <1.5 | <1.3 | 2.0 | 0.13 | <1.5 | <1.5 | <0.20 | <6.5 | <1.3 | <6.5 | <1.3 | <1.5 | <1.3 | <1.5 | <1.5 |
|  | median | <6.5 | <1.5 | <1.3 | <1.3 | <0.050 | <1.5 | <1.5 | -- | <6.5 | <1.3 | <6.5 | <1.3 | <1.5 | <1.3 | <1.5 | <1.5 |
|  | range | -- | -- | -- | <1.3-3.0 | <0.050-0.40 | -- | -- | ND-1.6 | -- | -- | -- | -- | -- | -- | -- | -- |
| **BTBPE** | average | 60 | 54 | 70 | 67 | 10 | 40 | <0.70 | 0.89 | <3.1 | <0.62 | <3.1 | <0.62 | <0.70 | <0.62 | <0.70 | <0.70 |
|  | median | 46 | 12 | 25 | 18 | 5.2 | 55 | <0.70 | -- | <3.1 | <0.62 | <3.1 | <0.62 | <0.70 | <0.62 | <0.70 | <0.70 |
|  | range | <3.1-100 | <0.70-130 | <0.62-100 | <2.8-160 | 0.19-34 | <1.0-57 | -- | ND-5.7 | -- | -- | -- | -- | -- | -- | -- | -- |
| **DBDPE** | average | 6.9 | <3.0 | <2.8 | 280 | 21 | <3.0 | 2.5 | 3.1 | <7.5 | <2.5 | <7.5 | <2.5 | <3.0 | <2.5 | <3.0 | <3.0 |
|  | median | <7.5 | <3.0 | <2.8 | <2.5 | 12 | <3.0 | -- | -- | <7.5 | <2.5 | <7.5 | <2.5 | <3.0 | <2.5 | <3.0 | <3.0 |
|  | range | <7.5-9.9 | -- | -- | <2.5-620 | 0.42-64 | -- | -- | ND-20 | -- | -- | -- | -- | -- | -- | -- | -- |
| ***syn*-DDC-CO** | average | 310 | 1600 | 170 | 140 | 3.8 | <0.36 | <0.36 | 0.17 | <1.6 | <0.32 | <1.6 | <0.32 | <0.36 | <0.32 | <0.36 | <0.36 |
|  | median | 76 | 170 | 90 | 5.0 | 0.60 | <0.36 | <0.36 | -- | <1.6 | <0.32 | <1.6 | <0.32 | <0.36 | <0.32 | <0.36 | <0.36 |
|  | range | <1.6-1400 | <0.36-8000 | 0.13-790 | 3.4-560 | 0.20-13 | -- | -- | ND-1.8 | -- | -- | -- | -- | -- | -- | -- | -- |
| ***anti*-DDC-CO** | average | 1200 | 3500 | 350 | 450 | 10 | <0.36 | <0.36 | 0.45 | <1.6 | <0.32 | <1.6 | <0.32 | <0.36 | <0.32 | <0.36 | <0.36 |
|  | median | 260 | 210 | 160 | 8.0 | 2.3 | <0.36 | <0.36 | -- | -- | -- | -- | -- | -- | -- | -- | -- |
|  | range | <1.6- | 7.5- | 0.32- | 5.1- | 0.83-31 | -- | -- | ND-4.8 | -- | -- | -- | -- | -- | -- | -- | -- |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1600 | 17000 | 3000 | 1800 | | | | | | | | | | | | |
| **ΣDDC-CO** | average | 1500 | 5100 | 520 | 590 | 14 | <0.36 | <0.36 | 0.59 | <1.6 | <0.32 | <1.6 | <0.32 | <0.36 | <0.32 | <0.36 | <0.36 |
| | median | 340 | 380 | 250 | 13 | 2.9 | <0.36 | <0.36 | -- | -- | -- | -- | -- | -- | -- | -- | -- |
| | range | <1.6-3000 | 7.5-25000 | 0.44-3800 | 4.0-2300 | 1.3-44 | -- | -- | ND-6.7 | -- | -- | -- | -- | -- | -- | -- | -- |
| **α-HBCDD** | average | 34 | 1700 | 600 | 2800 | 6.7 | 2.5 | 0.90 | -- | 0.10 | 0.10 | 17 | 1.6 | <0.020 | 1.0 | <0.020 | <0.020 |
| | median | 20 | 1000 | 640 | 2500 | 5.0 | 2.3 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- |
| | range | 1.2-55 | 180-2500 | 20-850 | 330-3500 | 1.0-8.9 | 0.10-3.3 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- |
| **β-HBCDD** | average | 0.15 | 28 | 0.06 | 79 | 4.8 | 0.41 | <0.020 | -- | <0.010 | <0.010 | <0.010 | <0.010 | <0.010 | <0.010 | <0.010 | <0.010 |
| | median | 0.25 | 20 | 0.10 | 70 | 3.8 | 0.51 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- |
| | range | 0.10-2.0 | 2.5-35 | 0.02-0.15 | 15-80 | 0.50-8.0 | 0.040-0.65 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- |
| **γ-HBCDD** | average | 5.3 | 1500 | 330 | 700 | 110 | 0.50 | 0.20 | -- | <0.020 | <0.020 | 0.57 | 0.95 | <0.020 | <0.020 | <0.020 | <0.020 |
| | median | 8.9 | 890 | 450 | 910 | 100 | 0.60 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- |
| | range | 0.10-15 | 160-3000 | 3.8-580 | 200-2300 | 56-710 | 0.050-0.12 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- |
| **ΣHBCDD** | average | 39 | 3200 | 930 | 3600 | 120 | 3.4 | 1.1 | -- | 0.10 | 0.10 | 18 | 2.6 | <0.050 | 1.0 | <0.050 | <0.050 |
| | median | 29 | 1900 | 1000 | 3500 | 110 | 3.4 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | range | 2.0-80 | 330-5500 | 25-1400 | 540-5800 | 0.030-580 | 0.20-4.1 | -- | -- | -- | -- | -- | -- | -- | -- | -- | -- |

a) Control samples collected in Vietnam; b) Control samples collected in Japan; c) from Someya et al., 2015; d) ND= not detected.

was only detected in chicken egg samples. In contrast, the most frequently detected compounds in foods from e-waste processing sites in Taizhou, eastern China were EH-TBB and BEH-TEBP (Labunska et al., 2015). This likely reflects different waste compositions at the two locations. Interestingly, in this study, despite the theoretical predictions that organic contaminants for which Log $K_{OW}>7$ become increasingly less prone to uptake by biota (Thomann, 1989), we detected DBDPE (for which Log $K_{OW}$ is a reported to be 11.1 (Covaci et al., 2011)) in chicken muscle, egg and pork samples in this study. The most abundant EFR detected in chicken was DDC-CO, with average concentrations of ΣDDC-CO in chicken ranged between 520 and 5,100 ng/g lw, comprising around 90% of the sum of EFRs measured in this study. This is consistent with previous findings for chicken egg samples from e-waste recycling sites in South China (Zheng et al., 2012). DDC-CO was not measured in samples taken from Taizhou in the study of Labunska et al. (2015) so comparison is not possible in this instance. The predominance of DDC-CO is likely attributable to its classification as a high production volume (HPV) compound (Xian et al., 2011), a term used to describe a chemical produced in the United States in quantities exceeding 450 t per year, and the significant biomagnification potentials of both *syn*- and *anti*-DDC-CO in food samples (Tomy et al., 2008a; Wu et al., 2010b). Furthermore, DDC-CO is used in electrical cable coatings (Sverko et al., 2011) and dismantling of electrical wires was one of the main e-waste processing activities in Bui Dau. Consequently, DDC-CO may be discharged to the environment during the handling of electrical cables. With respect to our other targeted EFRs, average concentrations in chicken samples of

PBBz, HBB, BEH-TEBP, BTBPE, and DBDPE were <1.5-2.0, 1.5-6.8, <1.3-2.0, 54-70, and <2.8-280 ng/g lw, respectively. The highest concentration of ΣDDC-CO (25,000 ng/g lw) was found in chicken liver, at the high end of values (nd-9630 ng/g lw) previously reported for ΣDDC-CO in food samples (Kang et al., 2010; Sun et al., 2015; Wu et al., 2010b; Zheng et al., 2012). Only limited studies of the presence of EFRs in food samples from e-waste processing sites, especially in different chicken tissues, are available. One previous study reported concentrations of ΣDDC-CO, DBDPE, BTBPE and HBB in chicken eggs from e-waste recycling sites in South China, to fall within the ranges 665-3,290, 5.97-37.9, 37.2-264 and 7.32-25.7 ng/g lw, respectively (Zheng et al., 2012). These concentrations exceed those found for ΣDDC-CO (4.0-2,300 ng/g lw) and HBB (<1.3-2.0 ng/g lw) in this study, but are similar to those we report here for DBDPE (<2.8-620 ng/g lw) and BTBPE (<2.8-160 ng/g lw) in eggs. Average concentrations of HBB and BTBPE in chicken livers (<0.15 and 15.0 ng/g lw, respectively), chicken muscle samples (0.41 and 1.46 ng/g lw) and chicken eggs (<0.15 and 2.93 ng/g lw) from e-waste processing areas in Taizhou (Labunska et al., 2015) are much lower than those in this study (results shown in Table 3.1). Moreover, the concentrations of ΣDDC-CO in chicken samples in this study were comparable to those in chicken liver (4.4 ng/g ww) and muscle samples (0.92 ng/g ww) from e-waste processing sites in China (Zheng et al., 2014). Our analyses of DDC-CO concentrations in liver, muscle and skin tissues taken from individual chickens, revealed DDC-CO concentrations were highest in chicken liver, followed by chicken muscle, similar to data reported recently for chicken in China

(Zheng et al., 2014). Additionally, concentrations of BTBPE in avian (watercock) and fish samples taken from an e-waste processing area in southern China ranged between 0.07-2.41 and <0.012-0.15 ng/g lw, respectively (Shi et al., 2009). These values are exceeded substantially in our chicken and fish samples. In contrast, concentrations of DBDPE in avian muscle and fish samples in our study are similar to those reported previously (Shi et al., 2009).

In samples originating from areas not impacted by e-waste processing activities, concentrations of HBB, BTBPE, and DBDPE were determined in a selection of UK and Irish food samples (Fernandes et al., 2010). Only BTBPE was detectable in this earlier study, at concentrations of 0.96, 0.75, 0.29, and 0.55 ng/g lw in fish, chicken liver, and chicken eggs, respectively (Fernandes et al., 2010), which are substantially lower than those detected in our study. Compared to the presence of BTBPE in UK food samples, BTBPE was not detected in control samples collected in Vietnam and Japan.

### 3.2.2 Concentrations in soil

In co-located soil samples, concentrations of HBB, BEH-TEBP, BTBPE, DBDPE, *syn*-DDC-CO, and *anti*-DDC-CO were in the range 0.15-21, <0.050-0.4, 0.19-34, 0.42-64, 0.20-13, and 0.83-31 ng/g dw, respectively. The detection frequency was 100% except for BEH-TEBP which was detected in only 20% of samples. PBBz was not detected in any soil samples in this study. DBDPE was the most dominant compound in soil, accounting for around 50% of the total EFRs in our study.

Concentrations of EFRs in soil in our study exceed (DBDPE and BTBPE) or are comparable with (DDC-CO) those detected in soils taken from locations in China surrounding but not directly impacted (e.g. close to workshops) by e-waste processing (Shi et al., 2009; Yu et al., 2010). Moreover, concentrations of ΣDDC-CO in soil in this study exceeded by 3 orders of magnitude those found in soils from residential, business and industrial areas in northern China (Ma et al., 2011). In contrast, the concentration of ΣDDC-CO in a single surface soil directly impacted (i.e. close to a workshop) by e-waste recycling in South China was 3,327 ng/g dw (Yu et al., 2010)- 2 orders of magnitude higher than the average concentration detected in our study. Similarly elevated concentrations of DDC-CO (5,900-10,000 ng/g dw) have also been reported in soil samples directly impacted by e-waste activities in Qingyuan county (Zheng et al., 2014).

## 3.3 Concentrations and diastereomer patterns of HBCDDs in food samples and co-located soil samples

### 3.3.1 HBCDDs in food

Concentrations of HBCDDs in food samples in this study are shown in Table 3.1. HBCDDs were detected in all chicken tissues, river fish, pork, and soil samples. In chicken and fish samples, the levels of HBCDDs tend to be higher than those of EFRs except DDC-CO while in pork samples the levels of HBCDDs were comparable to those of EFRs in line with reports by Labunska et al. (2015) and Zheng et al.(2012). While HBCDDs are known to be mainly used in polystyrene foam and fabrics, they were used to a minor extent in electrical equipment housing (Alaee et al., 2003). This latter minor application, could explain the elevated levels of HBCDDs in this study, as TVs, DVDs, computers and printer housings were processed on a large scale in Bui Dau. Coupled with their environmental stability, persistence and past high production volume (Covaci et al., 2006), these factors may account for the levels of HBCDDs in this study exceeding those for most of the EFRs monitored. In contrast, HBCDDs were not detected in pond fish. This study's finding that HBCDD concentrations in e-waste-impacted samples exceed those in corresponding controls differs to that of Labunska et al. (2015) who reported that HBCDD concentrations in some control samples exceeded those in samples derived from e-waste-impacted locations. To our knowledge, very few data exist about concentrations of HBCDDs in foods reared in locations where unregulated e-waste processing is conducted. Average concentrations of ΣHBCDDs in chicken liver (3,200 ng/g lw) and egg (3,600 ng/g lw) in our study exceeded substantially those found in chicken liver (42.5 ng/g lw) and eggs (42.6 ng/g lw) from an e-waste processing area in Taizhou City (Labunska et al., 2015) as well as in chicken eggs (44.2-350 ng/g lw) from another e-waste processing site in South China (Qingyuan City) (Zheng et al., 2012). Moreover, HBCDDs concentrations (0.59-670 ng/g ww) in food samples in this study exceed markedly those detected in similar foodstuffs around the world including China (<LOD to 9.2 ng/g lw)(Shi et al., 2009), the USA (12-616 ng/g lw) (Schecter et al., 2008), Romania (0.04-0.25 ng/g ww) (Dirtu and Covaci, 2010), Sweden (0.005-0.63 ng/g ww) (Törnkvist et al., 2011), Belgium (<0.01-0.35 ng/g ww) (Roosens et al., 2009a), and the UK (0.02-0.30 ng/g ww) (Driffield et al., 2008). As shown in Table 3.1, HBCDD concentrations in animal-related food sampled from the e-waste processing site in Vietnam varied substantially between species and different chicken tissues. The highest concentrations were found in chicken eggs, followed by chicken liver, chicken skin and chicken muscle; with concentrations in fish and pork samples much lower than those from chickens. Such interspecies differences indicate that the uptake and metabolism of HBCDDs is organism-dependent.

### 3.3.2 Concentrations in soil

The mean ΣHBCDD concentration in soil in this study was 120 ng/g dw, varying from 0.030 to 580 ng/g dw, comparable to HBCDD concentrations in surface soils from e-waste processing areas in South China, ranging from 0.38-284 ng/g dw (Gao et al., 2011). In contrast, concentrations of HBCDDs in soil from the vicinity of HBCDD production facilities in Sweden, Belgium, Germany and China (111-23,200 ng/g dw) exceed significantly those in this study; while those in soils from urban Guangzhou (1.7-5.6 ng/g dw) and from open waste dumping sites in India, Vietnam, Malaysia, Indonesia, and Cambodia (< nd to 2.4 ng/g dw) were at the low end of the range detected in our study (Gao et al., 2011; Li et al., 2012).

The HBCDD diastereomer profiles detected in foodstuffs and co-located soils in this study are shown in Figure 3.1 along with the profile reported for the HBCDD commercial formulation (Law et al., 2006b). γ-HBCDD was the dominant isomer in soil samples in this study, similar to profiles observed in commercial technical products and related abiotic environmental matrices such as sediment, soil and sewage sludge (Covaci et al., 2006). However, in all food samples (whether sourced from e-waste impacted or control locations), α-HBCDD predominated, in line with previous data for biota (Covaci et al., 2006; Reistad et al., 2006). Furthermore, α-HBCDD was relatively more abundant in chicken egg, muscle, and skin than in liver, indicating tissue-specific variation in the relative abundance of different diastereomers, whereby γ-HBCDD is more prevalent in liver samples than the other tissues studied. As

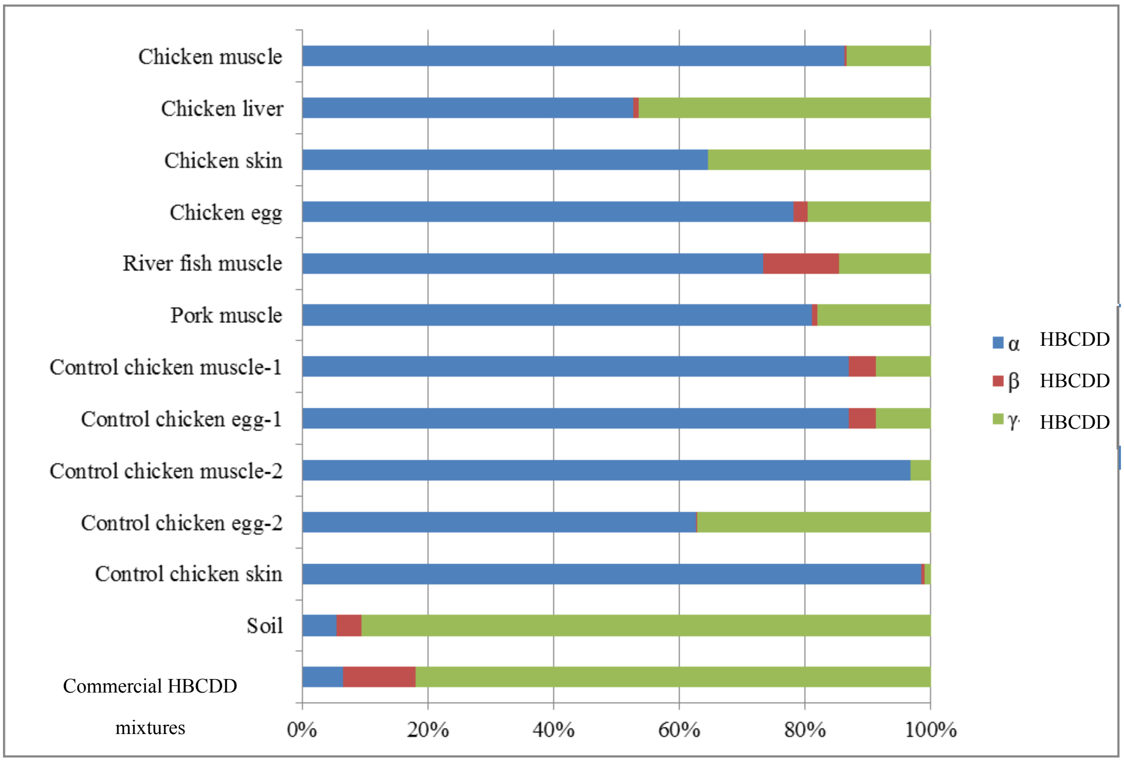


**Figure 3.1 Diastereomer profiles in food and co-located surface soil samples from e-waste processing areas in Vietnam in this study and commercial HBCDD mixtures (Covaci et al., 2006)**

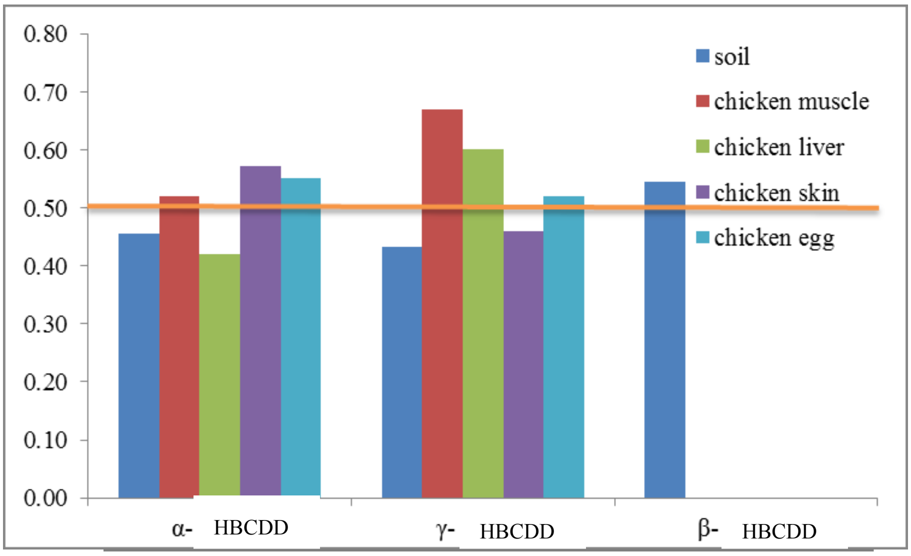


**Figure 3.2 Enantiomer fractions (EF) of α-HBCDD, γ-HBCDD and β-HBCDD in chicken and co-located soil samples (line at EF=0.50 indicates racemic value)**

highlighted previously (Labunska et al., 2015), we believe the diastereomer pattern in avian liver samples reflects more closely the pattern present in its diet and soil, as liver is the first organ exposed following the gastrointestinal tract. In contrast, other avian tissues display a pattern more influenced by metabolism post-exposure.

The α-, β-, and γ-HBCD diastereoisomers are chiral and thus may exist in the environment and biota as enantiomeric pairs (Janak et al., 2005). The enantiomeric composition was expressed as enantiomer fractions (EFs) calculated by the following formula (de Geus et al., 2000):

$$EF = \frac{(+)\mathrm{A}}{(+)\mathrm{A} + (-)\mathrm{A}}$$

where (+)A and (-)A are the peak areas of the corresponding (+) and (-) enantiomers, respectively.

Figure 3.2 shows the EF values for α-, β-, and γ-HBCDD in chicken and co-located soil samples. Average EF values in our soil samples were 0.46, 0.53, and 0.54 for α-, β-, and γ-HBCDD, respectively, compared with those in commercial HBCDD (0.514, 0.510, and 0.503) (Gao et al., 2011). This slight deviation from racemic suggests some edaphic enantioselective degradation of HBCDDs, consistent with an earlier study that reported enantioselective biodegradation of (+)-α-HBCDD in soils (He et al., 2010). In contrast, in soil samples from e-waste recycling areas and industrial areas in South China, negligible enantioselective degradation was implied by mean EF values ranging from 0.503 to 0.507, 0.494 to 0.506, and 0.502 to 0.511 for α-, β-, and γ-HBCDD, respectively (Gao et al., 2011).

With respect to biotic matrices, in this study, (+)-α-HBCDD was clearly enriched in chicken skin and egg while (-)-α-HBCDD dominated in chicken liver. As proposed above to explain the different diastereomer profile detected in chicken liver, the different enantiomer profile observed in the liver may reflect the profile to which the bird is exposed, while that in skin and egg may reflect *in vivo* enantioselective processing post-exposure. To the best of our knowledge, no studies have been reported to investigate the enantioselectivity of α-HBCDD enantiomers in all three of these chicken tissue types examined here. However, in an earlier study by our group of chicken liver, eggs and muscle tissue from e-waste impacted locations in Taizhou, China (Labunska et al., 2015), while (-)-α-HBCDD was enriched in all three tissue types, the enrichment was markedly greater in liver than in egg or muscle tissue – an observation not inconsistent with the present study.

Other studies have reported EFs of HBCDDs in birds. He et al. (2010) reported that spotted dove and Chinese francolin displayed EF values enriched in the (-)-α-enantiomer, while Chinese pond heron and its main prey (fish) displayed relatively more (+)-α-HBCDD in an e-waste region in South China. Similarly, Janak et al. found peregrine falcon eggs and common tern eggs were enriched in (-)-α-HBCDD, while white-tailed sea eagle eggs were depleted in the same enantiomer. Chicken muscle, egg, and liver in our study displayed relative enrichment of (+)-γ-HBCDD, consistent with previous reports for chicken eggs and muscle from e-waste recycling sites in eastern China (Labunska et al., 2015), bird samples from an e-waste area in South China (He et al., 2010) and predatory birds' eggs from Sweden and the Netherlands (Janák et al., 2008). In summary, our findings confirm the complex species and tissue-specific variations that exist in the enantioselective behaviour of HBCDDs in birds.

## 3.4 Relationships between concentrations of HFRs in chicken and co-located soil samples

Significant linear positive correlations were found between concentrations of *syn*-DDC-CO in soils and those in co-located chicken muscle, liver, and eggs ($p<0.05$, $r^2$= 0.921, 0.925, 0.928, respectively for muscle, liver, and egg). Similar correlations were found for *anti*-DDC-CO ($p<0.05$, $r^2$= 0.876, 0.879, 0.885, respectively for muscle, liver, and egg). Combined, these observations suggest that at our sampling sites, soil is an important source of DDC-COs in chickens, consistent with the findings of Zheng et al. (2014). This is further substantiated by comparison of values of the fraction of *anti*-DDC-CO ($f_{anti}$ – the fractional contribution of *anti*-DDC-CO to ΣDDC-CO (sum of *anti*- and *syn*-DDC-CO)) in soil and co-located chicken tissue samples. Observed $f_{anti}$ values ranged from 0.67-0.81 in soil, compared to 0.67-0.82 in chicken tissues and 0.65-0.80 in technical DDC-CO products (Qiu et al., 2007; Zheng et al., 2012). A slight caveat to this conclusion is the fact that significant correlation was not observed between concentrations of either DDC-CO isomer in chicken skin samples and co-located soil samples. With respect to other HFRs, the potential importance of soil as a source of BTBPE contamination in chickens at our sites is indicated by the observation of significant correlations between its concentrations in soil and in co-located samples of chicken liver ($r^2=0.985$, $p<0.01$) and muscle ($r^2=0.909$, $p<0.05$). Conversely, no such correlations were detected between BTBPE concentrations in soil and those in chicken skin and eggs. Similarly, no significant correlations were found between concentrations of any HBCDD isomer in any chicken tissue and soil, which suggests substantial metabolism and/or that food and air could be more important influences on the HBCDD concentrations in chickens.

## 3.5 Estimated daily intake of EFRs and HBCDDs via consumption of foodstuffs included in this study

Very few studies have estimated human dietary exposure to EFRs. Estimated dietary exposure of the sum of our target EFRs for adults and children in this study were 170 and 420 ng/kg bw/day, respectively. The health effects of this exposure cannot be assessed due to the current lack of health based limit values for EFRs exposure. We have, however, compared our estimates with those reported previously. DDC-COs account for >90% of estimated exposure for both adults and children (130 and 350 ng/kg bw/day, respectively) followed by BTBPE (31 and 61 ng/kg bw/day, respectively), while EH-TBB predominated in one study of dietary exposure of the population living in the vicinity of e-waste impacted sites in eastern China, in which DDC-COs were not investigated (Labunska et al., 2015). Another study calculated average estimated daily intakes of EFRs via eggs from one South China recycling area to range from 970 to 4,530 ng/day (Zheng et al., 2012), which is higher than our estimate of exposure via egg ingestion of 350 ng/day. Furthermore, the same study concurred with our finding that DDC-CO was the dominant contributor to EFRs exposure via egg ingestion.

Daily dietary exposure to HBCDDs of individuals living in an e-waste impacted area in this study was estimated at 480 and 1500 ng/kg bw/day for adults and children, respectively. This exceeds estimated dietary exposure to HBCDDs in e-waste impacted locations in China (10.4 and 36.1 ng/kg bw/day for adults and children) (Labunska et al., 2015) and is substantially in excess of estimated fish-related dietary exposure in the Netherlands and Sweden (0.12 and 0.14 ng/kg bw/day, respectively) (Törnkvist et al., 2011; van Leeuwen and de Boer, 2008) as well as estimated dietary exposure of non-e-waste impacted populations in Spain, Belgium and China (2.58, 0.99, and 0.432 ng/kg bw/day, respectively) (Eljarrat et al., 2014; Goscinny et al., 2011; Shi et al., 2009). However, as stated above, the main food supplies for the community in Bui Dau are from neighbouring communities. As a consequence, our assumption that Bui Dau inhabitants source all their fish, chicken meat, liver, and eggs from e-waste impacted sites and thus our estimates of the daily intake of EFRs and HBCDDs via consumption of such foodstuffs represents a worst case – albeit not wholly unrealistic – scenario (Table 3.2).

For most of our target EFRs and HBCDDs, the main contributors to dietary exposure of both adults and children in our study were chicken liver and chicken eggs while fish was the predominant contributor to dietary exposure to BTBPE in our study (Figure 3.3 and 3.4). In previous studies it has been reported that fish, seafood, meat and meat products are the principal contributors to HBCDD dietary exposure (Eljarrat et al., 2014; Goscinny et al., 2011; Shi et al., 2009; Törnkvist et al., 2011). We are aware of only one previous study of dietary exposure to EFRs in e-waste impacted areas (Labunska et al., 2015). In that study, consumption of pork was the principal contributor to dietary exposure of both adults and children to EH-TBB, BEH-TEBP, and BTBPE (Labunska et al., 2015).

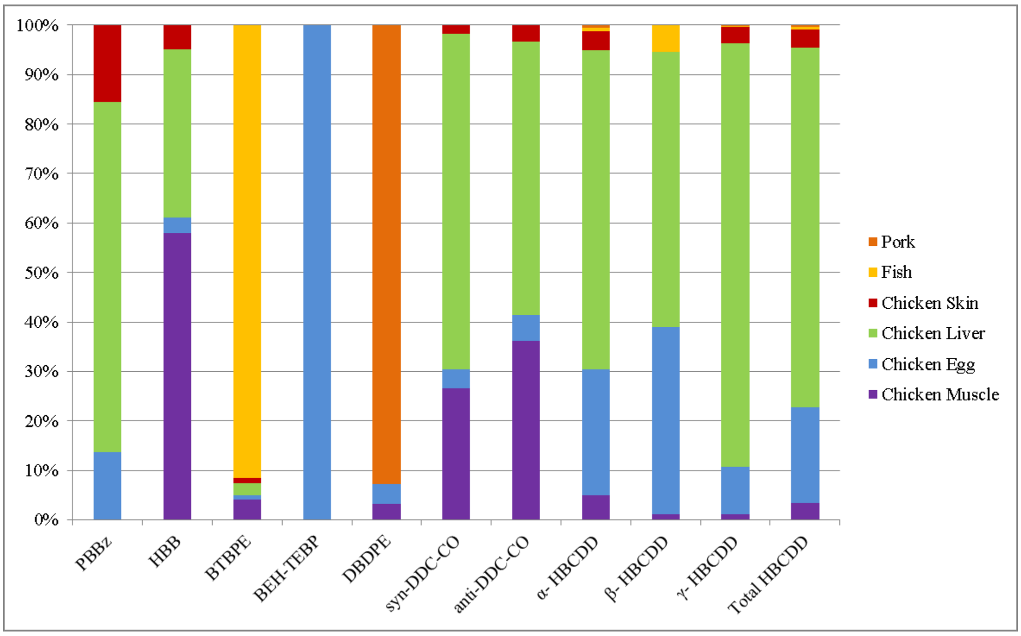


Figure 3.3 Contributions of different food groups to total dietary exposures to EFRs and HBCDDs of children in Bui Dau, Vietnam

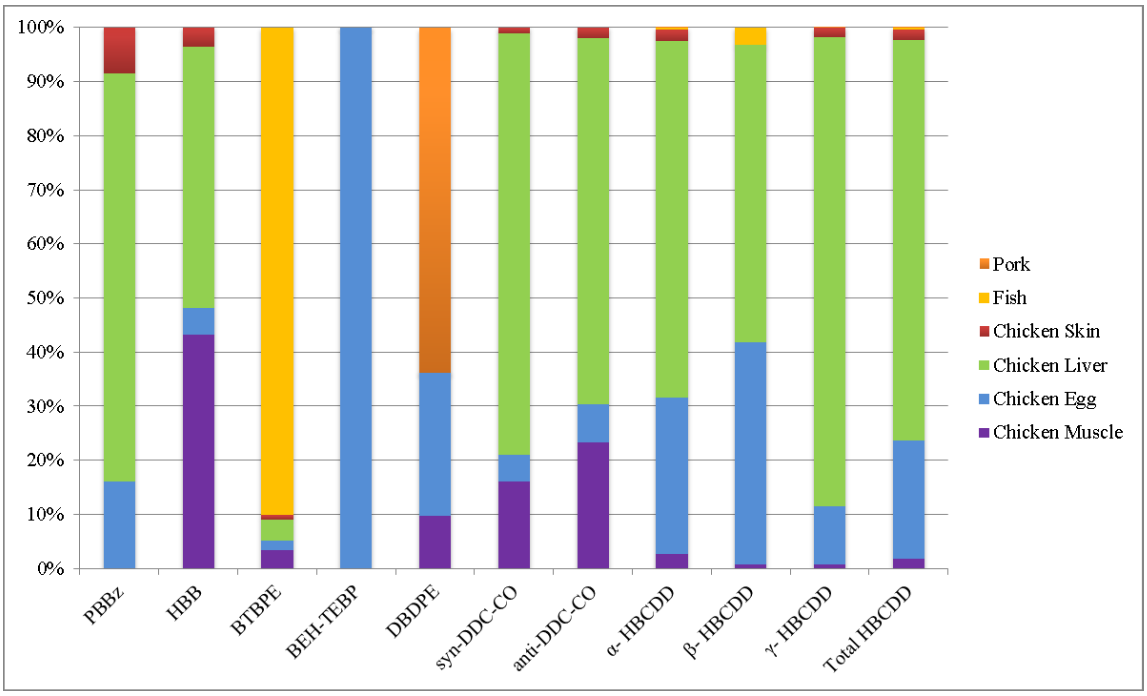


Figure 3.4 Contributions of different food groups to total dietary exposures to EFRs and HBCDDs of adults in Bui Dau, Vietnam

**Table 3.2 Estimated dietary exposure (ng/kg bw/day) to EFRs and HBCDD for adults and children living in e-waste impacted areas of Bui Dau, Vietnam. ne = not estimated**

| Foodstuff | Exposed group/HFR | PBBz | HBB | BTBPE | BEH-TEBP | DBDPE | *syn*-DDC-CO | *anti*-DDC-CO | α-HBCDD | β-HBCDD | γ-HBCDD | ΣHBCDD |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Chicken Muscle | Adult | ne | 0.17 | 1.2 | ne | 0.16 | 11 | 32 | 14 | 0.06 | 2.2 | 16 |
| | Children | ne | 0.28 | 2.1 | ne | 0.26 | 19 | 54 | 23 | 0.10 | 3.6 | 27 |
| Chicken Egg | Adult | 0.010 | 0.010 | 0.28 | 0.010 | 0.20 | 1.7 | 5.0 | 73 | 2.1 | 18 | 93 |
| | Children | 0.020 | 0.030 | 0.97 | 0.040 | 0.71 | 5.8 | 16 | 250 | 7.1 | 63 | 320 |
| Chicken Liver | Adult | 0.030 | 0.10 | 0.75 | ne | ne | 29 | 50 | 180 | 3.0 | 160 | 350 |
| | Children | 0.090 | 0.31 | 2.4 | ne | ne | 93 | 157 | 580 | 9.6 | 510 | 1100 |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Chicken Skin | Adult | 0.010 | 0.010 | 0.35 | ne | ne | 0.76 | 2.9 | 11 | ne | 6.2 | 17 |
| | Children | 0.010 | 0.020 | 0.58 | ne | ne | 1.3 | 4.8 | 18 | ne | 10 | 29 |
| Fish | Adult | ne | ne | 28 | ne | ne | ne | ne | 1.8 | 0.29 | 0.35 | 2.4 |
| | Children | ne | ne | 55 | ne | ne | ne | ne | 3.4 | 0.56 | 0.68 | 4.7 |
| Pork | Adult | ne | ne | ne | ne | 4.6 | ne | ne | 1.7 | ne | 0.37 | 2.0 |
| | Children | ne | ne | ne | ne | 1.7 | ne | ne | 0.61 | ne | 0.14 | 0.75 |
| Total | Adult | 0.040 | 0.29 | 31 | 0.010 | 5.0 | 43 | 90 | 290 | 5.0 | 190 | 480 |
| | Children | 0.12 | 0.64 | 61 | 0.040 | 2.7 | 120 | 230 | 880 | 17 | 590 | 1500 |

This chapter contains some sections of verbatim text adapted from Tao et al. (2016) published as part of this PhD. The author’s contribution: writing, contribution of ideas and numerical calculation.

# CHAPTER 4 EFRs, PBDEs and HBCDDs in UK indoor air and indoor dust

## 4.1 Synopsis

Concentrations of EFRs, PBDEs and HBCDDs were determined in indoor air and dust samples taken from offices and homes in Birmingham, UK. The manufacture and new use of PBDEs and HBCDDs has been banned or restricted in recent years (Newton et al., 2015). We thus hypothesise that this may result in increased production, use and thus elevated levels of EFRs and decreased levels of "legacy" FRs in the environment. Given the similarity in physicochemical properties between EFRs and "legacy" FRs, it has been suggested that their environmental behaviour may also be similar and that human exposure pathways to EFRs will mimic those of their legacy counterparts, with indoor exposures playing an important role. In this study, 16 EFRs were measured in samples of air and settled floor dust from a variety of indoor environments in the UK. In the same samples, concentrations were also measured of 8 PBDEs and 3 HBCDD diastereomers. Data for these legacy FRs were compared with those reported in previous UK studies to test the hypothesis that the aforementioned restrictions have been effective in reducing indoor contamination. Concentrations in this study are also compared with those for other countries, and with recent data for outdoor air in the West Midlands.

## 4.2 Sampling strategy

Air samples were collected in offices (n=20) and houses (n=15) using passive air sampling techniques in Birmingham, UK from February to May 2015. Each passive sampler was deployed for around 45 days around a height of the human breathing zone (see section 2.3.1 for details of sampling methodology).

Dust samples were collected in offices (n=42) from June 2013 to May 2015. With respect to houses, both living room and bedroom dust samples were collected in 15 houses, making 30 dust samples overall. Samples were collected under normal office and house use conditions to reflect actual human exposure (see section 2.3.2 for details of sampling methodology).

## 4.3 Concentration of EFRs, PBDEs and HBCDDs in indoor air

Table 4.1 and Table 4.2 summarise the concentrations of EFRs, PBDEs and HBCDDs in indoor air samples from homes and offices. All 16 EFRs, 8 PBDEs, and 3 HBCDD diastereomers were detected in indoor air.

### 4.3.1 EFRs

In both homes and offices, the more volatile EFRs included in this study (DBE-DBCH, TBX, PBBz, TBCT, PBT, PBEB, TBP-DBPE and HBB) were detected frequently in indoor air with detection frequencies (DF) exceeding 60%. This is likely attributable to a combination of greater indoor use of these chemicals coupled with their relatively higher vapour pressures and lower $K_{OA}$ values. The most abundant compounds in air were α-DBE-DBCH and β-DBE-DBCH, which when summed as ΣDBE-DBCH account for 63% and 80% of ΣEFRs in homes and offices, respectively. The median concentrations of ΣDBE-DBCH in homes and offices were 110 and 290 pg/m$^3$, respectively, which is higher than those in Norwegian households (77.9 pg/m$^3$) and classrooms (46.6 pg/m$^3$) (Cequier et al., 2014) and in Swedish offices, apartments, stores and schools (55 pg/m$^3$) (Newton et al., 2015). To the best of our knowledge, only one commercial product of DBE-DBCH (Saytex BCL-462) has been reported, which contains equal amounts of the two diastereomers, α- and β-DBE-DBCH (Arsenault et al., 2008). The isomeric ratio of β- to α-DBE-DBCH (expressed as $f_{\beta\text{-DBE-DBCH}}$) in this study ranged from 0.53 to 1.0, with a median value of 0.73 which is lower than in the commercial product ($f_{\beta\text{-DBE-DBCH}} = 1$). To the best of our knowledge, this is the first report of the ratio of β- to α-DBE-DBCH in indoor air. Possible explanations for the different diastereomer pattern in air compared to the commercial product include: differences in physicochemical properties (e.g. vapour pressure) between the two diastereomers, and possible isomerisation post-emission. The latter explanation appears unlikely, as thermal conversion of the DBE-DBCH isomers occurs at or above 125 ˚C (Arsenault et al., 2008). In contrast, such high temperatures occur during the incorporation of flame retardant additives like DBE-DBCH into polymeric products. Consequently, the $f_{\beta\text{-DBE-DBCH}}$ values observed in indoor air in this study may actually reflect the pattern present in DBE-DBCH-treated products rather than that present in the commercial Saytex product.

Other frequently detected EFRs were BTBPE, EH-TBB and BEH-TEBP with DFs above 70% in both microenvironment categories in this study. In contrast, DFs for these three compounds in Norwegian and Swedish indoor air were less than 33% (Cequier et al., 2014; Newton et al., 2015). Moreover, BTBPE was not detected in either of these previous studies (Cequier et al., 2014; Newton et al., 2015). No production information is available for the UK or for the EU overall. Both EH-TBB and BEH-TEBP have been reported to be replacements for Penta-BDE, and BTBPE a replacement for Octa-BDE (Covaci et al., 2011). It is therefore possible that after the phase out of "legacy" FRs, these EFRs were more widely used in the UK than Norway and Sweden as a result of the more stringent fire safety regulations in the UK (Harrad and Abdallah, 2011).

### 4.3.2 PBDEs

Of all PBDEs analysed, BDEs 28, 47, 100, 99, and 209 were more frequently detected (DFs >85%) than other PBDEs. Concentrations of ∑PBDEs (gas + particle phases) ranged from 36 to 6400 pg/m$^3$ with a median value of 280 pg/m$^3$ in homes which is comparable to studies conducted in USA (210-3980 pg/m$^3$) and Sweden (72-1400 pg/m$^3$) but higher than Germany (8.24-47 pg/m$^3$), Australia (0.5-179 pg/m$^3$) and Japan (17-55 pg/m$^3$) (Fromme et al., 2009; Johnson-Restrepo and Kannan, 2009; Takigami et al., 2009; Thuresson et al., 2012;

## Table 4.1 Descriptive Statistics for Concentrations of EFRs in UK indoor air (pg/m$^3$)

| Statistical Parameter | α-DBE-DBCH | β-DBE-DBCH | TBX | PBBz | TBCT | PBT | PBEB | TBP-DBPE | HBB | EH-TBB | BTBPE | BEH-TEBP | syn-DDC-CO | anti-DDC-CO | TBBPA-BDBPE | DBDPE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Birmingham, UK, this study,<br>Homes, n=15** | | | | | | | | | | | | | | | | |
| **DF** | 100% | 100% | 100% | 93% | 60% | 100% | 100% | 93% | 73% | 100% | 73% | 93% | 7% | 20% | 20% | 40% |
| **5th percentile** | 20 | 15 | 4.0 | 0.70 | <0.10 | 3.0 | 0.47 | 0.46 | <0.20 | 0.14 | <1.0 | 0.36 | <2.0 | <1.2 | <10 | <10 |
| **95th percentile** | 260 | 210 | 120 | 14 | 83 | 48 | 3.7 | 10 | 38 | 17 | 41 | 40 | 2.1 | 8.3 | 57 | 86 |
| **Mean** | 99 | 74 | 31 | 6.6 | 22 | 17 | 1.6 | 3.5 | 11 | 4.8 | 11 | 10 | <2.0 | 2.2 | 13 | 26 |
| **Median** | 64 | 45 | 9.7 | 5.3 | 9.9 | 11 | 1.3 | 2.0 | 4.2 | 2 | 5 | 2.1 | <2.0 | <1.2 | <10 | <10 |
| **Minimum** | 17 | 13 | 1.6 | <0.10 | <0.10 | 2.3 | 0.41 | <0.40 | <0.20 | 0.05 | <1.0 | <0.10 | <2.0 | <1.2 | <10 | <10 |
| **Maximum** | 350 | 250 | 190 | 22 | 240 | 63 | 5.4 | 14 | 91 | 44 | 50 | 130 | 4.6 | 20 | 87 | 97 |
| **Geometric Mean** | 68 | 50 | 14 | 4.1 | 1.6 | 11 | 1.3 | 2.0 | 2.3 | 1.7 | 4.1 | 1.9 | <2.0 | 0.92 | 7.4 | 13 |
| **Birmingham, UK, this study,<br>Offices, n=20** | | | | | | | | | | | | | | | | |
| **DF** | 100% | 100% | 100% | 100% | 65% | 65% | 100% | 85% | 85% | 100% | 100% | 90% | 5% | 5% | 5% | 5% |
| **5th percentile** | 79 | 58 | 3.6 | 4.6 | <0.10 | <0.10 | 0.41 | <0.40 | <0.20 | 0.82 | 0.70 | <0.10 | <2.0 | <1.2 | <10 | <10 |
| **95th percentile** | 410 | 240 | 36 | 42 | 9.8 | 30 | 17 | 56 | 61 | 80 | 100 | 7.8 | 1.3 | 1.8 | 7.3 | 7.5 |
| **Mean** | 180 | 140 | 16 | 16 | 1.6 | 15 | 4.2 | 18 | 19 | 22 | 32 | 2.2 | 1.3 | 1.8 | 7.3 | 7.5 |
| **Median** | 160 | 120 | 14 | 11 | <0.10 | 4.7 | 1.7 | 1.9 | 9.4 | 5.3 | 11 | 1.4 | <2.0 | <1.2 | <10 | <10 |
| **Minimum** | 74 | 41 | 2.6 | 3.1 | <0.10 | <0.10 | 0.31 | <0.40 | <0.20 | 0.67 | 0.70 | <0.10 | <2.0 | <1.2 | <10 | <10 |
| **Maximum** | 440 | 300 | 38 | 47 | 11 | 200 | 35 | 280 | 170 | 240 | 220 | 11 | 7.7 | 24 | 50 | 54 |
| **Geometric Mean** | 160 | 120 | 12 | 12 | 0.16 | 1.4 | 1.8 | 2.2 | 5.1 | 6.5 | 8.5 | 0.89 | 1.1 | 0.72 | 5.6 | 5.6 |

## Table 4.2 Descriptive Statistics for Concentrations of PBDEs and HBCDDs in UK indoor air (pg/m$^3$)

| Statistical Parameter | BDE 28 | BDE 47 | BDE 100 | BDE 99 | BDE 154 | BDE 153 | BDE 183 | BDE 209 | α–HBCDD | β–HBCDD | γ–HBCDD | Σ HBCDDs |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Birmingham, UK, this study,<br>Homes, n=15** | | | | | | | | | | | | |
| **DF** | 100% | 100% | 87% | 100% | 53% | 67% | 27% | 100% | 40% | 47% | 100% | -- |
| **5th percentile** | 0.35 | 0.15 | <0.10 | 0.5 | <0.50 | <0.4 | <1.0 | 41 | <2.6 | <2.2 | 32 | 34 |
| **95th percentile** | 95 | 560 | 200 | 570 | 59 | 160 | 11 | 2300 | 200 | 66 | 810 | 1200 |
| **Mean** | 22 | 120 | 44 | 130 | 14 | 24 | 2.8 | 660 | 43 | 17 | 270 | 320 |
| **Median** | 1.2 | 13 | 1.5 | 12 | 1.2 | 1.8 | <1.0 | 170 | <2.6 | <2.2 | 110 | 110 |
| **Minimum** | 0.21 | 0.15 | <0.10 | 0.05 | <0.50 | <0.4 | <1.0 | 23 | <2.6 | <2.2 | 16 | 19 |
| **Maximum** | 310 | 1700 | 600 | 1700 | 180 | 180 | 12 | 3800 | 400 | 160 | 1400 | 1500 |
| **Geometric Mean** | 1.3 | 1.4 | 1.7 | 12 | 1.2 | 2 | 1 | 250 | 5.8 | 3.6 | 140 | 170 |
| **Birmingham, UK, this study,<br>Offices, n=20** | | | | | | | | | | | | |
| **DF** | 100% | 100% | 85% | 100% | 40% | 55% | 10% | 100% | 59% | 53% | 100% | -- |
| **5th percentile** | 0.82 | 1.1 | <0.10 | 1.7 | <0.50 | <0.4 | <1.0 | 7.7 | <2.6 | <2.2 | 7.3 | 9.7 |
| **95th percentile** | 13 | 270 | 12 | 15 | 2.7 | 1.8 | 1.6 | 260 | 22 | 15 | 200 | 240 |
| **Mean** | 4.3 | 44 | 3.0 | 9.2 | 0.78 | 0.6 | <1.0 | 74 | 8.8 | 5.2 | 55 | 69 |
| **Median** | 2.7 | 6 | 1.5 | 7.9 | <0.50 | <0.4 | <1.0 | 26 | 5.4 | <2.2 | 34 | 41 |
| **Minimum** | 0.81 | 0.15 | <0.10 | 1.2 | <0.50 | <0.4 | <1.0 | 2.3 | <2.6 | <2.2 | 3.1 | 5.5 |
| **Maximum** | 13 | 380 | 17 | 42 | 6 | 3.6 | 3.8 | 350 | 31 | 15 | 320 | 360 |
| **Geometric Mean** | 3.1 | 8.3 | 1.0 | 6.7 | 0.43 | 0.37 | 0.58 | 35 | 4.6 | 2.9 | 32 | 39 |

Toms et al., 2009). The concentrations of ∑PBDEs (gas + particle phases) were in the range of 22-600 pg/m$^3$ with a median value of 54 pg/m$^3$ in offices which is comparable to Australia (15-487 pg/m$^3$) but much lower than USA (21-17200 pg/m$^3$) and Sweden (140-7300 pg/m$^3$) (Batterman et al., 2010; Takigami et al., 2009; Thuresson et al., 2012; Toms et al., 2009). BDE 209 was the dominating congener in homes and offices, contributing 73% and 50% of the total atmospheric PBDEs, respectively. This congener pattern matches broadly that reported in Sweden (Thuresson et al., 2012), Germany (Fromme et al., 2009), Australia (Toms et al., 2009), and Japan (Takigami et al., 2009), in all of which BDE 209 was the dominant congener in homes and offices. In contrast, in the USA, BDE 47 and BDE 99 were the most abundant compounds in homes (Johnson-Restrepo and Kannan, 2009), while BDE 17, BDE 47 and BDE 99 accounted for 72% of ΣPBDEs in offices (Batterman et al., 2010). This different pattern in the USA can be explained by the far more extensive use of Penta-BDE in USA (97.5% of global Penta-BDE demand) than other countries (Hale et al., 2003).

### 4.3.3 HBCDDs

Of the 3 HBCDD isomers monitored, γ-HBCDD was more frequently detected than α-HBCDD and β-HBCDD in indoor air. In homes in this study, concentrations of ∑HBCDDs (gas + particle phases) ranged from 19 to 1500 pg/m$^3$ with a median value of 110 pg/m$^3$ which is comparable with the range and median reported in the only previous UK study (range: 67-1300 pg/m$^3$; median: 180 pg/m$^3$ ) (Abdallah et al., 2008). By comparison, in offices, concentrations in this study of ∑HBCDDs (gas + particle phases) ranged from 5.5 to 360 pg/m$^3$ with a median value of 41 pg/m$^3$. These levels are lower than in offices in the previous UK study (range: 70-460 pg/m$^3$; median: 170 pg/m$^3$) (Abdallah et al., 2008). Outside the UK, concentrations of ∑HBCDDs in offices, apartments, stores and two schools in Sweden (range <1.3-19 pg/m$^3$; median: <1.3 pg/m$^3$) (Newton et al., 2015) were substantially lower than those reported here. γ-HBCDD was the dominant contributor to ΣHBCDD in both homes and offices in this study. On average, the composition is 87% γ-HBCDD, 8% α-HBCDD, and 5% β-HBCDD in homes, while for offices, it is 71% γ-HBCDD, 18% α-HBCDD, and 11% β-HBCD. This diastereomer pattern is similar to the previous UK study (in which the composition in indoor air was 65% γ-HBCDD, 22% α-HBCDD and 13% β-HBCDD) (Abdallah et al., 2008).

### 4.3.4 Relative abundance of different classes of FRs

As shown in Figure 4.1, PBDEs were the predominant FR class monitored in homes, contributing 44 % of ΣFRs. In contrast, EFRs were the principal contributor to ΣFRs in offices, accounting for 83% of ∑FRs. ∑HBCDDs contributed 18% and 8% to ΣFRs in homes and offices, respectively. The comparatively high abundance of EFRs in both offices and

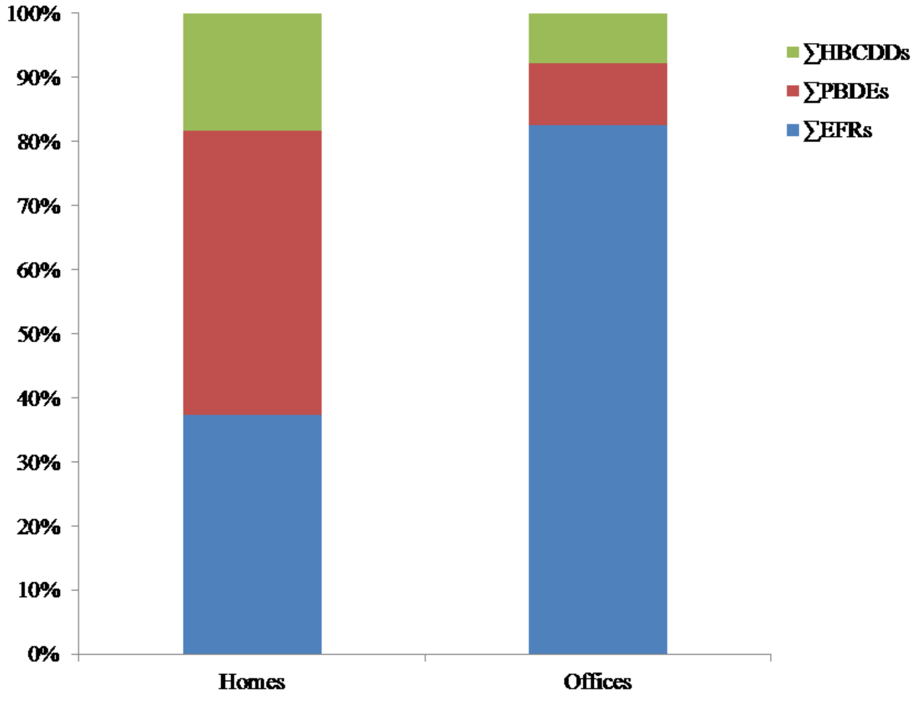


**Figure 4.1 Relative contributions of EFRs, PBDEs and HBCDDs to ΣFRs in UK indoor air**

homes, suggests widespread use of these chemicals in the UK. Particularly noteworthy is the far greater abundance of EFRs in offices. This could be attributable to different putative sources in homes and offices, or reflect a more rapid turnover of such source items in offices. Greater replacement in office environments of old source items containing “legacy” FRs with replacements containing EFRs, is a plausible explanation for the predominance of the latter chemical class in offices.

## 4.4 Concentration of EFRs, PBDEs and HBCDDs in indoor dust

Table 4.3 and Table 4.4 summarise the concentrations of EFRs, PBDEs and HBCDDs in indoor dust samples taken from homes and offices.

### 4.4.1 EFRs

In contrast to indoor air in this study, the less volatile compounds were more frequently detected in office and house dust. These include: DDC-CO, TBBPA-BDBPE and DBDPE (for which DFs all exceeded 60%). Of particular interest is the very high concentration of 4700,000 ng/g BTBPE detected in one UK office dust sample, which to the author’s knowledge is the highest BTBPE level reported to date in indoor dust globally. We are currently unable to give an explanation for the high concentration based on a survey of potential FR-treated items in the sampled office. TBBPA-BDBPE was the most abundant compound, comprising 87% and 71% of ∑EFRs in homes and offices based on median concentrations, respectively, followed by BEH-TEBP, BTBPE, and DBDPE. TBBPA-BDBPE was also the predominant EFR detected previously in UK classroom dust accounting for on average 48% of ΣEFRs (Ali et al., 2011a) indicating extensive use of this compound in the UK. The levels of TBBPA-BDBPE in this study exceeded those in both Belgian homes and offices (Ali et al., 2011a) and USA homes (Dodson et al., 2012). In office dust, levels of BTBPE, BEH-TEBP, and DBDPE in this study were comparable to those reported for offices in Beijing, China (Cao et al., 2014) but exceeded those reported for Belgian offices (Ali et al., 2011a). With respect to house dust, concentrations reported here were comparable to those measured in Belgium, New Zealand, Germany, Sweden, and Norway (Ali et al., 2012a, 2011a; Cequier et al., 2014; Dodson et al., 2012; Fromme et al., 2014; Sahlström et al., 2015) but were lower than those found in USA and Canada (Shoeib et al., 2012; Stapleton et al., 2008). In studies that did not target TBBPA-BDBPE, DBDPE and BEH-TEBP were the most abundant EFRs in house dust from Norway, USA, China, Sweden and Germany (Cequier et al., 2014; Dodson et al., 2012; Fromme et al., 2014; Qi et al., 2014; Sahlström et al., 2015), with similar abundances of DBDPE and BEH-TEBP as was seen in this study. Our data also represent the first report of the presence of DDC-CO in UK indoor dust at median levels of 5.7 and 73 ng/g in home and office dust, respectively. The fraction of ΣDDC-CO contributed by the *anti* DDC-CO isomer (expressed as $f_{anti}$) in the technical mixture has been reported to fall in the range 0.65−0.75 (Cequier et al., 2014). By comparison, $f_{anti}$ values in indoor dust in this study range from 0.27-0.99. This wider range is similar to previous studies of indoor dust from Canada, China and Sweden (Newton et al., 2015; Shoeib et al., 2012; Wang et al., 2011). As discussed above for DBE-DBCH in air, plausible explanations include: isomer-specific degradation, isomer-specific environmental behaviour arising from different physicochemical properties, as well as thermal isomerisation occurring during incorporation of the commercial DDC-CO product into source items.

DBE-DBCH was also detected in all indoor dust samples underlining the extensive use of this compound in the UK. We report here $f_{\beta\text{-DBE-DBCH}}$ for the first time in indoor dust with values falling in the range 0.32-2.88 (median value: 0.85) which is lower than the commercial product (1.0). Possible reasons for the generally lower ratios in dust compared to the commercial Saytex formulation are as described for indoor air in section 4.3.1. In brief, the possible explanations are differences in physicochemical properties between α-DBE-DBCH and β- DBE-DBCH and/or possible isomerisation post-emission. We conducted a paired t-test comparison of $f_{\beta\text{-DBE-DBCH}}$ values obtained for air and dust samples taken in the same rooms. This revealed that $f_{\beta\text{-DBE-DBCH}}$ values of indoor dust are significantly higher than those of indoor air (0.53-1.0) ($p<0.01$). There are very few studies that address differences in the physicochemical properties and environmental behaviour between DBE-DBCH isomers. Wong et al. (2012) reported the fate of DBE-DBCH in urban soil and found that β-DBE-DBCH had a slightly higher soil-air partition coefficient ($K_{SA}$) than α-DBE-DBCH, thereby indicating β-DBE-DBCH to be less volatile than α-DBE-DBCH. This may explain the significantly higher $f_{\beta\text{-DBE-DBCH}}$ in indoor dust than indoor air. Moreover, the slightly faster degradation rate of α-DBE-DBCH compared to β-DBE-DBCH reported by Wong et al. (2012), may also contribute to the observed DBE-DBCH profiles in dust. The fate of DBE-DBCH isomers in environment and elucidating the reason of the difference between diastereomer profiles in air and dust is therefore a research priority.

### 4.4.2 PBDEs

Each of the target individual PBDEs were detected in >90% of indoor dust samples except for BDE 28 and BDE 47. In house dust, concentrations of ∑PBDEs ranged from 180 to 370000 ng/g with a median value of 4600 ng/g. This is higher than concentrations reported from other countries in European and North America such as Germany (36.6-1580 ng/g), Denmark (65-61524), Sweden (53-4000 ng/g), USA (920-17000 ng/g), and Belgium (4-6509 ng/g) as well as Asian countries like China (131.6 to 3886.7 ng/g) but is comparable to those reported in Canada (170-170000 ng/g) (D’Hollander et al., 2010; Fromme et al., 2009; Harrad et al., 2008b; Thuresson et al., 2012; Vorkamp et al., 2011; Wilford et al., 2005; Yu et al., 2012). In office dust, concentrations of ∑PBDEs were 270-110000 ng/g with a median value of 3000 ng/g. This is comparable to USA, Sweden and China but higher than Belgium

# Table 4.3 Descriptive Statistics for Concentrations for EFRs in UK indoor dust (ng/g)

| Location (reference) | α-DBE-DBCH | β-DBE-DBCH | TBX | PBBz | TBCT | PBT | PBEB | TBP-DBPE | HBB | EH-TBB | BTBPE | BEH-TEBP | syn -DDC-CO | anti -DDC-CO | TBBPA-BDBPE | DBDPE |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Birmingham, UK, this study, Homes, n=30** | | | | | | | | | | | | | | | | |
| **DF** | 100% | 100% | 80% | 87% | 40% | 67% | 87% | 84% | 75% | 94% | 100% | 100% | 63% | 84% | 100% | 60% |
| **5th percentile** | 2.0 | 2.0 | <0.010 | <0.010 | <0.010 | <0.010 | <0.010 | <0.050 | <0.030 | <0.010 | 2.4 | 17 | <0.26 | <0.15 | 220 | <1.2 |
| **95th percentile** | 25 | 40 | 32 | 8.8 | 60 | 24 | 10 | 34 | 7.3 | 74 | 57 | 600 | 12 | 120 | 27000 | 1200 |
| **Mean** | 9.4 | 12 | 18 | 3.3 | 21 | 7.1 | 2.3 | 6.6 | 1.8 | 21 | 14 | 240 | 3.6 | 21 | 5800 | 240 |
| **Median** | 5.9 | 6.2 | 1.4 | 2.2 | <0.010 | 1.8 | 0.78 | 1.8 | <0.030 | 10 | 5.6 | 65 | 0.77 | 4.9 | 1000 | 41 |
| **Minimum** | 1.4 | 1.5 | <0.010 | <0.010 | <0.010 | <0.010 | <0.010 | <0.050 | <0.030 | <0.010 | 0.01 | 16 | <0.26 | <0.15 | 71 | <1.2 |
| **Maximum** | 52 | 77 | 410 | 12 | 300 | 90 | 21 | 47 | 12 | 85 | 110 | 3500 | 28 | 170 | 49000 | 2300 |
| **Geometric Mean** | 6.5 | 7.2 | 0.87 | 1.2 | 0.16 | 0.53 | 0.59 | 1.5 | 0.11 | 5.2 | 6.1 | 83 | 0.87 | 4.2 | 1500 | 28 |
| **Birmingham, UK, this study, Offices, n=47** | | | | | | | | | | | | | | | | |
| **DF** | 100% | 100% | 98% | 91% | 67% | 87% | 100% | 98% | 91% | 91% | 100% | 100% | 98% | 98% | 100% | 96% |
| **5th percentile** | 4.1 | 5.2 | 0.53 | <0.010 | <0.010 | <0.010 | 0.49 | 1.4 | <0.030 | 0.0065 | 5.9 | 58 | 1.0 | 3.3 | 480 | 70 |
| **95th percentile** | 52 | 44 | 11 | 11 | 44 | 30 | 6.7 | 130 | 42 | 490 | 20000 | 3600 | 430 | 1000 | 9500 | 8600 |
| **Mean** | 23 | 18 | 3.2 | 4.5 | 11 | 6.0 | 2.0 | 24 | 14 | 120 | 100000 | 1000 | 60 | 210 | 3400 | 1600 |
| **Median** | 15 | 13 | 2.1 | 3.3 | 3.4 | 2.5 | 1.4 | 4.7 | 9.9 | 31 | 160 | 160 | 11 | 62 | 2300 | 440 |
| **Minimum** | 2.5 | 2.7 | <0.010 | <0.010 | <0.010 | <0.010 | 0.36 | <0.050 | <0.030 | <0.010 | 0.019 | 54 | <0.26 | <0.15 | 310 | <1.2 |
| **Maximum** | 130 | 120 | 19 | 23 | 68 | 59 | 10 | 370 | 84 | 2000 | 4700000 | 25000 | 640 | 2100 | 14000 | 17000 |
| **Geometric Mean** | 16 | 14 | 1.7 | 2.0 | 0.54 | 1.3 | 1.4 | 6.1 | 5.6 | 18 | 200 | 250 | 11 | 43 | 2200 | 420 |

(Batterman et al., 2010; D’Hollander et al., 2010; Kang et al., 2011; Thuresson et al., 2012). Similar to indoor air in this study, BDE 209 was the predominant PBDE congener detected, contributing 99% and 94% of ΣPBDEs in homes and offices, respectively. This predominance of BDE 209 is in line with other countries in Europe and in Asia, but is more marked than in North America, where the greater use of Penta-BDE leads to a greater contribution from BDEs 47 and 99 (Batterman et al., 2010; D’Hollander et al., 2010; Fromme et al., 2009; Harrad et al., 2008a; Kang et al., 2011; Thuresson et al., 2012; Vorkamp et al., 2011; Wilford et al., 2005).

### 4.4.3 HBCDDs

All three targeted HBCDDs were detected in every indoor dust sample. Concentrations of ∑HBCDDs ranged from 50 to 110000 ng/g with a median value of 610 ng/g in homes which compares to the concentrations detected in house dust in a previous study in the West Midlands (range: 140-140000 ng/g; median: 1300 ng/g) (Abdallah et al., 2008). In office dust, concentrations of ∑HBCDDs were in the range 150-6400 ng/g with a median value of 1700 ng/g in offices. This again compares closely with previous UK data (range: 90-6600 ng/g; median: 760 ng/g) (Abdallah et al., 2008). The levels of ∑HBCDDs in office and house dust in this study exceeded those reported in dust from offices, apartments, stores and two schools in Sweden (range: 17-2900 ng/g; median: 150 ng/g) (Newton et al., 2015). In our study, α-HBCDD was the dominant contributor to ΣHBCDD in both house and office dust. The average composition is 53 % α-HBCDD, 29 % γ-HBCDD, and 18 % β-HBCDD in homes, while for offices; it is 56 % α-HBCDD, 27 % γ-HBCDD, and 17 % β-HBCDD. This diastereomer pattern differs from that observed in indoor air. To investigate whether the diasteromer pattern in air is statistically significant from that in dust in the same rooms, the fractional contributions of both α-HBCDD and γ-HBCDD to ΣHBCDDs detected in matched pairs (n = 32) of indoor air and dust samples, i.e., collected from the same room at the same time (dust was collected at the end of the air sampling period) were compared. Similar to a previous UK study (Abdallah et al., 2008), the results of an paired t-test revealed significantly greater relative abundance of α-HBCDD in dust compared to matched air samples, with the opposite trend observed for γ-HBCDD ($p < 0.001$). This was attributed to a postdepositional shift from γ-HBCDD to α-HBCDD, and/or preferential degradation of γ-HBCDD in indoor dust (Abdallah et al., 2008).

### 4.4.4 Composition profiles of FRs (including EFRs, PBDEs and HBCDDs)

As shown in Figure 4.2, PBDEs are the predominant FR class monitored in house dust, contributing 66% ΣBFRs, while EFRs dominated in office dust, accounting for 51% of ΣBFRs. ΣHBCDDs contributed 9% and 17% to ΣBFRs in house and office dust, respectively. This is similar to the composition profiles in indoor air and confirms the widespread use of EFRs in the UK, especially in offices.

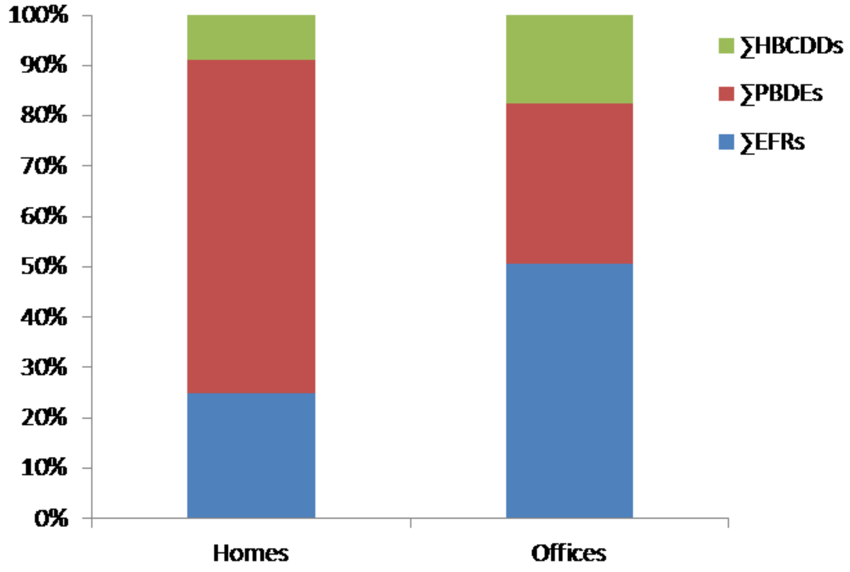


**Figure 4.2 Relative contributions of EFRs, PBDEs and HBCDDs to ΣBFRs in UK indoor dust**

## 4.5 Comparisons between indoor microenvironments

### 4.5.1 Comparisons between indoor microenvironments in indoor air

We compared concentrations in home and office air using an independent t-test. For EFRs, DBE-DBCH, PBBz and EH-TBB were significantly higher in offices than homes ($p < 0.001$ for DBE-DBCH, $p < 0.05$ for PBBz and EH-TBB), with no significant differences between homes and offices detected for other EFRs. Concentrations of BDE 209 were significantly higher in homes than offices ($p < 0.005$); no differences were found for other congeners. In contrast, previous studies on West Midlands indoor air focusing on tri- to hexa-BDEs (BDE 209 not measured) found concentrations in offices to exceed those in homes ($p < 0.05$ Mann-Whitney U-test) (Harrad et al., 2006, 2004). Finally with respect to HBCDDs, concentrations of all 3 isomers were significantly higher in homes than offices ($p < 0.005$ for α-HBCDD, $p < 0.05$ for β–HBCDD, $p < 0.001$ for γ-HBCDD). No such excess of HBCDDs in homes c.f. offices was observed in the previous UK study (Abdallah et al., 2008).

### 4.5.2 Comparisons between indoor microenvironments in indoor dust

As with indoor air, we conducted an independent t-test to check for significant differences in concentrations of our target FRs in home compared to office dust. For EFRs, DBE-DBCH, EH-TBB, BEH-TEBP, BTBPE, DDC-CO and DBDPE were found to be significantly higher in offices than homes ($p < 0.005$ for DBE-DBCH, EH-TBB and DDC-CO, $p < 0.001$ for BTBPE, BEH-TEBP and DBDPE). No other significant differences between offices and homes were found for other EFRs.

In house dust, levels of BDE 209 in homes and offices were comparable ($p > 0.05$), while for BDE 47 and BDE 99, concentrations in offices exceeded significantly ($p < 0.001$) those in homes. This result is in agreement with previous studies of PBDEs in Belgian and UK house and office dust (D'Hollander et al., 2010; Harrad et al., 2008a).

## Table 4.4 Descriptive Statistics for Concentrations of PBDEs and HBCDDs in UK indoor dust (ng/g)

| **Location (reference)** | **BDE 28** | **BDE 47** | **BDE 100** | **BDE 99** | **BDE 154** | **BDE 153** | **BDE 183** | **BDE 209** | **α–HBCDD** | **β–HBCDD** | **γ–HBCDD** | **Σ HBCDDs** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Birmingham, UK, this study, Homes, n=30** | | | | | | | | | | | | |
| **DF** | 57% | 77% | 100% | 100% | 94% | 100% | 90% | 100% | 100% | 100% | 100% | -- |
| **5th percentile** | <0. 03 | <0.04 | 0.99 | 6.5 | 0.35 | 0.025 | <0.13 | 180 | 42 | 11 | 24 | 77 |
| **95th percentile** | 11 | 31 | 10 | 76 | 7.4 | 14 | 33 | 150000 | 10000 | 6000 | 22000 | 38000 |
| **Mean** | 1.9 | 14 | 4.2 | 31 | 2.0 | 4.8 | 7.4 | 34000 | 2300 | 1100 | 4900 | 8300 |
| **Median** | 0.16 | 13 | 3.0 | 22 | 1.2 | 3.0 | 3.5 | 4500 | 320 | 85 | 93 | 610 |
| **Minimum** | <0.03 | <0.04 | 0.75 | 5.0 | <0.06 | 0.025 | <0.13 | 160 | 21 | 6.1 | 23 | 50 |
| **Maximum** | 15 | 50 | 16 | 92 | 9.3 | 24 | 51 | 370000 | 28000 | 12000 | 71000 | 110000 |
| **Geometric Mean** | 0.19 | 4.5 | 3.2 | 24 | 1.2 | 1.6 | 2.9 | 4700 | 340 | 110 | 160 | 670 |
| **Birmingham, UK, this study, Offices, n=47** | | | | | | | | | | | | |
| **DF** | 82% | 100% | 100% | 100% | 100% | 100% | 100% | 100% | 100% | 100% | 100% | -- |
| **5th percentile** | <0.03 | 11 | 2.7 | 19 | 1.2 | 0.025 | 0.66 | 750 | 190 | 54 | 64 | 310 |
| **95th percentile** | 17 | 280 | 61 | 290 | 24 | 92 | 160 | 32000 | 2400 | 510 | 2100 | 4300 |
| **Mean** | 3.9 | 83 | 18 | 100 | 7.7 | 28 | 29 | 9100 | 1100 | 300 | 630 | 2000 |
| **Median** | 2.6 | 37 | 12 | 77 | 3.9 | 9.2 | 9.8 | 2700 | 980 | 330 | 350 | 1700 |
| **Minimum** | <0.03 | 7.1 | 1.9 | 15 | 0.80 | 0.025 | 0.065 | 200 | 100 | 22 | 31 | 150 |
| **Maximum** | 22 | 660 | 120 | 480 | 68 | 190 | 220 | 110000 | 2800 | 590 | 3700 | 6400 |
| **Geometric Mean** | 1.1 | 43 | 11 | 72 | 4.5 | 7.8 | 9.3 | 3300 | 810 | 240 | 360 | 1500 |

For HBCDDs, no significant differences were detected between concentrations of ΣHBCDDs in dust from homes and offices ($p > 0.05$); a finding consistent with the previous study of UK indoor dust (Abdallah et al., 2008).

## 4.6 Comparisons with outdoor air samples

In one study by Drage et al. (2016), DBE-DBCH, DDC-CO, PBDEs and HBCDDs were detected in passive outdoor air samples collected from June 2012 to January 2013 on a rural-urban transect in UK West Midlands. Compared with our study, the levels of DBE-DBCH, BDE 28, 47, 100, 99, 209 and ΣHBCDDs in indoor air exceed significantly those in outdoor air (t-test on log transformed concentrations, $p < 0.05$), suggesting indoor air to be a source of contamination to the outdoor environment for those compounds. The levels of DDC-CO and BDE 154, 153 and 183 in the two studies were not compared as the DFs were low for these FRs in both.

## 4.7 Comparisons with previous studies in the UK

### 4.7.1 EFRs

To our knowledge, these are the first data about concentrations of EFRs in UK indoor air. Consequently, comparison with previous studies is not possible. With respect to indoor dust, however, concentrations of BTBPE and DBDPE in dust taken from houses (n=30) and offices (n=18) in the West Midlands collected between July 2006 and June 2007 have been reported (Harrad et al., 2008a). Following log-transformation of concentrations, we conducted a t-test to compare concentrations in these two studies. For house dust, concentrations of BTBPE in our study were statistically indistinguishable to those in the previous study ($p > 0.05$); in contrast, concentrations of DBDPE exceeded significantly ($p < 0.05$) those in the earlier study. Moreover, t-test comparison revealed concentrations of BTBPE and DBDPE in our office dust samples were both significantly higher than those reported previously ($p < 0.05$) (Harrad et al., 2008a). While based on a relatively limited number of samples, these findings support the hypothesis that restrictions on legacy FRs have led to a concomitant increase in concentrations in UK indoor environments of DBDPE and – to a lesser extent – BTBPE.

### 4.7.2 PBDEs

Two previous studies have reported concentrations of PBDEs in UK indoor air. The first reported concentrations of BDE 47, 99, 100, 153, and 154 in air from a range of office and domestic indoor microenvironments using high-volume active air samplers from 2001-2002 (Harrad et al., 2004); while the second - conducted between September 2003 and November 2005 - measured BDE 28, 47, 49, 66, 85, 99, 100, 153, and 154 using fully sheltered passive air samplers (Harrad et al., 2006). For house air, no significant differences between our study and two previous studies (Harrad et al., 2006, 2004) were found. While for office air, the levels of BDE 47, 99, and BDE 154 were significantly lower than these two previous studies (ANOVA test on log transformed concentrations, $p < 0.05$) (Harrad et al., 2006, 2004). We are aware of the potential difficulties of comparing concentrations derived using different air sampler configurations. Two studies that examined such difficulties are: (a) Melymuk et al. (2012), who measured PBDEs in outdoor air in Toronto, Canada and found no major differences between concentrations derived using passive air samplers and high volume active samples; and (b) Abdallah and Harrad (2010), who found no significant difference between indoor air concentrations of FRs including PBDEs measured using both passive and low volume active samplers. On the basis of these previous studies therefore, we conclude that the influence of the different sampling methods used in the three studies compared here, should not overly obscure the temporal trend.

Concentrations of BDE 209 in UK office dust were significantly lower ($p < 0.05$) than those reported in a previous study of UK offices (Harrad et al., 2008a); while for UK house dust, BDE 209 concentrations were comparable to those in the previous study ($p > 0.05$) (Harrad et al., 2008a), perhaps indicating more rapid removal of “legacy” PBDEs in offices than homes. No significant temporal differences were found for other PBDEs in both house and office dust. The absence of a marked decline for congeners present in the Penta- and Octa-BDE formulations may be attributable to the limited past use of these formulations in the UK.

### 4.7.3 HBCDDs

We compared our data for HBCDDs in indoor air with those in the previous UK study by independent t-test (Abdallah et al., 2008). In domestic air, no significant differences were detected; in contrast concentrations of all 3 diastereomers were significantly lower in office air in our study ($p < 0.001$). This may possibly indicate possible replacement of HBCDDs by EFRs such as DBE-DBCH which is used for housing insulation in expandable polystyrene beads (Newton et al., 2015). Turning to indoor dust, concentrations of γ-HBCDD in house dust were significantly lower in our study ($p < 0.001$) than in the previous study of Abdallah et al. (2008) but not for other diastereomers and ΣHBCDDs. No significant differences were observed for office dust.

## 4.8 Correlations between FRs in indoor microenvironments

Pearson correlation analysis was applied to assess the relationships between the target compounds in this study in indoor air and dust, respectively, (shown in Table 4.5 and Table 4.6) to investigate whether some chemicals shared similar emission sources. Unsurprisingly, α-DBE-DBCH concentrations significantly correlated with those of β-DBE-DBCH in both indoor air and dust samples ($p < 0.001$) and a similar finding was found for HBCDDs diastereomers in indoor air and dust implying the similar source of these diastereomers ($p < 0.05$).  Significant correlations between concentrations of individual PBDEs (BDE 47 to BDE

153) in indoor air were also found in this study ($p < 0.05$) indicating common sources of these congeners. Similarly significant correlations between the same congeners were also observed for both indoor office and house dust ($p < 0.001$) and BDE 183 was also correlated with BDE 47 ($p < 0.005$), BDE 100 ($p < 0.05$), BDE 153 ($p < 0.01$) and BDE 154 ($p < 0.001$). This probably reflects sources of these compounds are from banned commercial PentaBDE and OctaBDE. Additionally, correlation ($p < 0.01$) was obtained for BDE 209 with BDE 154 for indoor dust. PBBz was correlated significantly with PBEB, TBP-DPTE and HBB in indoor air ($p < 0.001$), and similar relationships were also found in indoor dust ($p < 0.05$). HBB also correlated with PBEB and TBP-DPTE in indoor air ($p < 0.001$). HBB and PBBz were also correlated with some PBDEs such as BDE 28, 47 and 99 in air and dust ($p < 0.05$). While some of these observed correlations are hard to rationalise and may be due to chance, those between PBEB, HBB and PBBz may result from a common source resulting from pyrolysis of polymeric BFRs. Conditions that may favour such formation may be encountered during the process of incorporating BFRs into goods. (Buser, 1986; Gouteux et al., 2008; Thoma and Hutzinger, 1987)

## 4.9 Correlations between FR concentrations in air and dust

If semi-volatile organic compounds (SVOCs) partitioning between the gaseous and particulate phases have reached equilibrium under ideal conditions, there should be a strong correlation between their concentrations in air and dust, depending on vapour pressure (Bergh et al., 2011). To investigate this hypothesis, Pearson correlation analysis was conducted to investigate the correlations between FR concentrations in matched air and dust (n=30) (shown in Table 4.7). Significant correlations between concentrations of EFRs in air and dust were found in this study ($p < 0.005$), especially for the more volatile EFRs, e.g., DBE-DBCH, PBT, and TBCT. Moreover, concentrations of BDE 28 and 47 in air and dust were also significantly correlated ($p < 0.05$). The partition coefficient of FRs between dust and air is expressed as $K_{da}$ ($K_{da}= C_{dust}/C_{air}$) ($C_{dust}$ and $C_{air}$ are the concentrations of FRs in dust and air, respectively.). From Figure 4.3, median values of Log $K_{da}$ are linearly correlated with their $K_{oa}$ for EFRs, PBDEs and ΣHBCDDs in homes and offices suggesting that thermodynamic equilibrium conditions have been reached between air and dust for most of the EFRs, PBDEs and ΣHBCDDs. Similar relationships were also found by Cequier et al. (2014) for PFRs and PBDEs in Norway homes and classrooms and Newton et al. (2015) for four BFRs (DBE-DBCH, PBT, HBB and BDE47). Such a relationship between $K_{OA}$ and $K_{DA}$ means that knowledge of concentrations of FRs in either air or dust facilitates accurate prediction of concentrations in the other phase given the $K_{oa}$ of the compound in question. Interestingly, no correlation between Log$K_{da}$ and Log$K_{oa}$ of HBCDD diastereomers were found in this study.  This is consistent with the previously observed post-depositional transformation in dust of HBCDDs including postdepositional shift from γ-HBCDD to α-HBCDD, and/or preferential degradation of γ-HBCDD in indoor dust (Abdallah et al., 2008; Harrad et al., 2009b).

## Table 4.5 Pearson correlations for the concentrations of FRs in air

| | α-DBE-DBCH | β-DBE-DBCH | TBX | PBBz | TBCT | PBT | PBEB | TBP-DBPE | HBB | EH-TBB | BTBPE | BEH-TEBP | BDE28 | BDE47 | BDE100 | BDE99 | BDE154 | BDE153 | BDE209 | α-HBCDD | β–HBCDD | γ-HBCDD |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| α-DBE-DBCH | 1 | | | | | | | | | | | | | | | | | | | | | |
| β-DBE-DBCH | .971** | 1 | | | | | | | | | | | | | | | | | | | | |
| TBX | .015 | -.010 | 1 | | | | | | | | | | | | | | | | | | | |
| PBBz | .273 | .246 | .211 | 1 | | | | | | | | | | | | | | | | | | |
| TBCT | .138 | .190 | .315 | .524 | 1 | | | | | | | | | | | | | | | | | |
| PBT | .253 | .329 | .060 | .284 | .754** | 1 | | | | | | | | | | | | | | | | |
| PBEB | .177 | .229 | .281 | .622** | .571* | .099 | 1 | | | | | | | | | | | | | | | |
| TBP-DBPE | .049 | .116 | .129 | .662** | .320 | .516** | .538** | 1 | | | | | | | | | | | | | | |
| HBB | .049 | .036 | .033 | .744** | .775** | .078 | .656** | .571** | 1 | | | | | | | | | | | | | |
| EH-TBB | .355* | .307 | -.405* | .103 | -.685** | .123 | -.143 | .230 | .021 | 1 | | | | | | | | | | | | |
| BTBPE | -.045 | -.144 | .333 | .006 | -.486 | .074 | -.054 | -.018 | -.302 | -.174 | 1 | | | | | | | | | | | |
| BEH-TEBP | .092 | .095 | .110 | .105 | .390 | .536** | .337 | .427* | .107 | .177 | .173 | 1 | | | | | | | | | | |
| BDE28 | .452** | .370* | -.099 | .456* | -.325 | .006 | .198 | .150 | .293 | .509** | .199 | .120 | 1 | | | | | | | | | |
| BDE47 | -.115 | -.177 | .179 | .182 | .524 | -.091 | .241 | .081 | .117 | -.201 | .238 | .418* | .228 | 1 | | | | | | | | |
| BDE100 | .318 | .297 | .007 | .317 | .625* | .300 | .307 | .294 | .440* | -.045 | -.073 | .286 | .171 | .551** | 1 | | | | | | | |
| BDE99 | .105 | .129 | -.022 | .302 | .681** | .373 | .250 | .293 | .450* | -.166 | -.048 | .243 | .075 | .483** | .816** | 1 | | | | | | |
| BDE154 | .057 | .051 | .190 | .145 | .572 | -.006 | .083 | -.115 | .167 | -.212 | .059 | -.038 | .053 | .258 | .563** | .571** | 1 | | | | | |
| BDE153 | -.078 | -.002 | -.153 | -.159 | .568 | .216 | .067 | .141 | .212 | -.047 | -.214 | .144 | -.029 | .076 | .462* | .379* | .352 | 1 | | | | |
| BDE209 | -.048 | -.083 | .149 | -.164 | .527 | .076 | .180 | -.067 | .227 | -.181 | .032 | .387* | -.168 | .218 | .564** | .205 | .106 | .179 | 1 | | | |
| α-HBCDD | -.030 | .004 | .484 | .233 | .795 | .067 | .815** | -.058 | .744 | -.686* | .089 | -.324 | -.510 | -.394 | .947** | .263 | .791* | .779* | .830** | 1 | | |
| β–HBCDD | -.151 | -.063 | .184 | .778* | .813 | .647 | .804* | .510 | .808 | -.671 | .032 | -.044 | -.603 | -.728 | .808* | .310 | .800 | .703 | .670 | .965** | 1 | |
| γ-HBCDD | .122 | .016 | .159 | -.112 | .407 | -.182 | -.115 | -.329 | .073 | -.190 | -.012 | -.110 | -.225 | -.132 | .471* | .020 | .246 | -.007 | .797** | .773* | .613 | 1 |

**. Correlation is significant at the 0.01 level (2-tailed; light purple); *. Correlation is significant at the 0.05 level (2-tailed; light green).

## Table 4.6 Pearson correlations for the concentrations of FRs in dust

| | α-DBE-DBCH | β-DBE-DBCH | TBX | PBBz | TBCT | PBT | PBEB | TBP-DBPE | HBB | EH-TBB | BTBPE | BEH-TEBP | syn-DDC-CO | anti-DDC-CO | TBBPA-BDBPE | DBDPE | BDE28 | BDE47 | BDE100 | BDE99 | BDE154 | BDE153 | BDE183 | BDE209 | α–HBCDD | β–HBCDD | γ–HBCDD |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| α-DBE-DBCH | 1 | | | | | | | | | | | | | | | | | | | | | | | | | | |
| β-DBE-DBCH | .893** | 1 | | | | | | | | | | | | | | | | | | | | | | | | | |
| TBX | -.019 | -.080 | 1 | | | | | | | | | | | | | | | | | | | | | | | | |
| PBBz | .352** | .386** | .389** | 1 | | | | | | | | | | | | | | | | | | | | | | | |
| TBCT | .038 | .256 | .359* | .494** | 1 | | | | | | | | | | | | | | | | | | | | | | |
| PBT | .070 | .117 | .337* | .309* | .568** | 1 | | | | | | | | | | | | | | | | | | | | | |
| PBEB | .225 | .229* | .507** | .334* | .531** | .280* | 1 | | | | | | | | | | | | | | | | | | | | |
| TBP-DBPE | .293* | .352** | .178 | .603** | .410** | .259 | .358** | 1 | | | | | | | | | | | | | | | | | | | |
| HBB | .228 | .079 | .013 | .338* | .027 | -.208 | .239 | .246 | 1 | | | | | | | | | | | | | | | | | | |
| EH-TBB | .218 | .147 | .153 | .056 | -.093 | .069 | .116 | .166 | .144 | 1 | | | | | | | | | | | | | | | | | |
| BTBPE | .419** | .329** | -.173 | .098 | -.140 | -.162 | .101 | -.057 | .211 | .226 | 1 | | | | | | | | | | | | | | | | |
| BEH-TEBP | .262* | .159 | -.071 | .111 | -.232 | -.081 | .081 | .104 | .219 | .581** | .369** | 1 | | | | | | | | | | | | | | | |
| syn-DDC-CO | .401** | .248* | -.142 | .142 | -.439** | -.195 | .114 | .213 | .335* | .095 | .288* | .350** | 1 | | | | | | | | | | | | | | |
| anti-DDC-CO | .357** | .278* | -.044 | .101 | -.314* | -.210 | .152 | .154 | .332* | .081 | .356** | .360** | .944** | 1 | | | | | | | | | | | | | |
| TBBPA-BDBPE | .153 | .147 | .042 | .333* | .299 | .458** | .288* | .406** | .119 | .257* | .017 | .296* | -.070 | -.027 | 1 | | | | | | | | | | | | |
| DBDPE | .352** | .256* | -.075 | .192 | -.420** | -.339** | .071 | .024 | .387** | .068 | .375** | .477** | .568** | .624** | -.197 | 1 | | | | | | | | | | | |
| BDE28 | .030 | -.023 | .232 | .017 | .044 | .204 | .160 | .244 | .197 | .272 | .134 | -.041 | -.208 | -.058 | .168 | -.269 | 1 | | | | | | | | | | |
| BDE47 | .208 | .184 | .082 | .114 | -.044 | .020 | .192 | .199 | .360** | .231 | .288* | .212 | .084 | .218 | .116 | .217 | .533** | 1 | | | | | | | | | |
| BDE100 | .250* | .194 | -.202 | .104 | -.122 | .057 | .085 | .159 | .258 | .184 | .399** | .250* | .227 | .289* | .116 | .266* | .491** | .893** | 1 | | | | | | | | |
| BDE99 | .170 | .109 | -.235 | .075 | -.192 | .096 | .037 | .113 | .155 | .202 | .406** | .237* | .125 | .184 | .181 | .241* | .524** | .790** | .907** | 1 | | | | | | | |
| BDE154 | .238* | .207 | -.276* | .151 | -.178 | -.068 | .030 | .202 | .269 | .168 | .362** | .312** | .457** | .409** | .046 | .364** | .214 | .736** | .839** | .795** | 1 | | | | | | |
| BDE153 | .108 | .002 | -.231 | -.073 | -.468** | -.315* | -.024 | -.008 | .324* | .145 | .503** | .479** | .496** | .470** | -.039 | .524** | -.002 | .541** | .572** | .562** | .672** | 1 | | | | | |
| BDE183 | .167 | .131 | .006 | .060 | -.077 | -.279* | .034 | .153 | .203 | .208 | .384** | .292* | .286* | .336** | .033 | .293* | .111 | .353** | .273* | .221 | .502** | .546** | 1 | | | | |
| BDE209 | -.091 | -.042 | -.192 | .088 | .109 | .052 | -.153 | .042 | .209 | .051 | .063 | .012 | .075 | .081 | .132 | -.075 | .131 | .182 | .178 | .200 | .295* | .183 | .205 | 1 | | | |
| α–HBCDD | -.006 | .166 | .048 | .176 | .758* | .494* | .093 | .556* | .402 | -.392 | -.198 | -.135 | -.101 | .175 | .417* | -.124 | .116 | .049 | .030 | .006 | -.064 | .265 | -.035 | .273 | 1 | | |
| β–HBCDD | -.045 | .108 | .077 | .087 | .606 | .355 | .068 | .534* | .447 | -.380 | -.240 | -.142 | -.078 | .190 | .358 | -.054 | .163 | -.063 | .026 | .024 | -.069 | .272 | -.107 | .203 | .965** | 1 | |
| γ–HBCDD | .091 | .229 | .098 | .364 | .817** | .581* | .128 | .636** | .194 | -.467* | -.153 | -.207 | .066 | .119 | .399* | -.048 | -.020 | .048 | .031 | .011 | -.029 | .116 | -.084 | .096 | .895** | .870** | 1 |

**. Correlation is significant at the 0.01 level (2-tailed; light purple); *. Correlation is significant at the 0.05 level (2-tailed; light green).

## Table 4.7 Pearson correlations for the concentrations of FRs in matched air (red) and dust (blue) (n=30)

| | α-DBE-DBCH | β-DBE-DBCH | TBX | PBBz | TBCT | PBT | PBEB | TBP-DBPE | HBB | EH-TBB | BTBPE | BEH-TEBP | BDE28 | BDE47 | BDE100 | BDE99 | BDE154 | BDE153 | BDE209 | α–HBCDD | β–HBCDD | γ–HBCDD |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| α-DBE-DBCH | .753$^{**}$ | .686$^{**}$ | .212 | .178 | .092 | .044 | -.021 | -.038 | -.186 | .200 | .128 | .008 | .389$^{*}$ | .024 | .224 | -.045 | .113 | -.130 | -.029 | .249 | -.003 | .279 |
| β-DBE-DBCH | .609$^{**}$ | .581$^{**}$ | .081 | .164 | .396 | .163 | -.031 | -.011 | -.140 | .068 | .056 | .031 | .348$^{*}$ | .047 | .370$^{*}$ | .149 | .273 | .048 | .055 | .568 | .447 | .346 |
| TBX | .009 | -.062 | .774$^{**}$ | .174 | .211 | .019 | .209 | .088 | .173 | -.298 | .374$^{*}$ | .209 | .008 | .262 | .285 | -.020 | .239 | -.035 | .315 | .488 | .161 | .233 |
| PBBz | -.036 | -.050 | .226 | .648$^{**}$ | .361 | .388 | .130 | .290 | .399$^{*}$ | -.151 | .149 | .066 | .190 | .123 | .166 | .236 | .227 | -.057 | -.147 | .446 | .764$^{*}$ | -.139 |
| TBCT | .032 | .016 | .214 | .527$^{*}$ | .883$^{**}$ | .300 | .403$^{*}$ | .248 | .540$^{**}$ | -.440$^{*}$ | .065 | .212 | -.018 | .417$^{*}$ | .584$^{**}$ | .544$^{**}$ | .458$^{*}$ | .440 | .271 | .802 | .880$^{*}$ | .347 |
| PBT | .003 | .051 | .319 | -.068 | .611$^{*}$ | .584$^{**}$ | .029 | .220 | -.094 | -.382$^{*}$ | .147 | .332 | -.198 | .238 | .398$^{*}$ | .299 | .235 | .307 | .216 | .633 | .594 | .085 |
| PBEB | .214 | .232 | .408$^{*}$ | .493$^{*}$ | .224 | .092 | .543$^{**}$ | .309 | .299 | -.239 | .216 | .070 | .213 | .325 | .455$^{*}$ | .227 | .403$^{*}$ | .162 | .047 | .685 | .580 | -.063 |
| TBP-DBPE | .130 | .154 | .193 | .631$^{**}$ | .220 | .292 | .299 | .642$^{**}$ | .365 | -.066 | .067 | .065 | .247 | .192 | .446$^{*}$ | .349$^{*}$ | .224 | .176 | -.127 | .387 | .790$^{*}$ | -.129 |
| HBB | .304 | .230 | -.107 | .523$^{*}$ | -.294 | -.350 | .152 | .156 | .598$^{**}$ | .211 | -.092 | -.059 | .388$^{*}$ | .046 | -.005 | .059 | -.061 | -.090 | -.389$^{*}$ | -.398 | -.311 | -.262 |
| EH-TBB | -.013 | -.117 | -.013 | -.229 | -.665$^{*}$ | -.307 | -.420$^{*}$ | -.260 | -.364 | .282 | .211 | -.034 | .360$^{*}$ | .255 | -.280 | -.283 | -.125 | -.532$^{**}$ | -.256 | -.746$^{*}$ | -.785$^{*}$ | -.153 |
| BTBPE | .338$^{*}$ | .256 | -.011 | .354 | -.347 | -.326 | -.108 | -.065 | -.061 | .228 | .311 | -.196 | .420$^{*}$ | .027 | -.185 | -.170 | .097 | -.213 | -.421$^{*}$ | -.418 | -.536 | -.130 |
| BEH-TEBP | .149 | .089 | -.037 | .056 | -.119 | -.288 | -.096 | .011 | .156 | .299 | -.110 | -.127 | .410$^{*}$ | .110 | -.144 | -.218 | -.093 | -.153 | -.237 | -.358 | -.660 | -.040 |
| BDE28 | -.003 | -.095 | -.155 | .106 | -.432 | -.301 | .133 | .073 | .145 | .285 | .122 | .154 | .441$^{*}$ | .567$^{**}$ | .100 | -.152 | -.122 | .010 | -.058 | -.509 | -.419 | -.183 |
| BDE47 | .287 | .228 | -.112 | .221 | -.322 | -.065 | .101 | -.037 | .095 | .355$^{*}$ | .118 | .039 | .568$^{**}$ | .490$^{**}$ | .140 | -.011 | .135 | -.074 | -.279 | -.562 | -.471 | -.401$^{*}$ |
| BDE100 | .165 | .129 | -.156 | .223 | -.322 | -.080 | .128 | -.018 | .068 | .260 | .067 | -.039 | .469$^{**}$ | .470$^{**}$ | .096 | .010 | .128 | -.078 | -.309 | -.600 | -.432 | -.461$^{*}$ |
| BDE99 | .136 | .108 | -.203 | .211 | -.371 | -.125 | .128 | -.046 | .052 | .252 | .022 | -.078 | .406$^{*}$ | .416$^{*}$ | .056 | -.013 | .141 | -.039 | -.353$^{*}$ | -.675$^{*}$ | -.421 | -.470$^{*}$ |
| BDE154 | .157 | .152 | -.105 | .201 | -.288 | .048 | .025 | .041 | -.077 | .232 | -.022 | -.026 | .349$^{*}$ | .383$^{*}$ | .049 | -.002 | .240 | .001 | -.369$^{*}$ | -.795$^{*}$ | -.619 | -.503$^{*}$ |
| BDE153 | .402$^{*}$ | .344 | -.138 | .181 | -.403 | -.045 | .036 | .011 | -.125 | .332 | .281 | -.160 | .566$^{**}$ | .132 | -.238 | -.192 | -.111 | -.065 | -.448$^{*}$ | -.797$^{*}$ | -.845$^{*}$ | -.471$^{*}$ |
| BDE209 | -.035 | -.016 | .108 | .038 | .010 | .032 | .120 | .169 | -.044 | -.016 | .139 | .184 | -.023 | .055 | -.003 | -.084 | -.081 | -.126 | -.018 | .333 | .396 | -.031 |
| α–HBCDD | .164 | .243 | .080 | .240 | .642$^{*}$ | .130 | .346 | .343 | .385 | -.297 | -.229 | .226 | .089 | .081 | .479$^{*}$ | .434$^{*}$ | .296 | .432 | .086 | .451 | .499 | -.088 |
| β–HBCDD | .075 | .128 | .088 | .272 | .564 | .056 | .362 | .382 | .453$^{*}$ | -.269 | -.183 | .209 | .177 | .132 | .458$^{*}$ | .427$^{*}$ | .253 | .425 | .153 | .474 | .538 | -.044 |
| γ–HBCDD | .286 | .354 | .160 | .356 | .792$^{**}$ | .234 | .400$^{*}$ | .364 | .431 | -.330 | -.264 | .126 | .159 | .242 | .639$^{**}$ | .622$^{**}$ | .508$^{*}$ | .454$^{*}$ | .145 | .387 | .369 | -.017 |

$^{**}$. Correlation is significant at the 0.01 level (2-tailed; light purple); $^{*}$. Correlation is significant at the 0.05 level (2-tailed; light green).

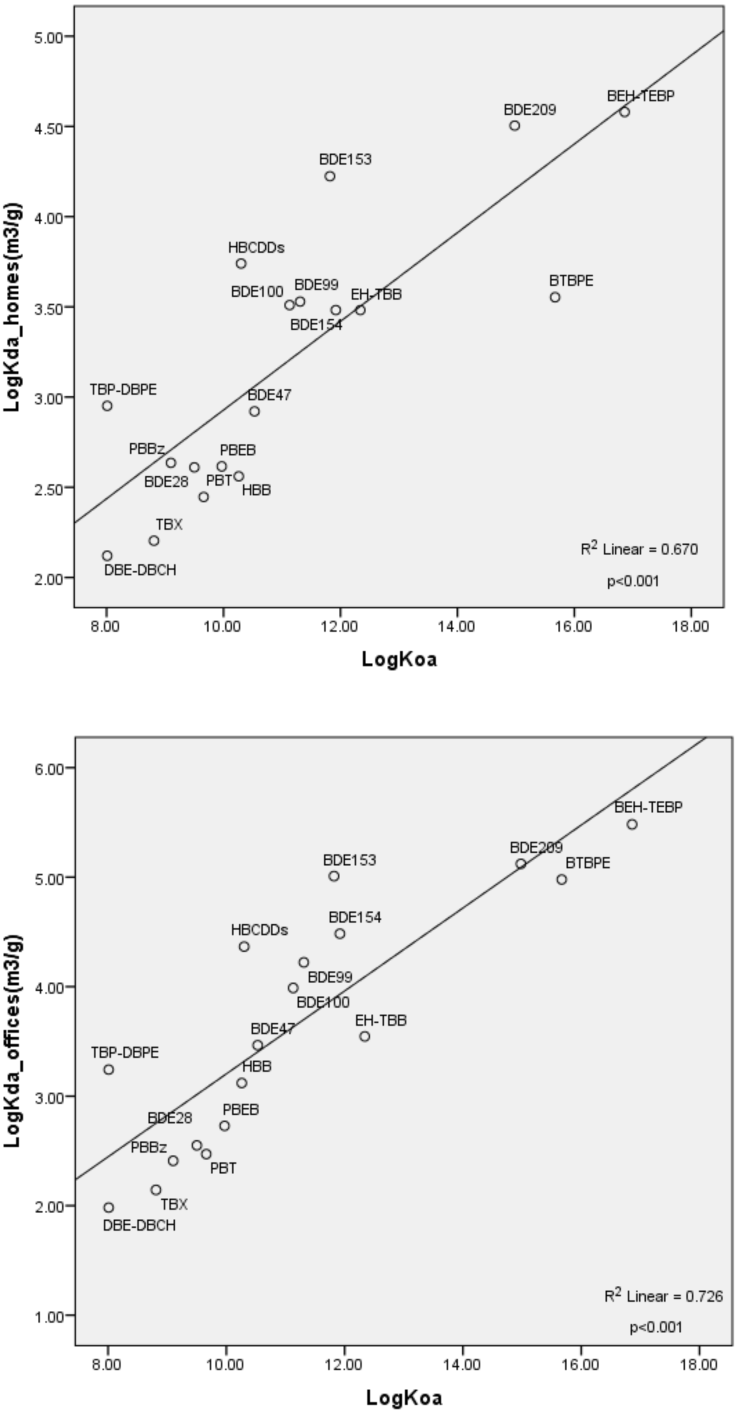


**Figure 4.3 Correlation of $K_{da}$ versus $K_{oa}$ for EFRs and PBDEs in homes (above) and offices (below)**

## 4.10 Exposure to FRs via inhalation and dust ingestion

Human exposure to FRs via air inhalation and dust ingestion was estimated based on concentrations reported in this study. Dust ingestion and air inhalation were assumed to occur pro-rata to typical activity patterns (for adults 78.9% home, 21.1% office; for toddlers 100% home) (Harrad et al., 2008a). We also assumed 100% absorption of intake of FRs (Jones-Otazo et al. 2005). Average dust ingestion rates of 20 and 50 mg/day were used for adult and toddler, respectively, and high dust ingestion rates were 50 mg/d and 200 mg/d for adults and toddlers, respectively (Jones-Otazo et al. 2005). Air inhalation rate figures for adults and toddlers were assumed to be on average 20 (Currado and Harrad, 1998) and 3.8 $m^3$/day (Wilford et al., 2004), respectively. We then estimated various plausible exposure scenarios, using minimum, 5th percentile, median, average, 95th percentile and maximum concentrations in our air and dust samples. Tables 4.8, 4.9 and 4.10 show the resulting estimates of the exposure of UK adults and toddlers to different classes of FRs via air inhalation. The median estimated human exposure estimates for toddlers via air inhalation to ∑EFRs, ∑PBDEs and ∑HBCDDs were 0.66, 0.43 and 0.14 ng/day, respectively. For adults, the estimated human exposure via air inhalation to ∑EFRs, ∑PBDEs and ∑HBCDDs were 4.3, 2.0 and 2.1 ng/day, respectively. Estimated exposure via air inhalation to ∑tri-hexa BDEs was previously found to be 0.16 and 0.82 ng/day for toddlers and adults, respectively (Harrad et al., 2006), which is comparable to the estimates of this study (0.12 and 0.60 ng/day for toddlers and adults, respectively). There are no previous estimates of human exposure to BDE 209 via air inhalation in the UK. For ΣHBCDDs, Abdallah et al. (2008) reported that daily intakes of toddlers and adults under a “typical” exposure scenario (i.e. air contaminated at the median concentration) were at 0.8 and 3.9 ng/day, respectively, slightly higher than our study. To our knowledge, this is first estimate of human exposure to EFRs via air inhalation. The main contributors to estimated exposures of FRs for adults through air inhalation were α-DBE-

DBCH, β-DBE-DBCH, BDE 209 and γ-HBCDD (contributing to 21%, 14%, 17% and 24% of the total exposure, respectively, based on median concentrations) (Figure 4.4). Similar findings were observed for toddlers, for whom α-DBE-DBCH, β-DBE-DBCH, BDE 209 and γ-HBCDD contributed to 20%, 14%, 26% and 11% of the total inhalation exposure, respectively, based on median concentrations (Figure 4.5).

Tables 4.11, 4.12 and 4.13 show our estimates of exposure of UK adults and toddlers to different classes of FRs via dust ingestion. Estimates of "typical" human exposure for toddlers (i.e. dust contaminated at the median concentration ingested at the mean dust ingestion rate) to ∑EFRs, ∑PBDEs and ∑HBCDDs were 38, 280 and 25 ng/day, respectively. For adults, estimates of human exposure under a "typical" exposure scenario to ∑EFRs, ∑PBDEs and ∑HBCDDs were 27, 100 and 15 ng/day, respectively. Harrad et al. (2008a) reported "typical" exposure of toddlers and adults to ∑PBDEs to be 234.3 and 612.6 ng/day respectively, which in both instances exceeds our exposure estimates. A previous report of estimated exposure to ∑HBCDDs via dust ingestion under a "typical" exposure scenario put such exposure at 86.9 and 32.5 ng/day for toddlers and adults respectively. This exceeds slightly the estimates reported in this study. For EFRs, our estimates of "typical" exposure via dust ingestion exceed substantially those reported in a previous study (Ali et al., 2011a) (Table 4.12).

Under an exposure scenario assuming mean dust ingestion rates and median concentrations of both air and dust, dust ingestion is a more significant contributor to human exposure to ∑EFRs, ∑PBDEs and ∑HBCDDs (shown in Table 4.14) when compared with air inhalation, which is similar to the findings reported by the previous studies for PBDEs and HBCDDs (Abdallah et al., 2008; Harrad et al., 2006). For individual FRs, air inhalation contributes more to UK adults' estimated exposure than dust ingestion for target compounds with relatively higher vapour pressures such as DBE-DBCH, PBBz, TBX, PBEB, and BDE 28 in both mean and high dust ingestion scenarios. For toddlers' intake of FRs, dust ingestion was the main route for all target compounds. BDE 209 showed the highest estimated exposures of FRs for adults through dust ingestion (contributing to 70% of the total exposure based on media concentration of FRs) followed by TBBPA-BDBPE (contributing to 10% of the total exposure based on media concentration of FRs) (Figure 4.6). Similar observations were found for toddlers, BDE 209 and TBBPA-BDBPE contributed to 81% and 7% of the total exposure, respectively (Figure 4.7).

## Table 4.8 Summary of estimates of exposure (ng/day) of UK Adults and Toddlers to EFRs via air

| | α-DBE -DBCH | β-DBE-DBCH | TBX | PBBz | TBCT | PBT | PBEB | TBP-DBPE | HBB | EH-TBB | BTBPE | BEH-TEBP | syn-DDC-CO | anti-DDC-CO | TBBPA-BDBPE | DBDPE | ∑ EFRs |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Toddlers** | | | | | | | | | | | | | | | | | |
| **5th percentile** | 0.075 | 0.057 | 0.015 | 0.0027 | 0.00019 | 0.011 | 0.0018 | 0.0018 | 0.00038 | 0.00053 | 0.0019 | 0.0014 | 0.0038 | 0.0023 | 0.019 | 0.019 | 0.21 |
| **95th percentile** | 0.97 | 0.80 | 0.45 | 0.054 | 0.32 | 0.18 | 0.014 | 0.040 | 0.14 | 0.064 | 0.16 | 0.15 | 0.0079 | 0.031 | 0.22 | 0.33 | 3.9 |
| **Mean** | 0.38 | 0.28 | 0.12 | 0.025 | 0.084 | 0.066 | 0.0062 | 0.013 | 0.040 | 0.018 | 0.042 | 0.038 | 0.0047 | 0.0083 | 0.051 | 0.099 | 1.3 |
| **Median** | 0.24 | 0.17 | 0.037 | 0.02 | 0.038 | 0.042 | 0.0050 | 0.0075 | 0.016 | 0.0075 | 0.019 | 0.0080 | 0.0038 | 0.0023 | 0.019 | 0.019 | 0.66 |
| **Minimum** | 0.066 | 0.05 | 0.0059 | 0.00019 | 0.00019 | 0.0086 | 0.0015 | 0.00076 | 0.00038 | 0.00019 | 0.0019 | 0.00019 | 0.0038 | 0.0023 | 0.019 | 0.019 | 0.18 |
| **Maximum** | 1.3 | 0.94 | 0.74 | 0.083 | 0.93 | 0.24 | 0.021 | 0.055 | 0.35 | 0.17 | 0.19 | 0.48 | 0.017 | 0.076 | 0.33 | 0.37 | 6.3 |
| **Geometric mean** | 0.26 | 0.19 | 0.052 | 0.016 | 0.0059 | 0.044 | 0.005 | 0.0077 | 0.0088 | 0.0065 | 0.016 | 0.0071 | 0.0042 | 0.0035 | 0.028 | 0.048 | 0.70 |
| **Adults** | | | | | | | | | | | | | | | | | |
| **5th percentile** | 0.65 | 0.48 | 0.078 | 0.03 | 0.001 | 0.047 | 0.0091 | 0.0082 | 0.002 | 0.0056 | 0.011 | 0.0059 | 0.02 | 0.012 | 0.1 | 0.1 | 1.6 |
| **95th percentile** | 5.8 | 4.4 | 2 | 0.4 | 1.4 | 0.89 | 0.13 | 0.4 | 0.85 | 0.6 | 1.1 | 0.66 | 0.038 | 0.14 | 0.93 | 1.4 | 21 |
| **Mean** | 2.3 | 1.7 | 0.56 | 0.17 | 0.36 | 0.34 | 0.043 | 0.13 | 0.25 | 0.17 | 0.31 | 0.17 | 0.025 | 0.042 | 0.24 | 0.44 | 7.3 |
| **Median** | 1.7 | 1.2 | 0.21 | 0.13 | 0.16 | 0.2 | 0.028 | 0.039 | 0.11 | 0.053 | 0.13 | 0.039 | 0.02 | 0.012 | 0.1 | 0.1 | 4.3 |
| **Minimum** | 0.58 | 0.38 | 0.036 | 0.014 | 0.001 | 0.036 | 0.0077 | 0.004 | 0.002 | 0.0036 | 0.011 | 0.001 | 0.02 | 0.012 | 0.1 | 0.1 | 1.3 |
| **Maximum** | 7.4 | 5.2 | 3.2 | 0.55 | 3.9 | 1.8 | 0.23 | 1.4 | 2.2 | 1.7 | 1.7 | 2 | 0.1 | 0.42 | 1.6 | 1.8 | 35 |
| **Geometric mean** | 1.8 | 1.3 | 0.27 | 0.11 | 0.025 | 0.19 | 0.029 | 0.041 | 0.058 | 0.054 | 0.1 | 0.033 | 0.022 | 0.018 | 0.14 | 0.22 | 4.4 |

## Table 4.9 Summary of estimates of exposure (ng/day) of UK Adults and Toddlers to PBDEs via air

| | BDE 28 | BDE 47 | BDE 100 | BDE 99 | BDE 154 | BDE 153 | BDE 183 | BDE 209 | ∑PBDEs |
|---|---|---|---|---|---|---|---|---|---|
| **Toddlers** | | | | | | | | | |
| **5th percentile** | 0.0022 | 0.027 | 0.00019 | 0.0059 | 0.00095 | 0.00076 | 0.0019 | 0.09 | 0.13 |
| **95th percentile** | 0.39 | 2.1 | 0.54 | 1.5 | 0.089 | 0.26 | 0.015 | 3.2 | 8.0 |
| **Mean** | 0.091 | 0.48 | 0.12 | 0.34 | 0.021 | 0.039 | 0.0046 | 0.94 | 2.0 |
| **Median** | 0.0055 | 0.058 | 0.0052 | 0.039 | 0.0018 | 0.0041 | 0.0019 | 0.32 | 0.43 |
| **Minimum** | 0.0019 | 0.021 | 0.00019 | 0.00048 | 0.00095 | 0.00076 | 0.0019 | 0.066 | 0.094 |
| **Maximum** | 1.3 | 6.2 | 1.6 | 4.3 | 0.27 | 0.28 | 0.016 | 5.1 | 19 |
| **Geometric mean** | 0.0066 | 0.076 | 0.0054 | 0.043 | 0.0027 | 0.0048 | 0.003 | 0.41 | 0.55 |
| **Adults** | | | | | | | | | |
| **5th percentile** | 0.013 | 0.12 | 0.001 | 0.032 | 0.005 | 0.0039 | 0.01 | 0.41 | 0.6 |
| **95th percentile** | 1.7 | 9.8 | 2.3 | 6.2 | 0.38 | 1.1 | 0.068 | 14 | 35 |
| **Mean** | 0.4 | 2.2 | 0.51 | 1.5 | 0.089 | 0.16 | 0.021 | 4.2 | 9.0 |
| **Median** | 0.035 | 0.31 | 0.027 | 0.19 | 0.0086 | 0.018 | 0.01 | 1.4 | 2.0 |
| **Minimum** | 0.012 | 0.098 | 0.001 | 0.0076 | 0.005 | 0.0039 | 0.01 | 0.31 | 0.45 |
| **Maximum** | 5.4 | 28 | 6.9 | 18 | 1.1 | 1.2 | 0.073 | 23 | 83 |
| **Geometric mean** | 0.042 | 0.38 | 0.026 | 0.21 | 0.013 | 0.022 | 0.015 | 1.8 | 2.5 |

## Table 4.10 Summary of estimates of exposure (ng/day) of UK Adults and Toddlers to HBCDDs via air

| | **Toddlers** | | | | **Adults** | | | |
|---|---|---|---|---|---|---|---|---|
| | **α-HBCDD** | **β-HBCDD** | **γ-HBCDD** | **∑HBCDDs** | **α-HBCDD** | **β-HBCDD** | **γ-HBCDD** | **∑HBCDDs** |
| **5th percentile** | 0.0049 | 0.0042 | 0.068 | 0.078 | 0.026 | 0.022 | 0.62 | 0.66 |
| **95th percentile** | 0.064 | 0.020 | 1.0 | 1.1 | 3.7 | 1.2 | 15 | 20 |
| **Mean** | 0.022 | 0.0072 | 0.29 | 0.32 | 0.79 | 0.3 | 4.8 | 5.9 |
| **Median** | 0.0049 | 0.0042 | 0.13 | 0.14 | 0.026 | 0.022 | 2.0 | 2.1 |
| **Minimum** | 0.0049 | 0.0042 | 0.059 | 0.068 | 0.026 | 0.022 | 0.34 | 0.39 |
| **Maximum** | 0.067 | 0.020 | 1.3 | 1.4 | 7.3 | 2.7 | 25 | 35 |
| **Geometric mean** | 0.010 | 0.0057 | 0.17 | 0.19 | 0.11 | 0.066 | 2.6 | 2.8 |

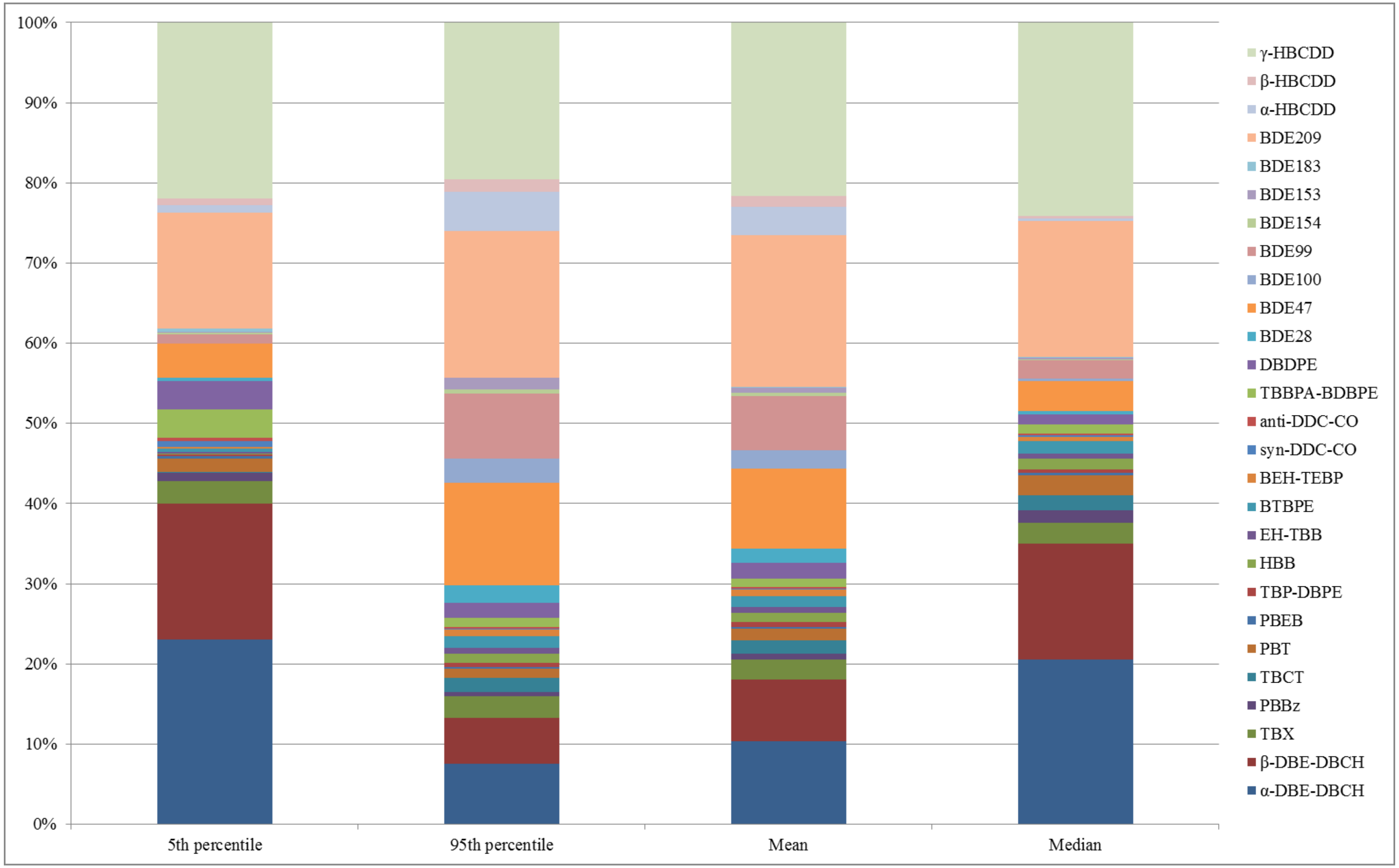


**Figure 4.4 Contributions to estimated exposure of UK adults via air inhalation of different FRs**

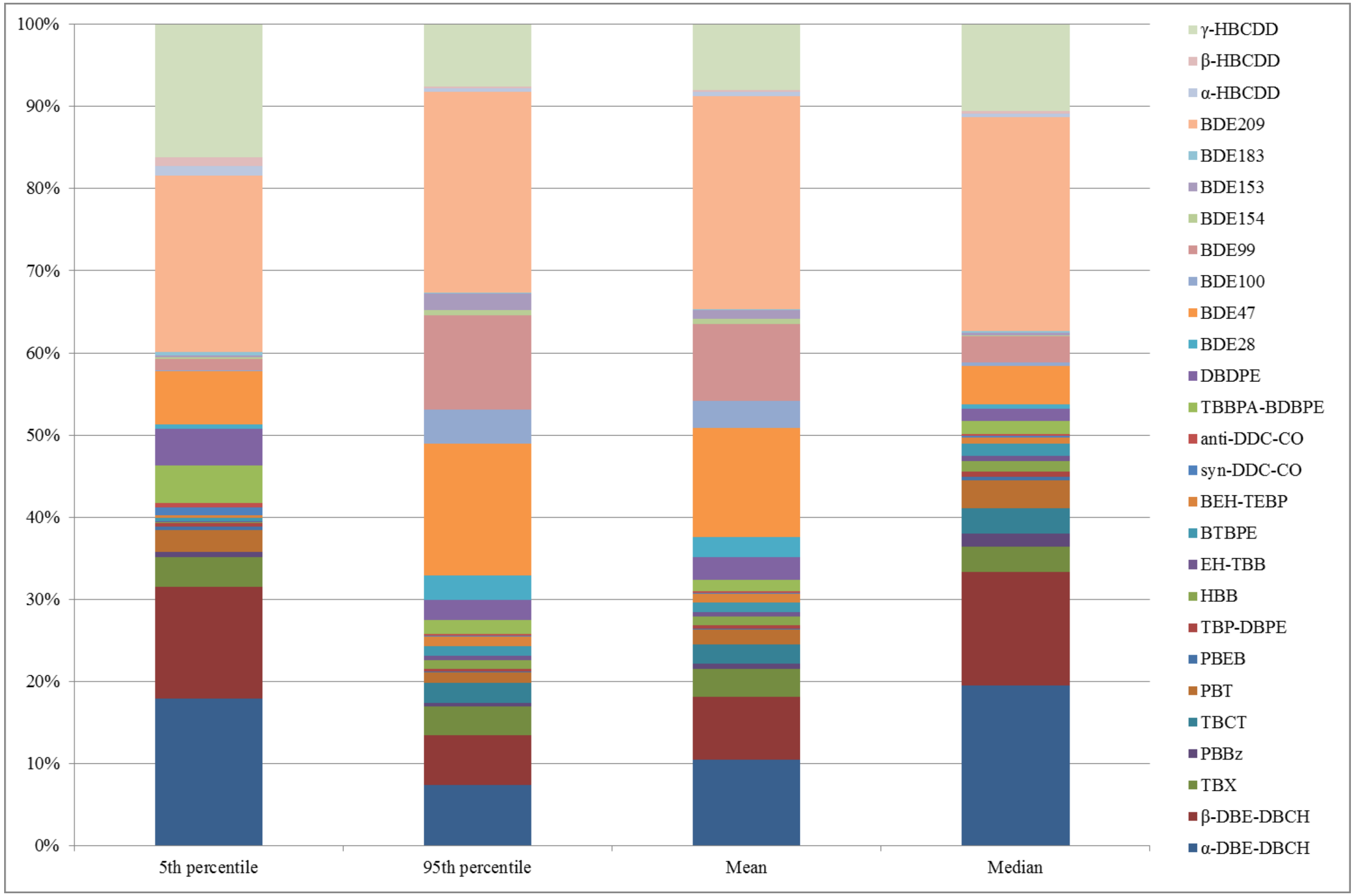


**Figure 4.5 Contributions to estimated exposure of UK toddlers via air inhalation of different FRs**

## Table 4.11 Summary of estimates of exposure (ng/day) of UK Adults and Toddlers to EFRs via dust

| | α-DBE-DBCH | β-DBE-DBCH | TBX | PBBz | TBCT | PBT | PBEB | TBP-DBPE | HBB | EH-TBB | BTBPE | BEH-TEBP | syn-DDC-CO | anti-DDC-CO | TBBPA-BDBPE | DBDPE | ∑ EFRs |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Toddlers** | | | | | | | | | | | | | | | | | |
| **Mean dust ingestion** | | | | | | | | | | | | | | | | | |
| **5th percentile** | 0.11 | 0.12 | 0.0015 | 0.0005 | 0.20 | 0.056 | 0.0055 | 0.0015 | 0.001 | 0.001 | 0.16 | 1.4 | 0.005 | 0.004 | 9.8 | 0.60 | 12 |
| **95th percentile** | 1.7 | 2.6 | 1.2 | 0.44 | 11 | 0.7 | 0.18 | 1.5 | 0.48 | 3.7 | 4.3 | 75 | 0.85 | 7.5 | 440 | 83 | 640 |
| **Mean** | 0.57 | 0.77 | 0.26 | 0.17 | 2.8 | 0.23 | 0.05 | 0.32 | 0.14 | 0.94 | 1.0 | 19 | 0.28 | 1.8 | 94 | 22 | 140 |
| **Median** | 0.31 | 0.44 | 0.065 | 0.11 | 0.42 | 0.15 | 0.017 | 0.058 | 0.083 | 0.35 | 0.31 | 4.7 | 0.014 | 0.46 | 23 | 7.8 | 38 |
| **Minimum** | 0.072 | 0.077 | 0.0015 | 0.0005 | 0.2 | 0.034 | 0.00050 | 0.0015 | 0.0010 | 0.0010 | 0.16 | 0.92 | 0.005 | 0.004 | 3.5 | 0.6 | 5.6 |
| **Maximum** | 2.6 | 3.8 | 1.9 | 0.61 | 15 | 0.88 | 0.23 | 1.9 | 0.61 | 3.7 | 5.4 | 170 | 1.4 | 8.4 | 600 | 110 | 940 |
| **Geometric mean** | 0.35 | 0.39 | 0.06 | 0.068 | 0.63 | 0.15 | 0.021 | 0.068 | 0.018 | 0.19 | 0.51 | 6.0 | 0.043 | 0.33 | 34 | 6.5 | 49 |
| **High dust ingestion** | | | | | | | | | | | | | | | | | |
| **5th percentile** | 0.45 | 0.47 | 0.006 | 0.002 | 0.81 | 0.22 | 0.022 | 0.006 | 0.004 | 0.004 | 0.66 | 5.5 | 0.02 | 0.016 | 39 | 2.4 | 50 |
| **95th percentile** | 6.8 | 10 | 5.0 | 1.8 | 45 | 2.8 | 0.73 | 6.2 | 1.9 | 15 | 17 | 300 | 3.4 | 30 | 1800 | 330 | 2500 |
| **Mean** | 2.3 | 3.1 | 1.0 | 0.66 | 11 | 0.91 | 0.2 | 1.3 | 0.55 | 3.8 | 4.1 | 76 | 1.1 | 7.2 | 380 | 86 | 580 |
| **Median** | 1.3 | 1.8 | 0.26 | 0.45 | 1.7 | 0.58 | 0.067 | 0.23 | 0.33 | 1.4 | 1.2 | 19 | 0.056 | 1.8 | 90 | 31 | 150 |
| **Minimum** | 0.29 | 0.31 | 0.006 | 0.002 | 0.79 | 0.14 | 0.002 | 0.006 | 0.004 | 0.004 | 0.63 | 3.7 | 0.02 | 0.016 | 14 | 2.4 | 22 |
| **Maximum** | 10 | 15 | 7.5 | 2.4 | 59 | 3.5 | 0.94 | 7.5 | 2.4 | 15 | 22 | 690 | 5.5 | 34 | 2400 | 460 | 3800 |
| **Geometric mean** | 1.4 | 1.6 | 0.24 | 0.27 | 2.5 | 0.6 | 0.086 | 0.27 | 0.07 | 0.77 | 2.1 | 24 | 0.17 | 1.3 | 140 | 26 | 200 |
| **Adults** | | | | | | | | | | | | | | | | | |
| **Mean dust ingestion** | | | | | | | | | | | | | | | | | |
| **5th percentile** | 0.061 | 0.058 | 0.0028 | 0.0083 | 0.073 | 0.023 | 0.0037 | 0.0061 | 0.014 | 0.041 | 0.13 | 0.85 | 0.02 | 0.062 | 5.2 | 1.5 | 8 |
| **95th percentile** | 0.69 | 0.94 | 0.41 | 0.17 | 3.6 | 0.25 | 0.07 | 0.56 | 0.3 | 3.8 | 99 | 50 | 0.76 | 5.0 | 170 | 95 | 440 |

| | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Mean** | 0.26 | 0.30 | 0.091 | 0.067 | 0.91 | 0.092 | 0.022 | 0.12 | 0.11 | 0.97 | 21 | 14 | 0.24 | 1.5 | 42 | 20 | 100 |
| **Median** | 0.16 | 0.18 | 0.027 | 0.047 | 0.16 | 0.054 | 0.009 | 0.031 | 0.07 | 0.19 | 1.5 | 2.6 | 0.058 | 0.52 | 15 | 6.5 | 27 |
| **Minimum** | 0.038 | 0.036 | 0.0026 | 0.0073 | 0.070 | 0.015 | 0.0017 | 0.0052 | 0.00040 | 0.0004 | 0.097 | 0.65 | 0.019 | 0.034 | 2.4 | 1.4 | 4.8 |
| **Maximum** | 1.0 | 1.4 | 0.62 | 0.23 | 4.8 | 0.53 | 0.11 | 0.75 | 0.49 | 9.4 | 130 | 160 | 1.4 | 7.7 | 230 | 110 | 660 |
| **Geometric mean** | 0.17 | 0.17 | 0.025 | 0.035 | 0.22 | 0.057 | 0.011 | 0.037 | 0.042 | 0.17 | 2.3 | 3.4 | 0.087 | 0.50 | 19 | 7.6 | 34 |
| **High dust ingestion** | | | | | | | | | | | | | | | | | |
| **5th percentile** | 0.15 | 0.15 | 0.007 | 0.021 | 0.18 | 0.057 | 0.0093 | 0.015 | 0.035 | 0.10 | 0.33 | 2.1 | 0.051 | 0.16 | 13 | 3.7 | 20 |
| **95th percentile** | 1.7 | 2.4 | 1.0 | 0.42 | 9.1 | 0.63 | 0.17 | 1.4 | 0.76 | 9.6 | 250 | 120 | 1.9 | 13 | 440 | 240 | 1100 |
| **Mean** | 0.64 | 0.75 | 0.23 | 0.17 | 2.3 | 0.23 | 0.054 | 0.31 | 0.26 | 2.4 | 54 | 35 | 0.60 | 3.8 | 100 | 50 | 250 |
| **Median** | 0.39 | 0.46 | 0.068 | 0.12 | 0.39 | 0.14 | 0.022 | 0.078 | 0.18 | 0.48 | 3.8 | 6.4 | 0.15 | 1.3 | 37 | 16 | 67 |
| **Minimum** | 0.094 | 0.09 | 0.0066 | 0.018 | 0.17 | 0.037 | 0.0042 | 0.013 | 0.001 | 0.001 | 0.24 | 1.6 | 0.047 | 0.084 | 6.0 | 3.5 | 12 |
| **Maximum** | 2.6 | 3.5 | 1.6 | 0.56 | 12 | 1.3 | 0.27 | 1.9 | 1.2 | 24 | 330 | 400 | 3.5 | 19 | 580 | 270 | 1600 |
| **Geometric mean** | 0.43 | 0.43 | 0.064 | 0.087 | 0.56 | 0.14 | 0.028 | 0.093 | 0.10 | 0.43 | 5.7 | 8.6 | 0.22 | 1.3 | 47 | 19 | 84 |

## Table 4.12 Summary of estimates of exposure (ng/day) of UK Adults and Toddlers to PBDEs via dust

| | **BDE 28** | **BDE 47** | **BDE 100** | **BDE 99** | **BDE 154** | **BDE 153** | **BDE 183** | **BDE 209** | **∑PBDEs** |
|---|---|---|---|---|---|---|---|---|---|
| **Toddlers** | | | | | | | | | |
| **Mean dust ingestion** | | | | | | | | | |
| **5th percentile** | 0.00050 | 0.29 | 0.074 | 0.58 | 0.022 | 0.054 | 0.065 | 9.1 | 10 |
| **95th percentile** | 2.8 | 38 | 37 | 60 | 24 | 22 | 5.8 | 4700 | 4900 |
| **Mean** | 0.63 | 9.0 | 8.4 | 14 | 5.3 | 5.3 | 1.3 | 1200 | 1200 |
| **Median** | 0.023 | 0.84 | 0.19 | 1.4 | 0.08 | 0.67 | 0.17 | 270 | 280 |
| **Minimum** | 0.00050 | 0.26 | 0.062 | 0.41 | 0.022 | 0.037 | 0.062 | 8.2 | 9.1 |
| **Maximum** | 8.9 | 120 | 120 | 190 | 78 | 54 | 13 | 8200 | 8800 |
| **Geometric mean** | 0.019 | 0.99 | 0.29 | 2.0 | 0.14 | 0.8 | 0.28 | 230 | 230 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **High dust ingestion** | | | | | | | | | |
| **5th percentile** | 0.002 | 1.1 | 0.29 | 2.3 | 0.089 | 0.22 | 0.26 | 36 | 41 |
| **95th percentile** | 11 | 150 | 150 | 240 | 94 | 89 | 23 | 19000 | 20000 |
| **Mean** | 2.5 | 36 | 34 | 57 | 21 | 21 | 5.2 | 4600 | 4800 |
| **Median** | 0.091 | 3.4 | 0.74 | 5.7 | 0.32 | 2.7 | 0.70 | 1100 | 1100 |
| **Minimum** | 0.002 | 1.0 | 0.25 | 1.6 | 0.088 | 0.15 | 0.25 | 33 | 36 |
| **Maximum** | 36 | 490 | 490 | 760 | 310 | 220 | 52 | 33000 | 35000 |
| **Geometric mean** | 0.076 | 4.0 | 1.1 | 7.9 | 0.57 | 3.2 | 1.1 | 920 | 940 |
| **Adults** | | | | | | | | | |
| **Mean dust ingestion** | | | | | | | | | |
| **5th percentile** | 0.0002 | 0.15 | 0.035 | 0.29 | 0.013 | 0.05 | 0.039 | 8.0 | 8.5 |
| **95th percentile** | 0.95 | 14 | 12 | 20 | 7.6 | 13 | 2.6 | 1600 | 1700 |
| **Mean** | 0.21 | 3.4 | 2.8 | 5.0 | 1.7 | 3.4 | 0.62 | 410 | 430 |
| **Median** | 0.012 | 0.5 | 0.11 | 0.82 | 0.054 | 0.49 | 0.11 | 100 | 100 |
| **Minimum** | 0.00020 | 0.14 | 0.031 | 0.23 | 0.010 | 0.031 | 0.030 | 6.5 | 6.9 |
| **Maximum** | 2.9 | 41 | 39 | 62 | 25 | 40 | 5.1 | 3000 | 3200 |
| **Geometric mean** | 0.010 | 0.58 | 0.15 | 0.96 | 0.070 | 0.59 | 0.17 | 91 | 93 |
| **High dust ingestion** | | | | | | | | | |
| **5th percentile** | 0.00050 | 0.36 | 0.087 | 0.72 | 0.032 | 0.13 | 0.099 | 20 | 21 |
| **95th percentile** | 2.4 | 35 | 30 | 51 | 19 | 33 | 6.5 | 4000 | 4100 |
| **Mean** | 0.53 | 8.4 | 6.9 | 12 | 4.3 | 8.4 | 1.5 | 1000 | 1100 |
| **Median** | 0.03 | 1.3 | 0.28 | 2.1 | 0.13 | 1.2 | 0.27 | 250 | 260 |
| **Minimum** | 0.0005 | 0.34 | 0.077 | 0.58 | 0.026 | 0.077 | 0.076 | 16 | 17 |
| **Maximum** | 7.2 | 100 | 98 | 150 | 62 | 100 | 13 | 7600 | 8100 |
| **Geometric mean** | 0.026 | 1.4 | 0.36 | 2.4 | 0.18 | 1.5 | 0.43 | 230 | 230 |

## Table 4.13 Summary of estimates of exposure (ng/day) of UK Adults and Toddlers to HBCDDs via dust

| | **Toddlers** | | | | **Adults** | | | |
|---|---|---|---|---|---|---|---|---|
| **Mean dust ingestion** | | | | | | | | |
| | **α-HBCDD** | **β-HBCDD** | **γ-HBCDD** | **∑HBCDDs** | **α-HBCDD** | **β-HBCDD** | **γ-HBCDD** | **∑HBCDDs** |
| **5th percentile** | 2.1 | 0.53 | 1.2 | 3.8 | 1.5 | 0.4 | 0.64 | 2.5 |
| **95th percentile** | 500 | 300 | 1100 | 1900 | 170 | 96 | 350 | 610 |
| **Mean** | 110 | 55 | 240 | 410 | 41 | 19 | 80 | 140 |
| **Median** | 16 | 4.3 | 4.6 | 25 | 9.2 | 2.7 | 3 | 15 |
| **Minimum** | 1.1 | 0.31 | 1.1 | 2.5 | 0.76 | 0.19 | 0.49 | 1.4 |
| **Maximum** | 1400 | 590 | 3500 | 5500 | 460 | 190 | 1100 | 1800 |
| **Geometric mean** | 17 | 5.4 | 8.2 | 31 | 8.8 | 2.7 | 4.1 | 16 |
| **High dust ingestion** | | | | | | | | |
| | **α-HBCDD** | **β-HBCDD** | **γ-HBCDD** | **∑HBCDDs** | **α-HBCDD** | **β-HBCDD** | **γ-HBCDD** | **∑HBCDDs** |
| **5th percentile** | 8.4 | 2.1 | 4.7 | 15 | 3.7 | 0.99 | 1.6 | 6.3 |
| **95th percentile** | 2000 | 1200 | 4300 | 7500 | 420 | 240 | 870 | 1500 |
| **Mean** | 460 | 220 | 980 | 1700 | 100 | 46 | 200 | 350 |
| **Median** | 65 | 17 | 19 | 100 | 23 | 6.8 | 7.4 | 37 |
| **Minimum** | 4.2 | 1.2 | 4.6 | 10 | 1.9 | 0.48 | 1.2 | 3.6 |
| **Maximum** | 5700 | 2400 | 14000 | 22000 | 1100 | 470 | 2800 | 4400 |
| **Geometric mean** | 69 | 22 | 33 | 120 | 22 | 6.8 | 10 | 39 |

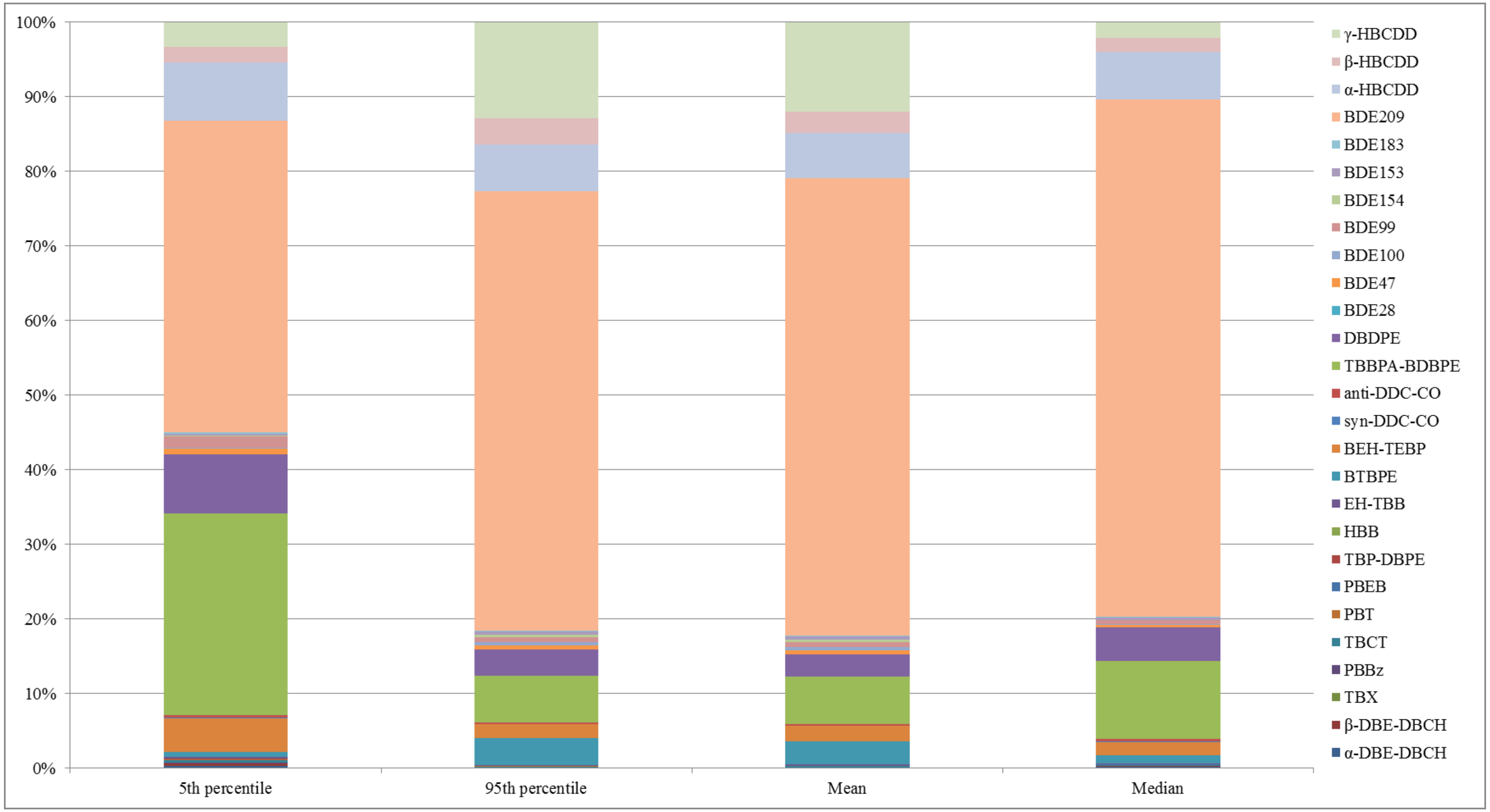


**Figure 4.6 Contributions to human exposure of UK adults via dust ingestion from different FRs**

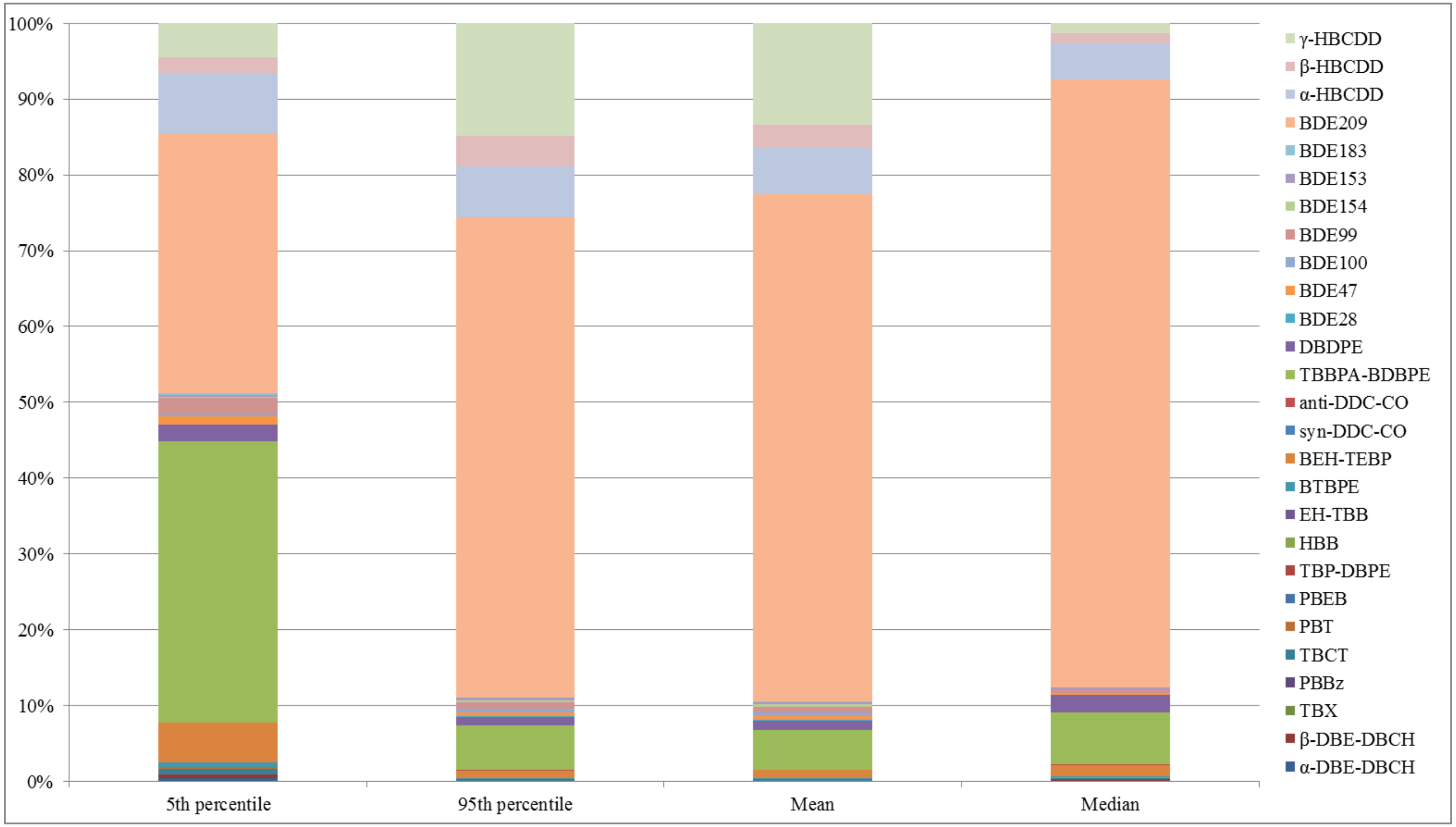


**Figure 4.7 Contributions to human exposure of UK toddlers via dust ingestion from different FRs**

### Table 4.14 Estimates of exposure (ng/day) of UK Adults and Toddlers to FRs via air and dust, and relative significance (%) of each pathway under typical exposure scenario [a]

| Compounds | Intake (ng/day) | | | | Percent contribution of exposure pathway to total exposure (%) | | | |
|---|---|---|---|---|---|---|---|---|
| | Toddlers | | Adults | | Toddlers | | Adults | |
| | Air | Dust | Air | Dust | Air | Dust | Air | Dust |
| ∑tri-hepta BDEs | 0.11 | 10 | 0.6 | 2.1 | 1.1% | 99% | 22% | 78% |
| BDE 209 | 0.32 | 270 | 1.4 | 100 | 0.1% | 100% | 1.4% | 99% |
| ∑HBCDDs | 0.14 | 25 | 2.1 | 15 | 0.6% | 99% | 12% | 88% |
| ∑ EFRs | 0.66 | 38 | 4.3 | 27 | 1.7% | 98% | 14% | 86% |
| α-DBE-DBCH | 0.24 | 0.31 | 1.7 | 0.16 | 44% | 56% | 91% | 9% |
| β-DBE-DBCH | 0.17 | 0.44 | 1.2 | 0.18 | 28% | 72% | 87% | 13% |
| TBX | 0.037 | 0.065 | 0.21 | 0.027 | 36% | 64% | 89% | 11% |
| PBBz | 0.02 | 0.11 | 0.13 | 0.047 | 15% | 85% | 73% | 27% |
| TBCT | 0.038 | 0.42 | 0.16 | 0.16 | 8.3% | 92% | 50% | 50% |
| PBT | 0.042 | 0.15 | 0.2 | 0.054 | 22% | 78% | 79% | 21% |
| PBEB | 0.005 | 0.017 | 0.028 | 0.009 | 23% | 77% | 76% | 24% |
| TBP-DBPE | 0.0075 | 0.058 | 0.039 | 0.031 | 11% | 89% | 56% | 44% |
| HBB | 0.016 | 0.083 | 0.11 | 0.07 | 16% | 84% | 61% | 39% |
| EH-TBB | 0.0075 | 0.35 | 0.053 | 0.19 | 2.1% | 98% | 22% | 78% |
| BTBPE | 0.019 | 0.31 | 0.13 | 1.5 | 5.8% | 94% | 8.0% | 92% |
| BEH-TEBP | 0.008 | 4.7 | 0.039 | 2.6 | 0.2% | 100% | 1.5% | 99% |
| syn-DDC-CO | 0.0038 | 0.014 | 0.02 | 0.058 | 21% | 79% | 26% | 74% |
| anti-DDC-CO | 0.0023 | 0.46 | 0.012 | 0.52 | 0.5% | 100% | 2.3% | 98% |
| TBBPA-BDBPE | 0.019 | 23 | 0.1 | 15 | 0.1% | 100% | 0.7% | 99% |
| DBDPE | 0.019 | 7.8 | 0.1 | 6.5 | 0.2% | 100% | 1.5% | 98% |

a) typical exposure scenario means assuming mean dust ingestion rates and median concentrations of both air and dust.

# CHAPTER 5 EFRs, PBDEs and HBCDDs in UK food samples

## 5.1 Synopsis

Food is considered as one of the main routes of human exposure to BFRs, including PBDEs (Fromme et al., 2009; Harrad et al., 2006; Roosens et al., 2009b), HBCDD (Abdallah and Harrad, 2011; Roosens et al., 2009a) and EFRs (Labunska et al., 2015). Because of recent bans and restrictions on the use of "legacy" BFRs (Stockholm Convention, 2009; European Court of Justice, 2008; UNEP, 2014), use of EFRs may increase as a result of the replacement of "legacy" compounds. The occurrence of relatively high concentrations of EFRs in the environment may also reflect rising use of EFRs (Ali et al., 2012a; Cequier et al., 2014; Dodson et al., 2012; Hoh et al., 2005; Newton et al., 2015; Shoeib et al., 2012; Stapleton et al., 2008). Furthermore, a few reports suggest that some EFRs are bioaccumulative. This information includes: high log BAF values of DBDPE (6.1 - 7.1) in fish that are one order of magnitude higher than those of BDE 209 (He et al., 2012), the long half-life and biomagnification potential of BTBPE in juvenile rainbow trout (Tomy et al., 2007a), and a reported high BCF value (2,153) for DBE-DBCH isomers, which have been identified as having high persistence and environmental bioaccumulation potential in this study (Howard and Muir, 2010). Therefore, the levels of EFRs in food may increase in the future. However, very limited information on human dietary exposure to EFRs has been reported so far. Hence, in this study, EFRs were measured in 14 groups of composite food samples covering meat, liver, oily fish, eggs and cheese to provide a preliminary estimate of UK dietary exposure. Additionally, concentrations of 8 PBDEs and 3 HBCDD diastereomers were measured in the same samples to compare with those reported in previous UK studies. By so doing, it is possible to evaluate the efficacy of regulations and restrictions designed to reduce human dietary exposure.

## 5.2 Concentrations and patterns of HFRs in food

### 5.2.1 EFRs

Food samples were collected from three markets in Birmingham, UK from May to June 2015 (see section 2.3.3.1 for sampling details). Table 5.1 summarises the concentrations detected in this study of EFRs in composite food samples collected in the UK. β-DBE-DBCH was detected in all food samples, with α-DBE-DBCH and EH-TBB detected in 97% and 77% of samples, respectively, while DBDPE was only detected in 33% of food samples. β-DBE-DBCH was the predominant compound in food samples, accounting for 35%-94% of ΣEFRs. To the best of our knowledge, only one previous study has reported concentrations of DBE-DBCH in food samples (fish, meat, vegetable oils, dairy products, and eggs) as part of a Swedish market basket study (Sahlström et al., 2015). In that study, DBE-DBCH was only found in four fish samples at levels (average 114 pg/g ww) lower than those in our study (fish: 240-1820 pg/g ww). Moreover, α-DBE-DBCH was the dominant EFR (Sahlström et al., 2015) in contrast to our study. The highest concentrations of ΣEFRs in the current study were detected in liver samples ranging from 42 to 93 ng/g lw, which exceeded those in meat (4.5-14 ng/g lw). Labunska et al. (2015) reported similarly elevated levels in liver and meat samples from chickens and ducks collected in an e-waste processing area in Eastern China for most EFRs, including: EH-TBB, BEH-TEBP, and BTBPE (Table 5.2). Very limited studies are available concerning the levels of EFRs in UK food samples. However, we are aware of one previous study of a selection of UK and Irish food samples for analysis of HBB, DBDPE, and BTBPE (Fernandes et al., 2010). HBB and DBDPE were not detected in any foods, whilst BTBPE occurred in some UK food samples such as meat (0.05 - 1.76 ng/g lw), offal (0.75 - 0.81 ng/g lw), fish (0.26 - 3.33 ng/g lw), and eggs (0.29 ng/g lw) (Fernandes et al., 2010) which is comparable to the levels of BTBPE in our study (Table 5.2). Meanwhile, Labunska et al. (2015) detected BTBPE, EH-TBB, BEH-TEBP and DBDPE in meat, fish, liver and egg samples from an e-waste processing area (Taizhou City) and control sites (Shanghai and Nanjing City) in South China at levels that exceed those in our study, with the exception of DBDPE which was not detected (Table 5.2). Another study reported levels of BTBPE and DBDPE in chicken eggs from an e-waste processing area, South China to also exceed those in our study (Table 5.2) (Zheng et al., 2012). With respect to fish, BTBPE and DBDPE were also detected in Canada (Law et al., 2006a), France (Munschy et al., 2011) and China (He et al., 2012; Li et al., 2015; Shi et al., 2009) at levels comparable to those in fish samples in our study (Table 5.2).

As mentioned earlier, β-DBE-DBCH was the dominant isomer detected in our study, with $f_{\beta\text{-DBE-DBCH}}$ in our food samples ranging from 1.20 to 220, with a median value of 7.3. Gauthier et al. (2008) also found β-DBE-DBCH to be the predominant isomer in herring gull egg pools. Furthermore, the isomer profile of DBE-DBCH in the blubber of Canadian arctic beluga whale was also largely dominated by β-DBE-DBCH (Tomy et al., 2008b). Interestingly, the $f_{\beta\text{-DBE-DBCH}}$ values in our food samples exceed significantly those detected in UK indoor dust (0.32-2.88) (t-test on log-transformed concentrations, $p < 0.01$), indoor air (0.53 to 1.0) (t-test on log-transformed concentrations, $p < 0.01$) and the commercial product (1.0), suggesting diastereomer-specific environmental degradation/metabolism and/or isomerisation in the food chain. Interestingly, the median value of $f_{\beta\text{-DBE-DBCH}}$ in liver (80) in this study was substantially higher than those found in meat (5.9), fish (6.1, tuna not included), eggs (7.1), and cheese (2.1). Moreover, the levels of DBE-DBCH were higher in liver than in other food samples studied except tuna, with the $f_{\beta\text{-DBE-DBCH}}$ value in tuna (median value: 83) exceeding those in other fish samples. These findings indicate that the uptake and metabolism of DBE-DBCH isomers is species- and organism-dependent.

**Table 5.1 Average concentrations of EFRs in composite food samples from Birmingham, UK (ng/g lw)**

| | Number of composite samples | Lipid weight (%) | Water content (%) | α-DBE-DBCH | β-DBE-DBCH | EH-TBB | BTBPE | BEH-TEBP | DBDPE | ∑EFRs |
|---|---|---|---|---|---|---|---|---|---|---|
| **DF** | | -- | -- | 97% | 100% | 77% | 60% | 63% | 33% | -- |
| **Meat** | | | | | | | | | | |
| **Beef** | 3 | 8.0 | 67 | 0.89 | 1.9 | 0.20 | <0.04 | 0.44 | <1.1 | 4.5 |
| **Lamb** | 3 | 8.0 | 68 | 0.62 | 6.2 | 0.19 | <0.05 | 0.28 | 3.5 | 11 |
| **Pork** | 3 | 8.0 | 66 | 0.95 | 4.1 | 1.4 | 2.4 | 0.20 | 4.6 | 14 |
| **Chicken** | 3 | 5.0 | 73 | 0.58 | 7.6 | 0.36 | 1.3 | 0.57 | <1.5 | 11 |
| **Liver** | | | | | | | | | | |
| **Beef liver** | 2 | 4.0 | 64 | 1.6 | 49 | 1.6 | <0.06 | 0.69 | 4.5 | 57 |
| **Lamb liver** | 2 | 5.0 | 65 | <0.26 | 55 | 0.19 | 0.35 | 0.94 | 7.6 | 65 |
| **Pork liver** | 1 | 5.0 | 69 | 1.1 | 85 | 0.63 | 0.24 | 5.0 | 1.5 | 93 |
| **Chicken liver** | 1 | 4.0 | 73 | 0.72 | 34 | 0.66 | 0.14 | 5.8 | <1.6 | 42 |
| **Fish** | | | | | | | | | | |
| **Salmon** | 3 | 9.0 | 65 | 1.3 | 4.4 | 0.32 | <0.04 | <0.1 | 6.6 | 13 |
| **Mackerel** | 2 | 20 | 49 | 1.1 | 4.9 | 0.22 | 0.17 | <0.2 | <0.63 | 7.0 |
| **Tuna** | 2 | 2.0 | 75 | 0.48 | 39 | 0.38 | 0.78 | 0.42 | 21 | 62 |
| **Trout** | 2 | 10 | 67 | 0.60 | 4.6 | 0.43 | 0.16 | 1.1 | <0.88 | 7.4 |
| **Egg and dairy products** | | | | | | | | | | |
| **Cheese** | 2 | 18 | 46 | 0.44 | 0.99 | 0.11 | 0.20 | 0.22 | <0.74 | 2.3 |
| **Egg** | 1 | 11 | 51 | 0.42 | 3.0 | 0.10 | 0.18 | 1.8 | <1.2 | 6.1 |

**Table 5.2 Average concentrations of EFRs (ng/g lw) in biota samples from different countries.**

| Samples | α-DBE-DBCH | β-DBE-DBCH | EH-TBB | BTBPE | BEH-TEBP | DBDPE | Location | References |
|---|---|---|---|---|---|---|---|---|
| **Meat** | | | | | | | | |
| **Meat** | 0.58-0.95 | 1.9-7.6 | 0.19-1.4 | <0.04-2.4 | 0.20-0.57 | <1.1-4.6 | UK | This study |
| **Beef** | | | | 0.56 | | <0.06[c] | UK | (Fernandes et al., 2010) |
| **Pork** | | | | 0.55 | | <0.06[c] | | |
| **Lamb** | | | | 0.05 | | <0.06[c] | | |
| **Turkey** | | | | 1.76 | | <0.06[c] | | |
| **Chicken** | | | 2.66 | <0.35 | 1.78 | <0.45 | Shanghai and Nanjing City, China | (Labunska et al., 2015) |
| **Duck** | | | 2.74 | 1.87 | <0.25 | <0.45 | | |
| **Pork** | | | 2.14 | 2.69 | 1.37 | <0.45 | | |
| **Chicken** | | | 24.7 | 1.46 | 8.97 | <0.45 | E-waste area, South China | |
| **Duck** | | | 24.2 | 4.57 | 7.23 | <0.45 | | |
| **Pork** | | | 38.2 | 5.4 | 12.4 | <0.45 | | |
| **Meat** [a] | | | | | | 100 | Shandong Province, North China | (Li et al., 2015) |
| **Liver** | | | | | | | | |
| **Liver** | <0.26-1.6 | 34-85 | 0.19-1.6 | <0.06-0.35 | 0.69-5.8 | <1.6-7.6 | UK | This study |
| **Pork liver** | | | | 0.81 | | <0.06[c] | UK | (Fernandes et al., 2010) |
| **Chicken liver** | | | | 0.75 | | <0.06[c] | | |
| **Chicken liver** | | | 5 | 3.38 | 2.61 | <0.45 | Shanghai and Nanjing City, China | (Labunska et al., 2015) |
| **Duck liver** | | | 8.2 | 3.27 | 1.69 | <0.45 | | |
| **Chicken liver** | | | 35 | 15 | 10.6 | <0.45 | E-waste area, South China | |
| **Duck liver** | | | 38.4 | 11.7 | 13.7 | <0.45 | | |
| **Fish** | | | | | | | | |
| **Fish** | 0.48-1.3 | 4.4-39 | 0.22-0.43 | <0.04-0.78 | <0.1-1.1 | <0.63-21 | UK | This study |
| **Fish** | | | 4 | 2.1 | 1.9 | <0.45 | Shanghai and Nanjing City, China | (Labunska et al., 2015) |
| **Fish** | | | 24.7 | 1.46 | 8.97 | <0.45 | E-waste area, South China | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Salmon** | | | | 0.26 | | <0.06[c] | UK | (Fernandes et al., 2010) |
| **Mackerel** | | | | 0.3 | | <0.06[c] | | |
| **Herring** | | | | 0.25 | | <0.06[c] | | |
| **Haddock** | | | | 0.83 | | <0.06[c] | | |
| **Lemon Sole** | | | | 3.33 | | <0.06[c] | | |
| **Whitebait** | | | | 0.77 | | <0.06[c] | | |
| **Fish**[a] | 97 | 17 | <14 | 1.1-3.6[b] | < 26 | | Sweden | (Sahlström et al., 2015) |
| **Fish** | | | | <0.05-3.72 | | <0.1-3.30 | Lake Winnipeg | (Law et al., 2006a) |
| **Juvenile common sole** | | | | 0.08-0.31 | | 0.28-1.13 | Nursery zones situated along the French coast | (Munschy et al., 2011) |
| **Fish** | | | | <0.012-0.15 | | <3.8 | E-waste area, South China | (Shi et al., 2009) |
| **Fish** | | | | | | <4.9-230 | Dongjiang River, South China | (He et al., 2012) |
| **Fish and seafood** | | | | | | 121 | Shandong Province, North China | (Li et al., 2015) |
| **Eggs** | | | | | | | | |
| **Egg** | 0.42 | 3.0 | 0.10 | 0.18 | 1.8 | <1.2 | UK | This study |
| **Chicken eggs** | | | 1.73 | <0.35 | <0.25 | <0.45 | Shanghai and Nanjing City, China | (Labunska et al.. 2015) |
| **Chicken eggs** | | | 4.8 | 2.93 | 1.16 | <0.45 | E-waste area, South China | |
| **Duck eggs** | | | 1.21 | <0.35 | <0.25 | <0.45 | Shanghai and Nanjing City, China | |
| **Duck eggs** | | | 4.03 | 2.11 | 1.11 | <0.45 | E-waste area, South China | |
| **Free range organic eggs** | | | | 0.29 | | <0.06[c] | UK | (Fernandes et al., 2010) |
| **Chicken eggs** | | | | 37.2-264 | | 5.97-37.9 | E-waste area, South China | (Zheng et al., 2012) |

a) the units are pg/g ww; b) mLOD – mLOQ; c) the units are ng/g ww.

### 5.2.2 PBDEs

Table 5.3 shows the average concentrations of PBDEs detected in composite food samples in our study. Each of our target congeners was frequently detected with the DF exceeding 73%. BDE 47 and BDE 99 were the major contributing congeners to ∑PBDEs in most of our samples, which is similar to previous studies of food samples in Spain (Domingo et al., 2008), the UK (Harrad et al., 2004) and the USA (Schecter et al., 2009). Of all the food samples analysed in this study, fish showed the highest average concentrations of ∑PBDEs either on a lipid weight basis (14 to 40 ng/g lw) or on a wet weight basis (400-2800 pg/g ww). These values are in line with previous studies in the UK (UK Food Standards Agency, 2006; UK Food and Environment Research Agency, 2009), as well as in Belgium (Stefan Voorspoels et al., 2007), Spain (Domingo et al., 2008), the Netherlands (Bakker et al., 2008), and Sweden (Törnkvist et al., 2011; Sahlström et al., 2015) (Table 5.4). Average levels of ∑PBDEs in all food categories in the UK in this study exceeded those in the USA (Schecter et al., 2009), Belgium (Stefan Voorspoels et al., 2007), Spain (Domingo et al., 2008), the Netherlands (Bakker et al., 2008), Sweden (Törnkvist et al., 2011; Sahlström et al., 2015), China (Su et al., 2012), and Romania (Dirtu and Covaci, 2010). The only exception was that the levels of ∑PBDEs in our fish samples were comparable to those in the Netherlands (Bakker et al., 2008) (Table 5.4). Interestingly, as shown in Table 5.4, the levels of ∑PBDEs in this study are higher than those recorded in previous studies in the UK conducted in 2003-2004 and 2006 (UK Food Standards Agency, 2006; UK Food and Environment Research Agency, 2009). Moreover, the levels of ΣPBDEs in fish from UK fresh water systems were found to be extremely high (mean level: 20 ng/g ww; maximum level: 130 ng/g ww) recently by Rose et al. (2015). However, in chapter 4, we showed evidence of a temporal decline in concentrations of BDE 209 in office dust and of BDE 47, 99 and 154 in office air (data on levels of BDE 209 in indoor air in previous UK studies are not available). This apparent contradiction may be attributable to a gradual shift over time of PBDEs from the indoor to the outdoor environment and thus our food supply (Harrad and Diamond, 2006). Furthermore, as BDE 209 has been reported to debrominate to lower brominated PBDEs in both terrestrial and aquatic biota (Gandhi et al., 2011; La Guardia et al., 2007; Letcher et al., 2014; Stapleton et al., 2006; Tian et al., 2012; Van den Steen et al., 2007), it is plausible that the continuous transfer from the indoor environment to outdoors and subsequent debromination of BDE 209 in biota, could drive temporal increases in levels of lower congener PBDEs in food samples. This hypothesis was supported by the relatively high ratios of tri-hexa-BDEs/BDE 209 in foodstuffs in this study compared to those in two previous studies (Figure. 5.1.) (UK Food Standards Agency, 2006; UK Food and Environment Research Agency, 2009) even though levels of BDE 209 in this study were comparable to those reported in two UK previous studies (Table 5.4) (UK Food Standards Agency, 2006; UK Food and Environment Research Agency, 2009).

## Table 5.3 Average concentrations (pg/g ww in parentheses) of PBDEs in composite food samples from Birmingham, UK (ng/g lw)

| | **Average Lipid weight (%)** | **Average Water content (%)** | **BDE 28** | **BDE 47** | **BDE 100** | **BDE 99** | **BDE 154** | **BDE 153** | **BDE 209** | **∑tri-hexa BDEs** | **∑PBDEs** |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **DF** | -- | -- | 73% | 93% | 90% | 100% | 83% | 100% | 97% | -- | -- |
| **Meat** | | | | | | | | | | | |
| **Beef** | 7.6 | 67 | 0.32 (23) | 5.5 (360) | 1.2 (73) | 7.2 (470) | 0.42 (27) | 0.6 (38) | 0.46 (32) | 15 (990) | 16 (1000) |
| **Lamb** | 8.0 | 68 | 0.082 (7.5) | 0.5 (45) | 0.48 (36) | 0.76 (65) | 0.10 (8.3) | 0.17 (15) | 0.28 (21) | 2.1 (177) | 2.4 (200) |
| **Pork** | 8.0 | 66 | 0.35 (15) | 7.4 (290) | 2.0 (82) | 9.6 (370) | 0.66 (25) | 0.88 (33) | 0.42 (24) | 21 (810) | 21 (830) |
| **Chicken** | 4.6 | 73 | <0.06 (<5.0) | 3.5 (160) | 1.0 (44) | 5.6 (250) | 0.43 (19) | 0.54 (24) | 0.63 (28) | 11 (500) | 12 (520) |
| **Liver** | | | | | | | | | | | |
| **Beef liver** | 4.0 | 64 | 0.62 (24) | 6.5 (250) | 2.0 (76) | 20 (760) | 1.1 (41) | 1.4 (53) | 3.3 (120) | 32 (1200) | 35 (1300) |
| **Lamb liver** | 5.3 | 65 | <0.18 (<7.8) | 0.034 (1.8) | 3.9 (160) | 0.70 (30) | 0.33 (17) | 0.41 (21) | 0.43 (23) | 5.5 (240) | 5.9 (260) |
| **Pork liver** | 4.7 | 69 | 0.33 (19) | 2.0 (82) | 0.17 (5.5) | 1.7 (63) | 0.14 (6.8) | 0.34 (20) | 0.51 (23) | 4.7 (200) | 5.3 (220) |
| **Chicken liver** | 4.6 | 73 | 0.12 (5.5) | 1.3 (59) | 0.069 (3.0) | 1.4 (61) | 0.16 (7.2) | 0.19 (8.7) | 0.47 (21) | 3.2 (150) | 3.7(170) |
| **Fish** | | | | | | | | | | | |
| **Salmon** | 9.1 | 65 | 0.8 (61) | 17 (1100) | 3.9 (230) | 15 (720) | 1.5 (94) | 1.4 (73) | 0.69 (41) | 40 (2300) | 40 (2300) |
| **Mackerel** | 24 | 49 | 0.63 (130) | 5.9 (1200) | 1.2 (250) | 3.8 (770) | 1.0 (210) | 0.39 (80) | 0.74 (150) | 13 (2600) | 14 (2800) |
| **Tuna** | 2.0 | 75 | 0.43 (8.9) | 6.0 (130) | 2.6 (54) | 3.7 (80) | 2.6 (59) | 1.2 (26) | 1.7 (39) | 16 (360) | 18 (400) |
| **Trout** | 9.2 | 67 | 0.76 (71) | 12 (950) | 2.5 (190) | 8.6 (550) | 2.0 (150) | 1.6 (120) | 0.34 (32) | 27 (2000) | 28 (2100) |
| **Egg and dairy product** | | | | | | | | | | | |
| **Cheese** | 18 | 46 | 0.068 (12) | 1.9 (340) | 0.59 (100) | 2.2 (390) | 0.12 (21) | 0.14 (26) | 0.21 (37) | 5.1 (900) | 5.3 (940) |
| **Egg** | 11 | 51 | <0.04 (<4.9) | 0.19 (20) | 0.13 (14) | 0.3 (32) | 1.2 (130) | <0.04 (<4.9) | 0.53 (56) | 1.9 (200) | 2.4 (250) |

**Table 5.4 Average concentrations of ∑PBDEs (pg/g ww) in food samples from different countries (median concentrations in parentheses).**

| Food category | | | | | | | | | | Sampling Year | Country | References |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Meat | | Liver | | Fish | | Cheese | | Egg | | | | |
| ∑PBDEs | BDE 209 | ∑PBDEs | BDE 209 | ∑PBDEs | BDE 209 | ∑PBDEs | BDE 209 | ∑PBDEs | BDE 209 | | | |
| 640<br>(13) [a] | 26<br>(0.45) [a] | 410<br>(10) [a] | 40<br>(0.94) [a] | 1900<br>(27) [a] | 63<br>(0.86) [a] | 940<br>(5.3) [a] | 37<br>(0.21) [a] | 250<br>(2.4) [a] | 56<br>(0.53) [a] | 2015 | UK | This study |
| 322 | 260 | 102 | 40 | 608 | 90 | NA | NA | 143 | 80 | 2003-2004 | UK | (UK Food Standards Agency, 2006) |
| 1.0 [a] | 0.66 [a] | 1.4 [a] | 1.0 [a] | 17 [a] | 0.34 [a] | 0.77 [a] | 0.30 [a] | 2.4 [a] | 0.57 [a] | 2006 | UK | (UK Food and Environment Research Agency, 2009) |
| NA | NA | NA | NA | 20000 | NA | NA | NA | NA | NA | 2014 | UK | (Rose et al., 2015) |
| 80.2 | -- | -- | -- | 32.7 | -- | 117.15 | -- | 89.3 | -- | 2009 | USA | (Schecter et al., 2009) |
| 70 | -- | -- | -- | 460 | -- | 120 | -- | 100 | -- | 2005 | Belgium | (Stefan Voorspoels et al., 2007) |
| 49.9 | -- | -- | -- | 563.9 | -- | 121.5 | -- | 94.8 | -- | 2006 | Spain [b] | (Domingo et al., 2008) |
| 50 | -- | -- | -- | 1460 | -- | 166 | -- | 71 | -- | 2003-2004 | Netherlands [b] | (Bakker et al., 2008) |
| 41 | -- | -- | -- | 422 | -- | -- | -- | 26 | -- | 2005 | Sweden [b] | (Törnkvist et al., 2011) |
| 17 | 4.6 | -- | -- | 332 | <3.6 | 10 | 5.3 | 17 | 13 | 2010 | Sweden [d] | (Sahlström et al., 2015) |
| | -- | -- | -- | 180 | -- | -- | -- | 181 | -- | 2006 | China | (Su et al., 2012) |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 120 | -- | -- | -- | -- | -- | 130 | -- | 30 | -- | 2007 | Romania [c] | (Dirtu and Covaci, 2010) |

a) lipid basis; b): BDE 209 was not measured; c) median concentrations of ∑PBDEs; d): dairy products; NA: not available.

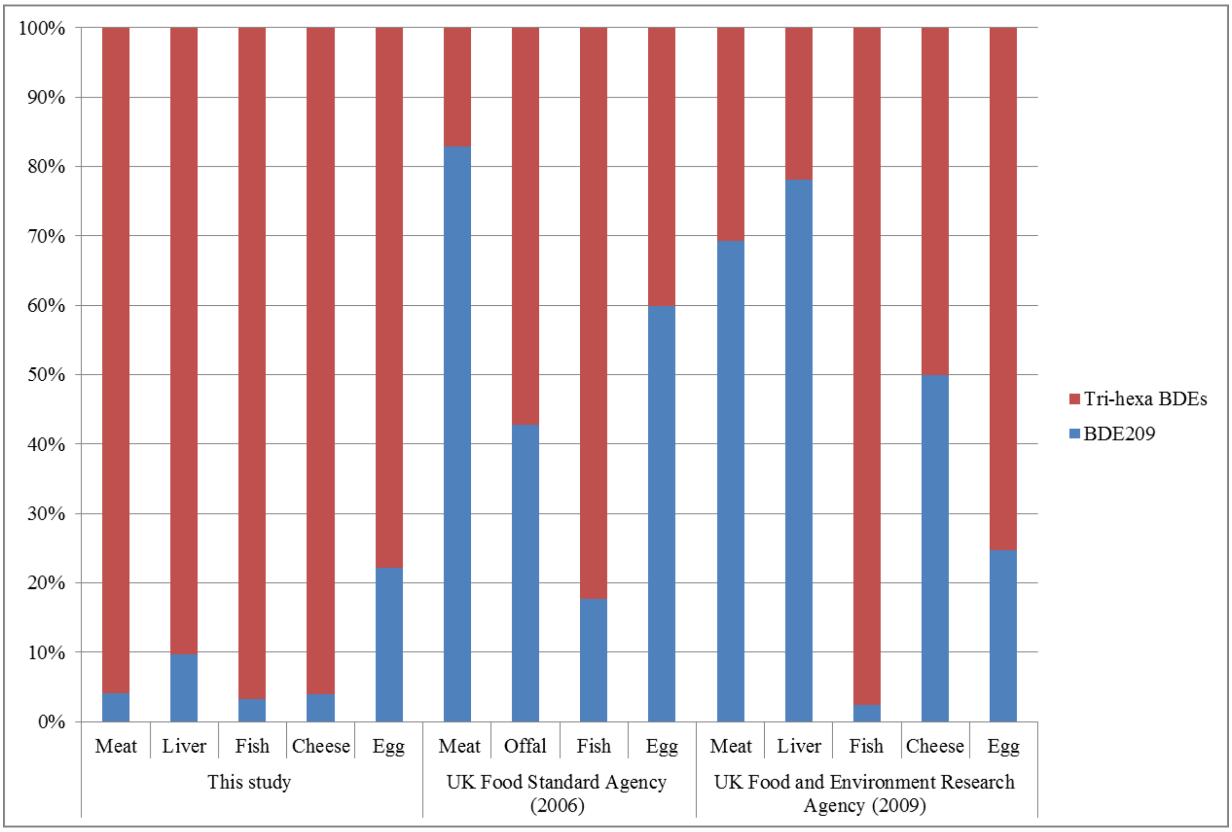


**Figure 5.1 Relative contributions of tri-hexa-BDEs and BDE 209 to ∑PBDEs in UK food samples in this study and two previous studies**

### 5.2.3 HBCDDs

Concentrations of HBCDDs in the food samples analysed in this study are shown in Table 5.5. The highest average concentrations of ∑HBCDD were found in fish (3.6-16 ng/g lw), followed by liver (<0.48-20 ng/g lw), meat (0.32-4.6 ng/g lw), and egg and dairy products (<0.24-1.3 ng/g lw). These levels were comparable to those found in the literature, with fish showing higher concentrations of ∑HBCDDs than other food categories (Eljarrat et al., 2014; Goscinny et al., 2011; Schecter et al., 2009; Shi et al., 2009; Törnkvist et al., 2011; Sahlström et al., 2015). Moreover, concentrations of ΣHBCDD (<0.48-20 ng/g lw; <22-830 pg/g ww) in food samples in this study were comparable to those detected in similar foodstuffs in two UK previous studies (<LOD-300 pg/g ww (Driffield et al., 2008); 65-680 pg/g ww (UK Food Standards Agency, 2006)) as well as those in other countries including: Romania (40-250

pg/g ww) (Dirtu and Covaci, 2010), Sweden (5.0-630 pg/g ww (Törnkvist et al., 2011) and 2.9-208 pg/g ww (Sahlström et al., 2015)), Belgium (<10-350 pg/g ww) (Roosens et al., 2009a), and the USA (nd-593 pg/g ww) (Schecter et al., 2009) but higher than those in China (<LOD-9.2 ng/g lw) (Shi et al., 2009). Generally, α-HBCDD was the dominant diastereomer contributing on average 57±22% ∑HBCDDs in all food samples, in line with previous studies in biota (Covaci et al., 2006; Labunska et al., 2015). The γ-HBCDD diastereomer is more prevalent in liver samples than the other food categories, accounting for 34%-53% of ∑HBCDDs in line with previous studies (Labunska et al., 2015; Tao et al., 2016) of avian samples, which suggested that this pattern arises as the liver is the first organ exposed following the gastrointestinal tract and thus reflects more closely the pattern of contamination sources such as dust, soil and air.

**Table 5.5 Average concentrations (pg/g ww in parentheses) of HBCDDs in composite food samples from Birmingham, UK (ng/g lw)**

| | **α-HBCDD** | **β-HBCDD** | **γ-HBCDD** | **∑HBCDDs** |
|---|---|---|---|---|
| **DF** | 83% | 70% | 73% | |
| **Meat** | | | | |
| **Beef** | 1.6 (100) | 0.44 (28) | 0.62 (40) | 2.7 (170) |
| **Lamb** | 0.22 (15) | <0.12 (<16) | <0.08 (<5.5) | 0.32 (26) |
| **Pork** | 2.6 (120) | 0.84 (34) | 1.2 (53) | 4.6 (200) |
| **Chicken** | 2.3 (100) | 0.91 (40) | 1.3 (56) | 4.5 (200) |
| **Liver** | | | | |
| **Beef liver** | 7.0 (260) | 2.2 (83) | 11 (400) | 20 (740) |
| **Lamb liver** | 0.48 (32) | 0.26 (17) | 0.6 (41) | 1.3 (90) |
| **Pork liver** | 3.3 (110) | 0.89 (30) | 3.7 (120) | 7.9 (270) |
| **Chicken liver** | <0.18 (<8.0) | <0.18 (<8.0) | <0.12 (<5.4) | <0.48 (<22) |
| **Fish** | | | | |
| **Salmon** | 7.3 (490) | 2.9 (220) | 1.7 (120) | 12 (830) |
| **Mackerel** | 2.4 (490) | 0.2 (40) | 1.0 (210) | 3.6 (740) |
| **Tuna** | 12 (110) | 2.2 (50) | 1.8 (18) | 16 (180) |
| **Trout** | 6.0 (350) | 1.2 (72) | 1.6 (90) | 8.8 (510) |
| **Egg and dairy products** | | | | |
| **Cheese** | <0.089 (<7.8) | <0.089 (<8.0) | <0.059 (<5.4) | <0.24 (<43) |
| **Egg** | 0.78 (190) | 0.28 (68) | 0.19 (45) | 1.3 (300) |

### 5.2.4 Relative abundance of different classes of FRs

As shown in Figure 5.2, PBDEs were the predominant FR class found in meat, fish, and egg and dairy products, contributing 44%, 46% and 52% of ΣFRs, respectively. In contrast, EFRs were more prevalent in liver samples, accounting for 81% of ∑FRs. This suggests the mechanisms of uptake, metabolism and excretion of different chemicals varies between food groups, and requires more detailed study. Furthermore, the relatively high levels of ΣEFRs found in our food samples requires attention given their propensity for bioaccumulation, long-range transport and toxicity (Bearr et al., 2010; Howard and Muir, 2010; Larsson et al., 2006; Mankidy et al., 2014; Pradhan et al., 2013; Wang et al., 2010; Harju et al., 2009).

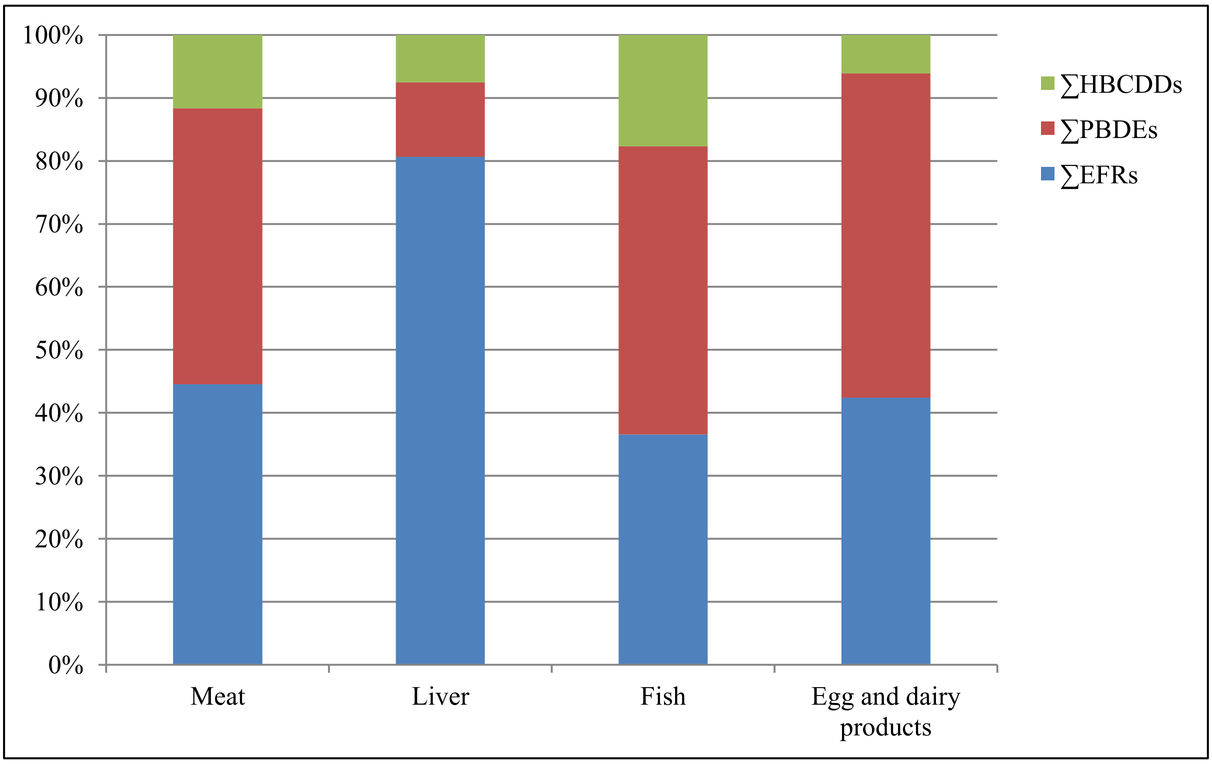


**Figure 5.2 Relative contributions of EFRs, PBDEs and HBCDDs to ΣFRs in UK food samples**

## 5.3 Estimation of daily dietary intakes

Estimated dietary intakes of FRs were calculated for toddlers and adults based on nutrient

**Table 5.6 Average (standard deviation in parentheses) quantities of food consumed by UK toddlers and adults (g/day) (Food Standards Agency, 2014)**

| | Toddlers [a] | Adults [b] |
|---|---|---|
| **Beef** | 17 (28) | 48 (71) |
| **Lamb** | 4 .1 (15) | 12 (39) |
| **Pork** | 3.1 (13) | 11 (32) |
| **Chicken** | 14 (21) | 61 (77) |
| **Beef liver** | 0.04 (0.44) | 0.34 (2.3) |
| **Lamb liver** | 0.04 (0.44) | 0.34 (2.3) |
| **Pork liver** | 0.04 (0.44) | 0.34 (2.3) |
| **Chicken liver** | 0.04 (0.44) | 0.34 (2.3) |
| **Salmon** | 0.58 (3.8) | 2.7 (7.7) |
| **Mackerel** | 0.58 (3.8) | 2.7 (7.7) |
| **Tuna** | 0.58 (3.8) | 2.7 (7.7) |
| **Trout** | 0.58 (3.8) | 2.7 (7.7) |
| **Cheese** | 8.6 (9.4) | 15 (19) |
| **Egg** | 8.5 (14) | 20 (38) |

a) derived from data of food consumption for age group (1.5-3 years old); b) . derived from data of food consumption for age group (19-64 years old).

intake data from the latest national diet and nutrition survey report published by Public Health England and the Food Standards Agency (2014) for different ages (Table 5.6). Consumption rates for each liver (four groups) and each oily fish (four groups) category were derived from the total liver and oily fish consumptions in the survey report divided by 4 (Food Standards Agency, 2014). The weight of each egg used for calculating daily intakes of FRs via egg consumption was corrected for the corresponding eggshell weight and concentrations on a whole egg basis as reported in one previous study (Labunska et al., 2013). Dietary intakes were calculated by multiplying food consumption rates for both average ("typical") and high-end consumers (the latter assumed to be those consuming the average consumption rate + 2 standard deviations) by average concentrations. It is notable that raw food samples were analysed in our study as preparation and cooking may affect the concentrations of chemicals (Perelló et al., 2009). Total dietary exposures to EFRs, PBDEs, and HBCDDs are compared in Figure 5.3.

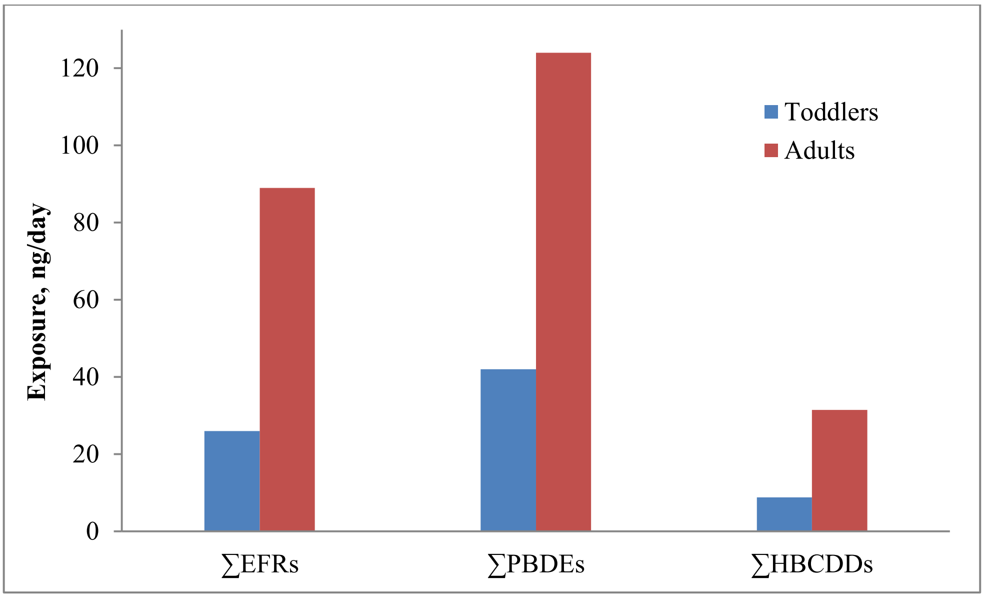


**Figure 5.3 Estimated average dietary exposures to EFRs, PBDEs, and HBCDDs**

### 5.3.1 EFRs

The estimated mean dietary intakes of ∑EFRs in the UK were 26 and 89 ng/day for toddlers and adults, respectively (Table 5.7). Estimated high-end daily ∑EFRs intakes via diet were one order of magnitude higher than the mean dietary intakes for toddlers and adults (Table 5.7). The main contributor to human dietary exposure to ∑EFRs was meat, followed by fish for both toddlers and adults (Figure 5.4). This is the first report of dietary exposure to EFRs in the UK. Furthermore, very limited information on human dietary exposure to EFRs has been reported anywhere to date. Labunska et al. (2015) investigated human dietary exposure to EFRs arising through consumption of foodstuffs from an e-waste recycling area in

**Table 5.7 Estimated average and high-end [a] intakes of ∑EFRs (ng/day) via consumption of food for UK adults and toddlers**

| | Toddlers | | Adults | |
|---|---|---|---|---|
| | Average consumer | High-end consumer | Average consumer | High-end consumer |
| **Meat** | 18 | 100 | 64 | 294 |
| **Liver** | 0.42 | 10 | 3.8 | 56 |
| **Fish** | 2.4 | 34 | 11 | 76 |
| **Cheese** | 3.6 | 11 | 6.3 | 22 |
| **Egg** | 1.7 | 7.3 | 4.0 | 19 |
| **Total** | 26 | 162 | 89 | 467 |

a) estimates of high end intakes were derived from food consumption figures in Table 5.6 by assuming that a high-end consumer of each food group consume the average quantities of food consumed + 2 × standard deviations (SD). This is because statistically the 95th percentile value equals average plus 2 × SD.

Taizhou, eastern China. Estimated dietary exposures to EFRs for both adults and children were 756 and 1827 ng/day, respectively, which is much higher than those in our study (Labunska et al., 2015). Tao et al. (2016) reported that estimated dietary exposure of EFRs for adults and children from an e-waste processing area in Vietnam were 8670 and 6153 ng/day, respectively, which is also extremly higher than our study. β-DBE-DBCH showed the highest estimated intakes of ∑EFRs in this study for both adults and children (14 and 50 ng/day respectively) followed by DBDPE (4.9 and 16 ng/day respectively). As DBE-DBCH were not investigated in Labunska et al.'s study (2015), it is impossible to compare these two studies directly. We are aware of another study on dietary exposure to EFRs for Swedish mothers and toddlers in which EFRs were only detected in fish (Sahlström et al., 2015). The estimated median daily intakes of EFRs were 6.8 and 3.3 ng/day in Sahlström et al.’s study (2015) which is one order of magnitude lower than our total EFRs dietary exposure estimates but similar to our estimated daily intakes of EFRs through fish consumption (10 and 2.2 ng/day, respectively).

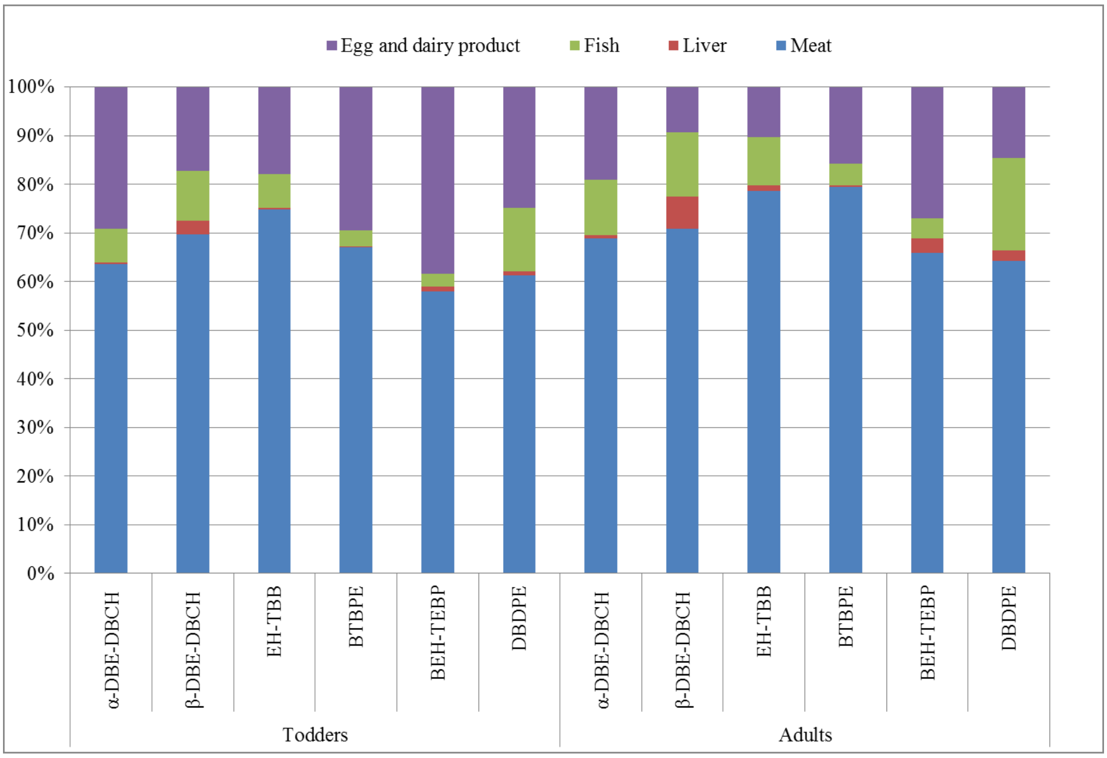


**Figure 5.4 Contributions to average total dietary exposures for EFRs from different groups of UK food**

### 5.3.2 PBDEs

Tables 5.8 and 5.9 show the estimated intakes of ∑PBDEs via consumption of food in the UK and other different countries. Total average daily intakes of ∑PBDEs were estimated in this study to be 42 and 124 ng/day for toddlers and adults, respectively, which is comparable to the adults' dietary intake of PBDEs in Spain (Domingo et al., 2008) but higher than those in the USA (Schecter et al., 2009), Belgium (Stefan Voorspoels et al., 2007), Netherlands (Bakker et al., 2008), Sweden (Törnkvist et al., 2011; Sahlström et al., 2015), China (Su et al., 2012) and Romania (Dirtu and Covaci, 2010). Notably, estimated high-end dietary intakes of ∑PBDEs were 5 times higher than the mean dietary intakes for both toddlers and adults (227 ng/day and 568 ng/day, respectively). In spite of the elevated levels of PBDEs in food samples in this study compared with previous UK studies (UK Food Standards Agency, 2006; UK Food and Environment Research Agency, 2009), the average daily intake of PBDEs was

**Table 5.8 Estimated average and high-end [a] intakes of ∑PBDEs (ng/day) via consumption of food for UK adults and toddlers**

| | Toddlers | | Adults | |
|---|---|---|---|---|
| | **Average consumer** | **High-end consumer** | **Average consumer** | **High-end consumer** |
| **Meat** | 28 | 135 | 89 | 372 |
| **Liver** | 0.08 | 1.8 | 0.61 | 8.9 |
| **Fish** | 4.4 | 61 | 20 | 133 |
| **Cheese** | 8.1 | 26 | 14 | 48 |
| **Egg** | 0.67 | 2.9 | 1.2 | 5.8 |
| **Total** | 42 | 227 | 124 | 568 |

a) estimates of high end intakes were derived from food consumption figures in Table 5.6 by assuming that a high-end consumer of each food group consume the average quantities of food consumed + 2 × standard deviations (SD). This is because statistically the 95th percentile value equals average plus 2 × SD.

found to be comparable to one previous study (Harrad et al., 2004) but lower than another one (UK Food Standards Agency, 2006). This may be attributable to the fact that Harrad et al's study (2004) was a duplicate diet study, while the study of the Food Standards Agency (2006) included more types of food samples (n=19) compared with the more limited range of animal-based sample types collected in our study. Meat was the main source of PBDEs for both toddlers and adults in our study, contributing > 58% of estimated daily intake, which is similar to previous studies conducted in China (Su et al., 2012) and Romania (Dirtu and Covaci, 2010). By comparison in the previous UK study (UK Food Standards Agency, 2006) meat products were the principal contributor which may also account for the relatively higher daily intake of PBDEs in this previous study compared to our study. However, fish was the predominant contributor to human exposure of PBDEs in several other countries such as Sweden (Törnkvist et al., 2011), Belgium (Stefan Voorspoels et al., 2007), and Spain (Domingo et al., 2008).

**Table 5.9 Estimated average adult intakes of ∑PBDEs (ng/day) via consumption of food in different countries**

| Country | Total PBDEs intake (ng/day) | Year | References |
|---|---|---|---|
| **UK** | 124 | 2015 | This study |
| **UK** | 413 | 2003-2004 | (UK Food Standards Agency, 2006) |
| **UK** | 107 (90.5 [a]) | 2006 | (Harrad et al., 2004) |
| **USA** | 50 | 2009 | (Schecter et al., 2009) |
| **Belgium** | 23-48 | 2005 | (Stefan Voorspoels et al., 2007) |
| **Spain** | 75.4 | 2006 | (Domingo et al., 2008) |
| **Netherlands** | 55.3 | 2003-2004 | (Bakker et al., 2008) |
| **Sweden** | 49 | 2005 | (Törnkvist et al., 2011) |
|  | 22 | 2010 | (Sahlström et al., 2015) |
| **China** | 9.9 | 2006 | (Su et al., 2012) |
| **Romania** [a] | 40 | 2007 | (Dirtu and Covaci, 2010) |

a) Based on median values of PBDEs and consumption of omnivorous diets only .

### 5.3.3 HBCDDs

Estimated daily intakes of ∑HBCDDs through food for adults and toddlers in the UK are shown in Table 5.10. The average dietary exposure to ∑HBCDDs is 8.8 and 31 ng/day for toddlers and adults, respectively. Estimated high-end daily ΣHBCDDs intakes (54 ng/day and 149 ng/day for toddlers and adults, respectively) via food consumption were significantly higher than the mean dietary intakes. Similar to PBDEs, the adults' daily intake estimation of ∑HBCDDs in this study was lower than that in a previous study (UK Food Standards Agency, 2006) even though the levels of ∑HBCDDs in this study were comparable to this previous study. This difference is likely due to the wider range of food groups analysed in the earlier study. Compared with other countries, our estimate of UK adult dietary intake of HBCDDs is comparable to those in the USA (Schecter et al., 2009), Sweden (Törnkvist et al.,

**Table 5.10 Estimated average and high-end [a] intakes of ∑HBCDDs (ng/day) via consumption of UK food for adults and toddlers**

| | **Toddlers** | | **Adults** | |
|---|---|---|---|---|
| | **Average consumers** | **High-end consumers** | **Average consumers** | **High-end consumers** |
| | **Mean** | **Mean** | **Mean** | **Mean** |
| **Meat** | 6.4 | 30 | 23 | 93 |
| **Liver** | 0.04 | 1.0 | 0.37 | 5.5 |
| **Fish** | 1.3 | 18 | 6.1 | 41 |
| **Cheese** | 0.18 | 0.6 | 0.32 | 1.1 |
| **Egg** | 0.79 | 3.4 | 1.9 | 8.8 |
| **Total** | 8.8 | 54 | 31 | 149 |

a) estimates of high end intakes were derived from food consumption figures in Table 5.6 by assuming that a high-end consumer of each food group consumes the average quantities of food consumed + 2 × standard deviations (SD). This is because statistically the 95th percentile value equals average plus 2 × SD.

2011) and China (Shi et al., 2009); but lower than those in Belgium (Goscinny et al., 2011), Spain (Eljarrat et al., 2014), the Netherlands (de Winter-Sorkina R et al., 2003) and Romania (Dirtu and Covaci, 2010) (Table 5.11). The calculated contributions of the different food groups to the total estimated daily intake of HBCDDs showed that meat was a predominant contributor for both toddlers and adults, accounting for 73% and 74% of the total exposure to HBCDDs, respectively, which is similar to the findings in the USA (Schecter et al., 2009), Belgium (Goscinny et al., 2011), Netherland (de Winter-Sorkina et al., 2003), China (Shi et al., 2009) and Romania (Dirtu and Covaci, 2010), followed by fish (13% and 20% for toddlers and adults, respectively). In contrast, milk and fruit were the main contributors to dietary exposure to HBCDDs in a previous UK study (Food Standards Agency, 2006), while in Spain (Eljarrat et al., 2014) and Sweden (Törnkvist et al., 2011), fish was the main source of dietary intake of HBCDDs.

**Table 5.11 Estimated average intakes of ∑HBCDDs (ng/day) via consumption of food in different countries**

| Country | Total HBCDDs intake (ng/day) | Year | References |
|---|---|---|---|
| **UK** | 31 | 2015 | This study |
| **UK** | 413 [a] | 2003-2004 | (UK Food Standards Agency, 2006) |
| **USA** | 16 | 2009 | (Schecter et al., 2009) |
| **Belgium** [a, b] | 69 | 2006-2007 | (Goscinny et al., 2011) |
| **Spain** | 177 | 2009 | (Eljarrat et al., 2014) |
| **Netherlands** | 99-191 | 2002 | (de Winter-Sorkina et al., 2003) |
| **Sweden** | 10.2 | 2005 | (Törnkvist et al., 2011) |
|  | 11 | 2010 | (Sahlström et al., 2015) |
| **China** | 27 | 2007 | (Shi et al., 2009) |
| **Romania** [a] | 77 | 2007 | (Dirtu and Covaci, 2010) |

a) assuming the body weight was 70 kg for daily intake estimation calculation.

## 5.4 Conclusions

This study reveals the presence of EFRs in various UK food samples. Meat was the main source of dietary intakes of PBDEs, EFRs and HBCDDs for both toddlers and adults under an average consumer scenario. Estimated dietary exposures to EFRs were comparable to dietary intakes of PBDEs but higher than those of HBCDDs for both toddlers and adults (Figure 5.4). This may be a health concern as some EFRs show similar persistence, bioaccumulation potential and toxicity (PBT) properties to legacy FRs (Johnson et al., 2013; Muir and Howard, 2006; Stieger et al., 2014; Tomy et al., 2007a), exacerbated by likely future increases in use of EFRs due to the banned and restricted use of legacy FRs (Stockholm Convention, 2009; European Court of Justice, 2008; UNEP, 2014).

# CHAPTER 6 EFRs, PBDEs and HBCDDs in UK human milk: implications for infant exposure and relationship to external exposure

## 6.1 Introduction

EFRs have been widely detected in environmental matrices including indoor air, indoor dust, outdoor air, and sediment as well as biological matrices. (Cequier et al., 2014; Fernandes et al., 2010; Gauthier et al., 2008; Guerra et al., 2012; Karlsson et al., 2007; Labunska et al., 2015; Law et al., 2006; Li et al., 2015; Möller et al., 2011a, 2011b; Munschy et al., 2011; Newton et al., 2015; Salamova et al., 2014; Shi et al., 2009; Yang et al., 2012). In contrast, very little information is available to estimate the production volume of EFRs. Figures available put worldwide production/usage of BTBPE at an estimated 16,710 tons in 2001 (Covaci et al., 2011) which exceeds the estimated total consumption of Penta-BDEs (7,500 tons) and Octa-BDEs (3,790 tons), and is one third of the estimated total consumption of Deca-BDEs (56,100 tons) in 2001 (BSEF, 2006) and around half that of HBCDD (~28,000 tons) (Yang, 2014). With respect to other EFRs, around 1,000 to 5,000 tons of DBDPE were imported to the EU, primarily to Germany in 2001 (Covaci et al., 2011), with BEH-TEBP and EH-TBB also extensively used in additive FR products such as Firemaster 550, BZ54 and DP-45 (Covaci et al., 2011). Of concern is the fact that some EFRs (e.g. DBE-DBCH and BTBPE) display persistence and bioaccumulative potential giving rise to increasing concern over their adverse human health effects (Muir and Howard, 2006; Tomy et al., 2007a). In one study, concentrations of BEH-TEBP and DBDPE in indoor dust were both positively associated with total T3 in exposed men, suggesting that exposure to these contaminants in indoor dust may be leading to endocrine disruption in males (Johnson et al., 2013). Moreover, animal and *in vitro* studies have shown some EFRs to pose potential health risks including: endocrine disruption (DBDPE, BTBPE, EH-TBB, BEH-TEBP and DBE-DBCH), neurodevelopmental and behavioural outcomes (EH-TBB, BEH-TEBP), hepatotoxicity (DBDPE), impaired reproductive physiology (DBDPE and DBE-DBCH), gene expression (BTBPE), morphological abnormalities and mortality (DBE-DBCH), DNA damage (EH-

TBB, BEH-TEBP and DBE-DBCH) and possible cancer (DBE-DBCH) (Bearr et al., 2010; Egloff et al., 2011; Ezechiáš et al., 2012; Johnson et al., 2013; Khalaf et al., 2009; Larsson et al., 2006; Mankidy et al., 2014; McGregor et al., 1991; Nakari and Huhtala, 2009; Noyes et al., 2015; Park et al., 2011; Pradhan et al., 2013; Saunders et al., 2013; Wang et al., 2010).

The relationships between levels of legacy BFRs including PBDEs and HBCDDs in human matrices and external exposure have been reported in several previous studies (Abdallah and Harrad, 2014, 2011; Roosens et al., 2009a). However, very few data exist about the presence of EFRs in human matrices and the extent to which the known contamination of indoor environments with EFRs influences human body burdens. Therefore, this study measures the concentrations of EFRs in UK human milk for the first time and uses a simple one-compartment pharmacokinetic model to predict the body burdens of the studied EFRs in UK adults and compare these predicted burdens to the concentrations detected in human milk.

## 6.2 Concentrations and patterns of FRs in human milk

### 6.2.1 EFRs

Table 6.1 shows a statistical summary of concentrations of our target EFRs in archived human milk samples (collected in 2010) (human milk group 1) and human milk samples collected from 2014-2015 (human milk group 2). No significant differences were found between the levels of individual EFRs in human milk group 1 and group 2 ($p > 0.05$). Moreover, the DFs of all EFRs in human milk group 1 were lower than those in human milk group 2 even though the sample size of the latter is smaller than the former, indicating the more widespread usage of these EFRs currently than hitherto. The DFs of DBDPE and BTBPE were low (<50%) in both human milk groups which is similar to the study of Zhou et al. (2014) even though these two chemicals were frequently detected in UK indoor dust samples at high concentrations (seen in Chapter 4, Table 4.1). Given its similar structure to

BDE 209, the physicochemical properties and behaviour in the environment and biota of DBDPE were assumed to be in line with that of BDE 209 which has very low bioavailability and a high biotransformation rate (Abdallah and Harrad, 2014; Wan et al., 2013). However, to the best of our knowledge, no studies have been conducted to investigate the bioavailability and biotransformation of DBDPE and BTBPE in human tissues. Both DBDPE and BTBPE have been reported to have a high bioaccumulation potential in fish (He et al., 2012; Tomy et al., 2007a). In contrast, DBDPE was found to be less bioaccumulative in rats (Wang et al., 2010) and chickens (Zheng et al., 2015) compared to BDE 209, while several oral ingestion studies of BTBPE in rats also reported poor gastrointestinal absorption, high transformation and substantial faecal excretion (>94%) (Nomeir et al., 1993; Verreault et al., 2007; Hakk et al., 2004). Furthermore, biotransformation of DBDPE and BTBPE was found to occur in rats (Wang et al., 2010; Verreault et al., 2007), with greater depletion of DBDPE (44-74% of 90 pmol) observed in various arctic marine-feeding mammals and in laboratory rats relative to BDE 209 (14-25% of 30 pmol) in such species (Mckinney et al., 2011b). Such findings for DBDPE and BTBPE in the mammalian model species may suggest these two chemicals have low bioaccessibility and relatively high biotransformation potential. Combined, these factors may explain the low DFs of these chemicals in human milk. Interestingly, very high levels of BTBPE (56 and 54 ng/g lw) were found in two archived human milk samples which may reflect recent human exposure to BTBPE. Moreover, a very high concentration of 4,700,000 ng/g BTBPE was found in one UK dust sample (Chapter 4, Table 4.1), so another explanation for the high levels of BTBPE in these two human milk samples may be relatively high dust exposure. The DFs of EH-TBB were higher than BEH-TEBP which is in line with Zhou et al.'s study (2014) and may be attributable to the higher bioaccessibility of EH-TBB compared to BEH-TEBP (Fang and Stapleton, 2014), or possibly explained by preferential partitioning of EH-TBB from blood to milk in humans relative to BEH-TEBP (Zhou et al., 2014). Similar observations were made by Liu et al. (2016), i.e. that EH-TBB was detected more frequently than BEH-TEBP in human hair, fingernails, toenails and serum especially in human serum. Specifically, EH-TBB was detected in 92% of the serum samples, while BEH-TEBP was detected in only 16%, with BTBPE present in <15% of samples. In our study, of all targeted compounds, β-DBE-DBCH showed the highest DFs and concentrations in both human milk groups which is similar to the findings in UK diet samples (Chapter 5, Table 5.1) indicating the high bioaccumulative potential in human milk. To our knowledge, this is the first report of β-DBE-DBCH in human milk samples. The values of $f_{\beta\text{-DBE-DBCH}}$ ranged from 0.88 to 608, with a median of 9.6 which is significantly higher than those in UK indoor air (0.53-1.0) ($p < 0.001$) and dust (0.32-2.88) ($p < 0.001$) indicating diastereomer-specific environmental degradation/metabolism and/or isomerisation in humans. Of note, $f_{\beta\text{-DBE-DBCH}}$ values in human milk were statistically indistinguishable to those in diet samples ($p > 0.05$) indicating the relatively higher abundance of β-DBE-DBCH compared with α-DBE-DBCH may also result from dietary exposure to DBE-DBCH.

Despite the ubiquity of EFRs in the environment, very few studies have reported the presence of EFRs in humans. In this study, the only compound detected in >50% of samples in our two human milk groups was β-DBE-DBCH, with concentrations statistically indistinguishable between the two groups. In Sahlström et al.'s study (2015), only α-DBE-DBCH were found above the detection limit in the two pooled breast milk samples in Sweden with mean concentrations at 4.0 pg/g ww which is lower than in our study (mean: 41 and 24 pg/g ww in human milk group 1 and group 2, respectively). In North America, Zhou et al. (2014) measured several EFRs including EH-TBB, BEH-TEBP, BTBPE, and DBDPE in human milk (n=105) collected in 2008-2009 in Canada. The concentrations reported of EH-TBB ranged between nd-24 ng/g lw which is higher than in our study (Table 6.1), while

**Table 6.1 Descriptive statistics for concentrations of EFRs in UK human milk (ng/g lw) [a]**

| | Lipid weight (%) | α-DBE-DBCH | β-DBE-DBCH | EH-TBB | BTBPE | BEH-TEBP | DBDPE | ∑EFRs |
|---|---|---|---|---|---|---|---|---|
| **Archived human milk samples collected in 2010 (n=25)** | | | | | | | | |
| **DF** | | 20% | 76% | 44% | 28% | 36% | 4% | |
| **5th percentile** | 2.1 | <0.13 | 0.063 | <0.1 | <0.1 | <0.1 | <0.78 | 0.76 |
| **95th percentile** | 4.3 | 1.1 | 34 | 2.0 | -- | 1.9 | -- | -- |
| **Mean** | 3.2 | -- | 6.8 | -- | -- | -- | -- | -- |
| **Median** | 3.5 | <0.13 | 3.1 | <0.01 | <0.1 | <0.1 | <0.78 | 7.9 |
| **Minimum** | 1.9 | <0.13 | <0.13 | <0.01 | <0.1 | <0.1 | <0.78 | 0.57 |
| **Maximum** | 4.4 | 1.7 | 38 | 2.1 | 56 | 4.6 | 250 | 260 |
| **Human milk collected in 2014-2015 (n=10)** | | | | | | | | |
| **DF** | | 100% | 100% | 90% | 40% | 50% | 10% | |
| **5th percentile** | 2.2 | 0.34 | 0.47 | 0.041 | <0.1 | <0.1 | <0.78 | 2.0 |
| **95th percentile** | 5.2 | 1.0 | 8.3 | 0.40 | 0.50 | 0.65 | -- | -- |
| **Mean** | 3.9 | 0.67 | 2.5 | 0.21 | -- | 0.25 | -- | -- |
| **Median** | 4.1 | 0.60 | 1.2 | 0.16 | <0.1 | <0.1 | <0.78 | 3.1 |
| **Minimum** | 1.5 | 0.30 | 0.43 | <0.01 | <0.1 | <0.1 | <0.78 | 1.9 |
| **Maximum** | 5.3 | 1.1 | 10 | 0.48 | 0.71 | 0.73 | 58 | 59 |

a) the mean concentrations were calculated when the DF>50%.

concentrations of BEH-TEBP and DBDPE were nd-6.6 and nd-25 ng/g lw, respectively, comparable to those in our study (Table 6.1). With respect to BTBPE, while detected in the minority of samples in our study, it was not detected in human milk in the Canadian study (Zhou et al., 2014). Our concentrations of EH-TBB and BEH-TEBP in human milk were much lower than those detected in human hair, fingernails and toenails (EH-TBB: 7.6-4540 ng/g; BEH-TEBP: 13-2600 ng/g) and also serum (TBB: 1.3-54 ng/g lw; BEH-TEBP: 19-69 ng/g) in the USA population (Liu et al., 2016). Moreover, while BTBPE was undetectable in serum in the USA study, concentrations in our study were exceeded by those in human hair, fingernails and toenails (BTBPE: 0.75-8.7 ng/g) in the USA study (Liu et al., 2016).

### 6.2.2 PBDEs

The concentrations of PBDEs from human milk group 2 are shown in Table 6.2. The levels of PBDEs in human milk group 1 (n=35) were reported by our group in a previous study (Abdallah and Harrad, 2014). The mean concentrations of Σtri-hexa BDEs in this study (6.5 ng/g) are comparable to those reported in UK human milk samples collected in 2010 (n=54, average: 5.9 ng/g lw) from human milk group 1 (Abdallah and Harrad, 2014) and in Australian human milk samples (7.6 ng/g lw) collected in 2007 (Toms et al., 2009). However, concentrations in our study are still slightly higher than those in most Asian and European countries such as: China (0.8 ng/g lw), India (1.1 ng/g lw), Korea (2.7 ng/g lw), France (2.5 ng/g lw), Spain (2.1 ng/g lw), Italy (1.3 ng/g lw), Norway (2.7 ng/g lw), Sweden(2.2 ng/g lw) and Belgium (3.0 ng/g lw) (Alivernini et al., 2011; Antignac et al., 2009; Devanathan et al., 2012; Gómara et al., 2011; Kim et al., 2011; Roosens et al., 2010; Shi et al., 2013; Thomsen et al., 2010; Sahlström et al., 2015). However, concentrations reported here, are significantly lower than those in the USA (34 ng/g lw) (Schecter et al., 2003) and Canada (50.4 ng/g lw) (She et al., 2007). Concentrations of BDE 209 (< 0.22 ng/g lw) in this study were lower than in other European countries including Belgium (5.9 ng/g lw), France (1.6 ng/g lw) and Spain (2.5 ng/g lw) (Antignac et al., 2009; Gómara et al., 2011; Roosens et al., 2010) but similar to Sweden (0.33 ng/g lw) (Sahlström et al., 2015). Similar to the previous UK study (Abdallah and Harrad, 2014), BDE 47, 99, and 153 were the most abundant compounds in this study, in the order: BDE 47 > BDE 153 > BDE 99. The relatively higher levels of BDE 153 than BDE 99 in this study were in agreement with several previous studies (Abdallah and Harrad, 2014; Dunn et al., 2010; Frederiksen et al., 2009; Hassine et al., 2012). By comparison, in UK indoor air, dust (Chapter 4, Table 4.2 and 4.4) and diet samples (Chapter 5, Table 5.3) concentrations of BDE 153 are much lower than those of BDE 99, indicating that the external exposures through indoor air, dust and diet were not the reason for the elevated abundance of BDE 153 in human milk. Therefore, the relatively higher levels of BDE 153 compared with BDE 209 in human milk are likely due to the higher bioaccumulation potential of BDE 153 in humans and/or the possible stepwise debromination from BDE 209 to BDE 153 (Abdallah and Harrad, 2014), consistent with the significant correlations between the levels of BDE 153 and BDE 209 in this study ($p < 0.05$).

The concentrations of BDE 47, 100, 99, 154, and 153 in human milk group 2 were comparable to the previous study for human milk group 1 (Table 6.2) (Abdallah and Harrad, 2014). Moreover, Schuhmacher et al. (2009) and Shy et al. (2012) also reported no time trend for ΣPBDEs (except BDE 209) in human milk samples in Spain and Taiwan, respectively (2002-2007 and 2000/2001-2007/2009 for Spain and Taiwan, respectively). Similarly, concentrations of PBDEs in Canadian human milk appear to have stabilized between 2002 and 2005 (Ryan and Rawn, 2014). As diet and dust are the main pathways of external human exposure to PBDEs (Harrad et al., 2004, 2008; Lorber, 2008), it is of interest that while levels of Σtri-hexa BDEs in our UK food samples are higher than those reported in two previous UK food surveys (Chapter 5, Table 5.4), no significant temporal differences for Σtri-hexa BDEs were found in dust (Chapter 4, section 4.7.2). The contrasting trends in diet and dust levels of tri-hexa BDEs are consistent with the hypothesis put forward in Harrad's study (2006) suggesting that both dust- and diet-related exposure exert strong influences on UK human body burdens for this period. Therefore, it is possible that the steady human body burden levels observed here are attributable to a combination of: (a) increased dietary exposure, offset by (b) decreased dust-related exposure to BDE 209 (Chapter 4, section 4.6.2) followed by *in vivo* metabolism to tri-hexa-BDEs (Stapleton et al., 2004; Van den Steen et al., 2007; Wang et al., 2010). The levels of BDE 209 in human milk group 2 are comparable to those in group 1 (Abdallah and Harrad, 2014) (Table 6.2).

## Table 6.2 Descriptive statistics for concentrations for PBDEs in UK human milk (ng/g lw)

| | BDE 28 | BDE 47 | BDE 100 | BDE 99 | BDE 154 | BDE 153 | Σtri-hexa BDEs | BDE 209 |
|---|---|---|---|---|---|---|---|---|
| **Archived human milk samples collected in 2010 (n=35) (Abdallah and Harrad, 2014)** | | | | | | | | |
| **DFs** | -- | 100% | 89% | 94% | 77% | 97% | -- | 69% |
| **5th percentile** | -- | 0.27 | 0.03 | 0.05 | 0.03 | 0.09 | 0.58 | 0.03 |
| **95th percentile** | -- | 8.23 | 0.98 | 1.7 | 0.68 | 3.16 | 12.52 | 0.82 |
| **Mean** | -- | 3.3 | 0.45 | 0.71 | 0.30 | 1.10 | 5.9 | 0.31 |
| **Median** | -- | 2.8 | 0.38 | 0.69 | 0.21 | 0.91 | 5.00 | 0.25 |
| **Min** | -- | 0.17 | <0.05 | <0.06 | <0.06 | <0.06 | 0.20 | <0.06 |
| **Max** | -- | 14.65 | 1.86 | 3.43 | 11.10 | 4.57 | 26.10 | 0.92 |
| **Human milk collected from 2014-2015 (n=10)** | | | | | | | | |
| **DFs** | 90% | 100% | 100% | 100% | 90% | 100% | -- | 40% |
| **5th percentile** | 0.03 | 0.63 | 0.08 | 0.45 | 0.06 | 0.62 | 2.4 | 0.08 |
| **95th percentile** | 0.41 | 7.0 | 2.1 | 1.7 | 0.21 | 2.4 | 13 | 0.47 |
| **Mean** | 0.19 | 2.8 | 0.73 | 1.0 | 0.13 | 1.7 | 6.5 | <0.22 |
| **Median** | 0.14 | 2.2 | 0.53 | 0.77 | 0.12 | 1.8 | 5.8 | <0.22 |
| **Min** | <0.03 | 0.52 | 0.07 | 0.42 | <0.03 | 0.49 | 1.7 | <0.22 |
| **Max** | 0.41 | 7.7 | 2.2 | 2.0 | 0.24 | 2.7 | 14 | 0.67 |

Fängström et al. (2008) and Shy et al. (2012) also reported no time trend for BDE 209 in human milk samples in Sweden and Taiwan respectively (1980-2004 and 2000/2001-2007/2009 for Sweden and Taiwan, respectively). Consistent with this, no substantial differences were found between concentrations of BDE 209 in this study and in two UK previous dietary studies (Chapter 5, Table 5.4) (UK Food Standards Agency, 2006; UK Food and Environment Research Agency, 2009). In contrast, concentrations of BDE 209 in UK office dust decreased significantly compared with those in a previous UK study (Harrad et al., 2008a) (Chapter 4, section 4.6.2). Consequently, the steady temporal trend in BDE 209 levels in human milk suggests that concentrations of this congener in dust exert a relatively minor influence on body burdens. This may be attributable to the very low bioaccessibility from dust (~14%) of BDE 209, combined with its very short human half-life of and preferential partitioning to serum rather than milk fat (Abdallah and Harrad, 2014).

### 6.2.3 HBCDDs

Concentrations of ∑HBCDDs in human milk group 2 samples ranged between 0.69-7.1 ng/g lw (Table 6.3) i.e. slightly lower than those in UK human milk group 1 from a previous study (Abdallah and Harrad, 2011). α-HBCDD was the predominant HBCDD diastereomer contributing on average 62±11% ∑HBCDDs in all human milk samples, with γ-HBCDDs comprising 32±17% of ΣHBCDDs, in agreement with human milk group 2 (Abdallah and Harrad, 2011), which was suggested to be a result of preferential metabolism of β- and γ-HBCDDs (Zegers et al., 2005) and greater bioavailability of α-HBCDD compared to the other isomers (Abdallah et al., 2009). As mentioned in Chapter 4 (section 4.6.3) and Chapter 5 (section 5.2.3), concentrations of ΣHBCDD in food samples in this thesis were comparable to those detected in similar foodstuffs in two UK previous studies (Driffield et al., 2008; UK Food Standards Agency, 2006). By comparison, the levels of ΣHBCDDs in indoor air and dust measured in this thesis show a decreasing trend which may account for the slight downward trend observed in UK human milk. Concentrations of HBCDDs in group 2 samples (mean: 3.20 ng/g lw) were at the lower end of those reported for other European countries including Norway (<LOQ-31 ng/g lw) (Thomsen et al., 2010), France (2.5-5 ng/g lw) (Antignac et al., 2008), Spain (3-188 ng/g lw) (Eljarrat et al., 2009) and Sweden (<0.04-1.3 ng/g lw) (Lignell et al., 2012) but lower than those reported for Australia (<3.8-19 ng/g lw) (Toms et al., 2012) and Canada (0.1-28.2 ng/g lw) (Ryan and Rawn, 2014). In contrast, concentrations of HBCDDs in UK human milk exceeded those in USA (0.2-0.9 ng/g lw)

(Ryan et al., 2006) and in Asian countries like the Philippines (<0.01-0.91 ng/g lw) (Malarvannan et al., 2013), India (mean: 0.38 ng/g lw) and China (<LOQ-2.8 ng/g lw) (Shi et al., 2009).

**Table 6.3 Descriptive statistics for concentrations for HBCDDs in UK human milk (ng/g lw)**

| | **α-HBCDD** | **β-HBCDD** | **γ-HBCDD** | **∑HBCDDs** |
|---|---|---|---|---|
| **Archived human milk samples collected in 2010 (n=34)** (Abdallah and Harrad, 2011) | | | | |
| **5th percentile** | 1.10 | 0.09 | 0.15 | 1.68 |
| **95th percentile** | 15.27 | 0.67 | 2.11 | 18.00 |
| **Mean** | 4.91 | 0.32 | 0.73 | 5.95 |
| **Median** | 3.71 | 0.30 | 0.56 | 3.83 |
| **Min** | 0.75 | 0.08 | 0.13 | 1.04 |
| **Max** | 19.71 | 0.75 | 2.29 | 22.37 |
| **Human milk collected from 2014-2015 (n=10)** | | | | |
| **DFs** | 100% | 60% | 100% | -- |
| **5th percentile** | 0.65 | <0.10 | 0.29 | 1.1 |
| **95th percentile** | 3.7 | 0.46 | 1.9 | 5.7 |
| **Mean** | 2.1 | 0.25 | 0.90 | 3.2 |
| **Median** | 1.9 | 0.23 | 0.73 | 2.9 |
| **Min** | 0.40 | <0.10 | 0.16 | 0.69 |
| **Max** | 4.4 | 0.61 | 2.2 | 7.1 |

### 6.2.4 Relative abundance of different classes of FRs

As shown in Figure 6.1, PBDEs are the predominant FR class in human milk, contributing

50% ΣFRs, followed by EFRs, accounting for 38% of ΣFRs while ΣHBCDDs contributed 12% to ΣFRs in human milk. Despite the bans and restrictions on the use of PBDE commercial products, PBDEs remain the most abundant class out of our target FRs in human milk which may reflect the continuous release of these "legacy" BFRs from old furniture and appliances and bioaccumulation of these compounds in humans. In future, because of the more widespread usage of EFRs and the biomagnification of these chemicals in the food chain, concentrations of EFRs in humans could increase with consequent potential adverse human health effects.

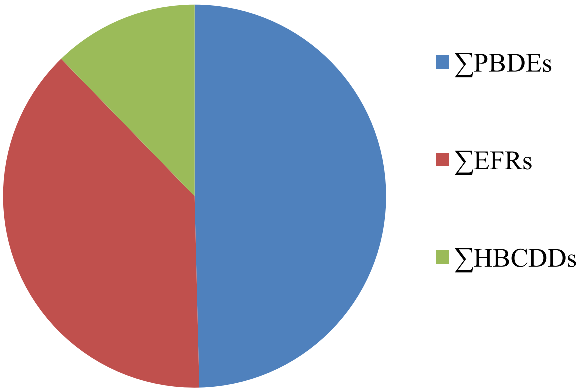


**Figure 6.1 Average relative contributions of EFRs, PBDEs and HBCDDs to ΣFRs in UK human milk**

## 6.3 Nursing infants' dietary intake of HFRs via breast milk

Breast milk is a recognized medium for direct transfer of POPs to nursing infants. We estimated a nursing infants' dietary intake of the studied FRs via breast milk using equation (6.1):

$$\mathrm{Di} = \frac{C_{FRs} * F_{lipid}}{B_W} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (6.1)$$

where Di is the estimated dietary intake (ng/kg bw/day); $C_{FRs}$ is the concentration of target

FRs in milk (ng/g lw); $F_{lipid}$ is the daily lipid intake via breast milk (g/day) and Bw is the body weight (4.14 kg) (U.S. EPA, 2002.). The infant's daily lipid intake via breast milk ($F_{lipid}$) was calculated using U.S. EPA guidelines (U.S. EPA, 2002) which suggest an average intake of 702 mL milk per day for a 1 month old infant weighing 4.14 kg. The median lipid content of the analysed milk samples was 3.47 g lipid per 100 mL of breast milk resulting in a daily lipid intake of 24.4 g lipid/day.

Table 6.4 and Table 6.5 show the estimated dietary intake of target FRs via breast milk under different exposure scenarios. The daily intake estimation of EFRs through breast-feeding from human milk group 1 was compared with the data from human milk group 2. Notably, the median dietary exposure estimate of an infant to ΣEFRs via breast milk was 18 ng/kg bw/day which exceeds estimated dietary intakes of both UK adults (1.1 ng/kg bw/day for 70 kg adults) and toddlers (2 ng/kg bw/day for 10 kg toddlers) (Chapter 5, Table 5.7). Similarly, the estimated dietary exposure of an infant to ΣHBCDDs and Σtri-hexa BDEs via breast milk exceeded the estimated upper-bound dietary intakes of both UK adults and toddlers (Abdallah and Harrad, 2014, 2011).

**Table 6.4 Estimated exposure [a, b] (ng/kg bw/day) of a 1 month old infant to the target FRs via ingestion of breast milk sampled in 2010**

| | 5th percentile | Mean | Median | 95th percentile |
|---|---|---|---|---|
| **α-DBE-DBCH** | 0.38 | -- | 0.38 | 6 |
| **β-DBE-DBCH** | 0.37 | 40 | 18 | 200 |
| **EH-TBB** | 0.29 | -- | 0.29 | 12 |
| **BTBPE** | 0.29 | -- | 0.29 | -- |
| **BEH-TEBP** | 0.03 | -- | 0.03 | 11 |
| **DBDPE** | 2.3 | -- | 2.3 | -- |
| **∑EFRs** | 4.5 | -- | 47 | -- |
| **BDE 47** [c] | 1.6 | 19.3 | 16.3 | 48.4 |
| **BDE 100** [c] | 0.16 | 2.7 | 2.2 | 5.8 |
| **BDE 99** [c] | 0.30 | 4.2 | 4.0 | 10.0 |
| **BDE 154** [c] | 0.17 | 1.7 | 1.3 | 4.0 |
| **BDE 153** [c] | 0.52 | 6.5 | 5.3 | 18.7 |
| **Σtri-hexa BDEs** [c] | 3.4 | 34.9 | 29.4 | 73.8 |
| **BDE 209** [c] | 0.18 | 1.8 | 1.2 | 4.8 |
| **α-HBCDD** [d] | 6.4 | 29 | 18 | 89 |
| **β-HBCDD** [d] | 0.5 | 1.8 | 1.8 | 3.9 |
| **γ-HBCDD** [d] | 0.9 | 4.2 | 3.3 | 12 |
| **∑HBCDDs** [d] | 9.8 | 35 | 22 | 110 |

a) Values below LOQ were assumed to be 1/2 LOQ; b) Based on an average body weight of 4.14 kg and a daily lipid intake of 24.4 g lipid/day (U.S. EPA, 2002); c) Data from Abdallah and Harrad (2014); d) Data from Abdallah and Harrad (2011).

**Table 6.5 Estimated exposure [a, b] (ng/kg bw/day) of a 1 month old infant to the target FRs via ingestion of breast milk sampled in 2014-15**

| | 5th percentile | Mean | Median | 95th percentile |
|---|---|---|---|---|
| **α-DBE-DBCH** | 2.0 | 3.9 | 3.5 | 5.9 |
| **β-DBE-DBCH** | 2.8 | 15 | 7.1 | 49 |
| **EH-TBB** | 0.24 | 1.2 | 0.94 | 2.4 |
| **BTBPE** | 0.29 | 0.88 | 0.29 | 2.9 |
| **BEH-TEBP** | 0.03 | 1.5 | 0.03 | 3.8 |
| **DBDPE** | 2.3 | -- | 2.3 | -- |
| **∑EFRs** | 12 | -- | 18 | -- |
| **BDE 28** | 0.18 | 1.1 | 0.83 | 2.4 |
| **BDE 47** | 3.7 | 17 | 13 | 41 |
| **BDE 100** | 0.47 | 4.3 | 3.1 | 12 |
| **BDE 99** | 2.7 | 5.9 | 4.5 | 10 |
| **BDE 154** | 0.35 | 0.77 | 0.71 | 1.2 |
| **BDE 153** | 3.7 | 10 | 11 | 14 |
| **Σtri-hexa BDEs** | 14 | 38 | 34 | 77 |
| **BDE 209** | 0.47 | 0.65 | 0.65 | 2.8 |
| **α-HBCDD** | 3.8 | 12 | 11 | 22 |
| **β-HBCDD** | 0.29 | 1.5 | 1.4 | 2.7 |
| **γ-HBCDD** | 1.7 | 5.3 | 4.3 | 11 |
| **∑HBCDDs** | 6.5 | 19 | 17 | 34 |

a) based on levels in analysed human milk collected from 2014-2015 (n=10); values below LOQ were assumed to be 1/2 LOQ; b) Based on an average body weight of 4.14 kg and a daily lipid intake of 24.4 g lipid/day (U.S. EPA, 2002)

## 6.4 Relationship between FR intake and human body burdens

To examine the relationship between our estimated intakes via various pathways and the body burdens indicated via levels in human milk, a simple one-compartment, first order pharmacokinetic (PK) model was used (Lorber, 2008; Abdallah and Harrad, 2011). The studied FRs were hypothesized to accumulate in lipids (the single compartment in the model). Therefore, the change in HFR concentrations in lipids over time can be calculated by equation (6.2):

$$\frac{\delta C_{\mathrm{FR}}}{\delta t} = \frac{I_{FR}(\mathrm{t})^{*}AF_{FR}}{BL(t)} - K_{FR} * C_{FR}(t) \text{………………………..} (6.2)$$

where $C_{FR}$ is the compound specific concentration in lipids (ng/g lw); $I_{FR}$ is the daily intake of the target FR (ng/day); $AF_{FR}$ is the absorption fraction; BL is body lipid mass (g) and $K_{FR}$ is the compound specific first order dissipation rate ($\mathrm{day}^{-1}$).

If $K_{FR}$ is assumed constant over time, then equation (6.2) can be changed into:

$$\mathrm{C}_{FR}(\mathrm{t}) = \mathrm{C}_{FR}(0) * e^{(-K_{FR}*t)} + \left[\frac{I_{FR}(\mathrm{t})^{*}AF_{FR}}{BL(t)}\right] * \left[\frac{(1-e^{(-K_{FR}*t)})}{K_{FR}}\right] \text{………………………} (6.3)$$

where $C_{FR}(0)$ is the studied FR body lipid concentration at time 0 (initial concentration before intake). Assuming a constant dose over time at constant body lipid mass (i.e., when t approaches infinity), the steady state BFR lipid concentration can be calculated from equation (6.4):

$$\mathrm{C}_{FR} = \frac{I_{FR}{}^{*}AF_{FR}}{BL*K_{FR}} \text{………………………..} (6.4)$$

While equation (6.4) is used to predict the body burdens of the target FRs, it is stressed that the assumption of steady state conditions is an inherent uncertainty with this approach.

**Table 6.6 Assumed absorption fractions and human half-lives of the individual PBDEs and EFRs**

| EFR | molecular weight | number of bromines | BFR | molecular weight | number of bromines | human half-lives of PBDEs (days) | absorption fractions of PBDEs |
|---|---|---|---|---|---|---|---|
| **DBE-DBCH** | 427.80 | 4 | **BDE 47** | 485.79 | 4 | 1096 [a] | 0.58 [c] |
| **EH-TBB** | 549.90 | 4 | **BDE 47** | 485.79 | 4 | 1096 [a] | 0.58 [c] |
| **BTBPE** | 687.60 | 6 | **BDE 183** | 722.48 | 7 | 94 [b] | 0.90 [d] |
| **BEH-TEBP** | 706.10 | 4 | **BDE 183** | 722.48 | 7 | 94 [b] | 0.90 [d] |
| **DBDPE** | 971.20 | 10 | **BDE 209** | 959.17 | 10 | 15 [b] | 0.14 [c] |

a) Geyer, H. J., Schramm, K.-W., Darnerud, P. O., Aune, M., Feicht, A., Fried, K. W., Mcdonald, T. a. (2004). Terminal elimination half-lives of the brominated flame retardants TBBPA , HBCD , and lower brominated PBDEs in humans. Organohalogen Compounds, 66, 3820–3825.; b) Thuresson, K., Höglund, P., Hagmar, L., Sjödin, A., Bergman, Å.˚, & Jakobsson, K. (2006). Apparent half-lives of hepta- to decabrominated diphenyl ethers in human serum as determined in occupationally exposed workers. Environmental Health Perspectives, 114(2), 176–181.; c) Abdallah, M. A.-E., Tilston, E., Harrad, S., & Collins, C. (2012). In vitro assessment of the bioaccessibility of brominated flame retardants in indoor dust using a colon extended model of the human gastrointestinal tract. Journal of Environmental Monitoring, 14, 3276–3283.; d) Lorber, M. (2008). Exposure of Americans to polybrominated diphenyl ethers. Journal of Exposure Science and Environmental Epidemiology, 18(1), 2–19.

To convert daily adult intakes of FRs via different exposure pathways to expected body burdens, the dust and diet absorption fractions and human half-lives for PBDEs and HBCDDs (Abdallah and Harrad, 2011; Abdallah et al., 2012; Geyer et al., 2004; Lorber, 2008; Thuresson et al., 2006) were used in equation (6.4) (Table 6.6) while the inhalable fraction was assumed to be 100% bioavailable. The body lipid mass was estimated based on a 25% body fat for an average adult weighing 70 kg (U.S. EPA, 1997). Finally, $K_{FR}$ was calculated as $0.693/t_{0.5}$; where $t_{0.5}$ is the half-life of the studied FRs in the body lipid compartment (Table 6.6).

To the best of our knowledge, no information is available for the absorption fractions and half-lives of EFRs in humans. Based on the number of bromines and molecular weight, the absorption fractions and human half-lives for individual EFRs were assumed equivalent to literature reported absorption fractions and human half-lives for PBDEs (Table 6.6). To illustrate, the absorption fractions and human half-lives for DBE-DBCH (number of bromine: 4; molecular weight: 427.8) were assumed equivalent to that for BDE 47 (number of bromine: 4; molecular weight: 485.79).

Generally, predicted body burdens appear reasonably close to measurements for PBDEs in this study. In one previous study, good agreement was also observed between the predicted body burdens through diet, air and dust and the observed levels of main target PBDEs in UK human milk (Abdallah and Harrad, 2014). Even though the levels of PBDEs in human milk in this study (Table 6.2) were comparable to those in the previous study of Abdallah and Harrad (2014), the values of PBDEs in air, dust and diet used as input data in this study differed from those used in the previous study. For example, concentrations of BDE 209 in dust were lower than in the previous study (Abdallah and Harrad, 2014) while the dietary intake of PBDEs was lower than the UK Food Standards Agency (2006) data used in Abdallah and Harrad's study (2014).

However, the similar good agreement between the predicted and the observed body burdens of target PBDEs were observed. This is consistent with the hypothesis that the main exposure route of PBDEs is gradually shifting from indoor air and dust to our diet following declining use of PBDEs (Harrad and Diamond, 2006). In this study, dietary intakes were the major exposure pathway contributing to PBDE body burdens (56%-85% for tri-hexa BDEs) in the UK population except for BDE 209 - for which dust ingestion accounted for ~90% of overall body burden.

**Table 6.7 Comparison of predicted adult body burdens arising from average and median daily exposures [a] to major target PBDEs with observed levels in human milk samples**

| | **BDE 28** | **BDE 47** | **BDE 100** | **BDE 99** | **BDE 154** | **BDE 153** | **BDE 209** |
|---|---|---|---|---|---|---|---|
| **Average intake (ng/day) [b]** | | | | | | | |
| Dust | 0.21 | 3.40 | 2.80 | 5.00 | 1.70 | 3.40 | 410 |
| Diet | 2.50 | 45.54 | 11.40 | 54.98 | 7.07 | 5.11 | 6.20 |
| Air | 0.40 | 2.20 | 0.51 | 1.50 | 0.09 | 0.16 | 4.20 |
| **Median intake (ng/day) [b]** | | | | | | | |
| Dust | 0.01 | 0.50 | 0.11 | 0.82 | 0.05 | 0.49 | 100 |
| Diet | 2.52 | 35.99 | 8.27 | 37.68 | 6.34 | 3.94 | 6.28 |
| Air | 0.04 | 0.31 | 0.03 | 0.19 | 0.01 | 0.02 | 1.40 |
| **Average predicted body burdens (ng/g lw)** | | | | | | | |
| Dust | 0.03 | 0.18 | 0.13 | 0.18 | 0.09 | 0.58 | 0.07 |
| Diet | 0.41 | 2.39 | 0.53 | 1.93 | 0.39 | 0.87 | 0.001 |
| Air | 0.07 | 0.20 | 0.04 | 0.13 | 0.02 | 0.06 | 0.01 |
| Sum | 0.51 | 2.76 | 0.71 | 2.24 | 0.50 | 1.50 | 0.08 |
| **Median predicted body burdens (ng/g lw)** | | | | | | | |
| Dust | 0.002 | 0.03 | 0.01 | 0.03 | 0.003 | 0.08 | 0.02 |
| Diet | 0.41 | 1.89 | 0.39 | 1.32 | 0.35 | 0.67 | 0.001 |
| Air | 0.01 | 0.03 | 0.002 | 0.02 | 0.001 | 0.01 | 0.002 |
| Sum | 0.42 | 1.94 | 0.39 | 1.37 | 0.36 | 0.76 | 0.02 |
| **Observed body burdens (ng/g lw)** | | | | | | | |
| Average | 0.19 | 2.8 | 0.73 | 1.0 | 0.13 | 1.7 | <0.22 |
| Median | 0.14 | 2.2 | 0.53 | 0.77 | 0.12 | 1.8 | <0.22 |

a) Values below LOQ were assumed to be 1/2 LOQ; b) Based on average adult dust ingestion rate of 20 mg/day (Jones-Otazo et al., 2005) and average inhalation rate of 20 m3/day (Currado and Harrad, 1998) and average adult weight of 70 kg.

**Table 6.8 Comparison of predicted adult body burdens arising from average and median daily exposures [a] to major target HBCDDs with observed levels in human milk in this study**

| | **α-HBCDD** | **β-HBCDD** | **γ-HBCDD** |
|---|---|---|---|
| **Average intake (ng/day)** | | | |
| Dust | 41.00 | 19.00 | 80.00 |
| Diet [c] | 16.77 | 5.40 | 7.43 |
| Diet [d] | 203.44 | 105.43 | 112.24 |
| Air | 0.79 | 0.30 | 4.80 |
| **Median intake (ng/day)** | | | |
| Dust | 9.20 | 2.70 | 3.00 |
| Diet [c] | 18.18 | 5.41 | 8.51 |
| Diet [d] | 203.44 | 105.43 | 112.24 |
| Air | 0.03 | 0.02 | 2.00 |
| **Average predicted body burdens (ng/g lw)** | | | |
| Dust | 0.51 | 0.07 | 0.26 |
| Diet [c] | 0.21 | 0.02 | 0.02 |
| Diet [d] | 2.55 | 0.38 | 0.37 |
| Air | 0.01 | 0.001 | 0.02 |
| Sum [c] | 0.73 | 0.09 | 0.31 |
| Sum [d] | 3.07 | 0.45 | 0.65 |
| **Median predicted body burdens (ng/g lw)** | | | |
| Dust | 0.12 | 0.01 | 0.01 |
| Diet [c] | 0.34 | 0.03 | 0.05 |
| Diet [d] | 2.55 | 0.38 | 0.37 |
| Air | 0.0004 | 0.0001 | 0.01 |
| Sum [c] | 0.34 | 0.03 | 0.05 |
| Sum [d] | 2.66 | 0.39 | 0.39 |
| **Observed body burdens (ng/g lw)** | | | |
| Average | 2.10 | 0.25 | 0.90 |
| Median | 1.90 | 0.23 | 0.73 |

a) Values below LOQ were assumed to be 1/2 LOQ; b) Based on average adult dust ingestion rate of 20 mg/day (Jones-Otazo et al., 2005) and average inhalation rate of 20 m3/day (Currado and Harrad, 1998) and average adult weight of 70 kg; c) Values based on food samples collected in 2015 (chapter 5); d) Values based on food samples in a previous study (UK Food Standards Agency, 2006).

For HBCDDs, the predicted body burdens were lower than the observed levels of individual HBCDDs in UK human milk when using the estimated dietary intake values of HBCDDs from this study. This may be attributable to our focus on meat-related food samples in our study while HBCDDs showed highest levels in vegetable, fruit and milk in previous UK studies compared with meat and fish (Driffield et al., 2008; UK Food Standards Agency, 2006). As the levels of ∑HBCDDs in meat-related food samples in this study were comparable to this previous study (UK Food Standards Agency, 2006), we also used the dietary intake estimated values of HBCDDs in the previous study to predict HBCDDs body burden. This resulted in closer agreement between the predicted and observed body burdens of individual HBCDDs. This indicates that it is important to collect vegetables, fruits, milk and high water content food samples when investigating the dietary intake estimation of HBCDDs.

To our knowledge, this is the first attempt to predict the body burden of EFRs in human milk. In general, the predicted adult body burdens were in agreement with the observed levels in human milk. As these predicted body burdens were based on assumed half-lives and absorption efficiencies of EFRs extrapolated from known values for PBDEs, the good agreement between predicted and observed body burdens indicates our target EFRs possess similar physicochemical properties to PBDEs. However, more study is critical to obtain empirical values of the bioaccessibility via various exposure routes and human half-lives for EFRs.

In this study, dietary exposure was shown to be the main contributor to UK adult body burdens of DBE-DBCH and EH-TBB (64%-73%), while dust ingestion plays a more important role in driving body burdens of BTBPE, BEH-TEBP, and DBDPE (accounting for 61%-83% of the whole body burden) in UK adults.

In conclusion, good agreement was achieved between predicted and observed body burdens for our target PBDEs, HBCDDs and EFRs given the simplicity of the used PK model, the scarcity of information about crucial parameters like the half-lives of target compounds in human tissues, and uncertainties about the bioaccessibility of target chemicals. Additionally, we have not considered exposure via pathways including dermal contact with dust and flame-retarded materials which may make important contributions.

**Table 6.9 Comparison of predicted adult body burdens arising from average and median daily exposures [a] to major target EFRs with observed levels in human milk samples**

| | **α-DBE-DBCH** | **β-DBE-DBCH** | **EH-TBB** | **BTBPE** | **BEH-TEBP** | **DBDPE** |
|---|---|---|---|---|---|---|
| **Average intake (ng/day)** | | | | | | |
| Dust | 0.26 | 0.30 | 0.97 | 21.00 | 14.00 | 20.00 |
| Diet | 8.20 | 49.84 | 3.34 | 5.79 | 6.04 | 15.71 |
| Air | 2.30 | 1.70 | 0.17 | 0.31 | 0.17 | 0.44 |
| **Median intake (ng/day)** | | | | | | |
| Dust | 0.16 | 0.18 | 0.19 | 1.50 | 2.60 | 6.50 |
| Diet | 7.72 | 49.59 | 3.48 | 4.19 | 6.75 | 9.53 |
| Air | 1.70 | 1.20 | 0.05 | 0.13 | 0.04 | 0.10 |
| **Average predicted body burdens (ng/g lw)** | | | | | | |
| Dust | 0.01 | 0.02 | 0.04 | 0.15 | 0.10 | 0.003 |
| Diet | 0.43 | 2.61 | 0.15 | 0.04 | 0.04 | 0.003 |
| Air | 0.21 | 0.15 | 0.02 | 0.002 | 0.001 | 0.001 |
| Sum | 0.65 | 2.78 | 0.21 | 0.19 | 0.14 | 0.01 |
| **Median predicted body burdens (ng/g lw)** | | | | | | |
| Dust | 0.01 | 0.01 | 0.01 | 0.01 | 0.02 | 0.001 |
| Diet | 0.40 | 2.60 | 0.16 | 0.03 | 0.05 | 0.002 |
| Air | 0.15 | 0.11 | 0.005 | 0.001 | 0.0003 | 0.0001 |
| Sum | 0.57 | 2.72 | 0.17 | 0.04 | 0.07 | 0.003 |
| **Observed body burdens (ng/g lw)** | | | | | | |
| Average | 0.67 | 2.50 | 0.21 | 0.15 | 0.25 | -- |
| Median | 0.60 | 1.20 | 0.16 | <0.1 | <0.1 | <0.78 |

a) Values below LOQ were assumed to be 1/2 LOQ; b) Based on average adult dust ingestion rate of 20 mg/day (Jones-Otazo et al., 2005) and average inhalation rate of 20 m3/day (Currado and Harrad, 1998) and average adult weight of 70 kg; c) The average value were not calculated as the DF < 50%.

## 6.5 Conclusion

This chapter provides the first data on the levels of EFRs in UK human milk. The most frequently detected compounds were α-DBE-DBCH, β-DBE-DBCH and EH-TBB. We also examined the relationship between our estimated intakes via different pathways and the body burdens using a simple one-compartment PK model. The results of this showed predicted adult body burdens to be in agreement with observed levels in human milk for all studied FRs. In summary, dust ingestion appears to constitute the major exposure pathway for adults to BDE 209, BTBPE, BEH-TEBP, and DBDPE, while dietary exposure was the major exposure pathway contributing to body burdens of HBCDDs, tri-hexa BDEs, DBE-DBCH and EH-TBB.

# CHAPTER 7 Summary and Conclusions

The main achievements of this thesis relative to its objectives are summarised below:

## 7.1 The optimisation and validation of analytical methodology

- Different extraction and clean-up methods were compared and applied successfully to a variety of samples including air, dust, diet and human milk.
- Analytical methods based on GC-NCI-MS were optimised and validated for the separation and analysis of emerging and legacy FRs, as well as DBE-DBCH and DDC-CO isomers.

## 7.2 Food, an e-waste processing area in Vietnam

- A number of EFRs were found in chicken, fish, and pork samples from an e-waste processing site in Vietnam, as well as high levels of HBCDDs. The most abundant EFR detected was DDC-CO.
- Some enantioselective enrichment of both α- and γ-HBCDD was observed in chicken samples.
- For most of our target compounds, the main contributors to dietary exposure of both adults and children were chicken liver and chicken eggs. Estimated daily dietary intakes of HBCDDs were higher than those reported from other countries.
- This study provides evidence that EFRs are already entering the waste stream leading to environmental contamination when such waste is treated in an unregulated fashion. The

elevated concentrations of DDC-CO are of particular note. We hypothesise that over time, environmental contamination with EFRs will rise as increasing numbers of products containing these chemicals reach the end of their useful life.

## 7.3 Dust and air, UK

- In both homes and offices, the more volatile EFRs included in this study such as DBE-DBCH and PBBz were detected frequently in indoor air. In contrast to indoor air in this study, the less volatile compounds were more frequently detected in office and house dust. These include: DDC-CO, TBBPA-BDBPE and DBDPE.
- The most abundant compounds in air were $\alpha$-DBE-DBCH and $\beta$-DBE-DBCH in both homes and offices. However, TBBPA-BDBPE was the most abundant compound in home and office dust, followed by BEH-TEBP, BTBPE, and DBDPE.
- The $f_{\beta\text{-DBE-DBCH}}$ values fell between 0.32-2.88 (median value: 0.85) in indoor dust which is lower than observed in the commercial product (1.0) but significantly higher than we detected in indoor air (0.53-1.0; median =0.73; $p<0.01$).
- PBDEs were the predominant FR class monitored to be detected in homes, contributing 44 % of ∑FRs. In contrast, EFRs were the principal contributor to ∑FRs in offices, accounting for 83% of ∑FRs. Similarly, PBDEs were the predominant FR class in home dust, contributing 66 % ∑FRs, while EFRs dominated in in office dust, accounting for 51% of ∑FRs.
- Concentrations of some PBDEs are lower and those of some EFRs higher than in previous UK studies, with such contrasting temporal trends suggesting replacement of PBDEs by EFRs.

## 7.4 Food, UK

- This is the first investigation of concentrations of EFRs in UK food samples. α-DBE-DBCH, β-DBE-DBCH, EH-TBB, BTBPE, BEH-TEBP and DBDPE were detected in UK food samples. β-DBE-DBCH was the most frequently detected and the predominant compound in food samples.
- The $f_{\beta\text{-DBE-DBCH}}$ values (1.20 to 220) in our food samples exceed those detected in UK indoor dust (0.32 to 2.88) ($p < 0.01$), indoor air (0.53 to 1.0) ($p < 0.01$) and the commercial product (1.0), suggesting diastereomer-specific environmental degradation/metabolism and/or isomerisation in the food chain.
- The median value of $f_{\beta\text{-DBE-DBCH}}$ in liver (80) in this study was substantially higher than those found in meat (5.9), fish (6.1, tuna not included), eggs (7.1), and cheese (2.1) suggesting that the uptake and metabolism of DBE-DBCH isomers is species- and organism-dependent.
- The levels of ∑PBDEs in this study are higher than those recorded in previous studies in the UK conducted in 2003-2004 and 2006 (UK Food Standards Agency, 2006; UK Food and Environment Research Agency, 2009) suggesting a gradual shift over time of PBDEs from the indoor to the outdoor environment and thus our food supply.

## 7.5 Human milk, UK

- Concentrations of our target EFRs in archived human milk samples (collected in 2010) (human milk group 1) and human milk samples collected from 2014-2015 (human milk group 2) were investigated for the first time. The DFs of all EFRs in human milk group 1 were lower than those in human milk group 2 even though the sample size of the latter is smaller than the former, indicating the more widespread usage of these EFRs currently than hitherto.

- β-DBE-DBCH showed the highest DFs and concentrations in both human milk groups which is similar to the findings in UK diet samples indicating the high bioaccumulative potential in human milk. To our knowledge, this is the first report of β-DBE-DBCH in human milk samples.
- The values of $f_{\beta\text{-DBE-DBCH}}$ ranged from 0.88 to 608, with a median of 9.6 which is significantly higher than observed in UK indoor air (0.53-1.0) ($p < 0.001$) and dust (0.32-2.88) ($p < 0.001$), indicating diastereomer-specific environmental degradation/metabolism and/or isomerisation in humans. Of note, $f_{\beta\text{-DBE-DBCH}}$ values in human milk were statistically indistinguishable to those in diet samples ($p > 0.05$) indicating the relatively higher abundance of β-DBE-DBCH compared with α-DBE-DBCH may also result from dietary exposure to DBE-DBCH.
- PBDEs are the predominant FR class in human milk, contributing 50% ∑FRs, followed by EFRs.

## 7.6 Human exposure, UK

- The estimated mean dietary intakes of ∑EFRs in the UK were 89 and 26 ng/day for adults and toddlers, respectively, which are substantially higher than those received via inhalation (4.3 and 0.66 ng/day for adults and toddlers, respectively) and comparable to those intake via dust ingestion (27 and 38 ng/day for adults and toddlers, respectively).
- In this study, dust ingestion appears to constitute the major exposure pathway for adults to BDE-209, BTBPE, BEH-TEBP, and DBDPE, while dietary exposure was the major exposure pathway contributing to body burdens of HBCDDs, tri-hexa BDEs, DBE-

DBCH and EH-TBB.

## 7.7 Comparison of FRs intake to human body burdens

- Predicted body burdens appear reasonably close to measurements for PBDEs in this study. This is consistent with the hypothesis that the main exposure route of PBDEs is gradually shifting from indoor air and dust to our diet following declining use of PBDEs (Harrad and Diamond, 2006). In this study, dietary intakes were the major exposure pathway contributing to PBDE body burdens (56%-85% for tri-hexa BDEs) in the UK population except for BDE-209 - for which dust ingestion accounted for ~90% of overall body burden.
- For HBCDDs, observed levels of individual HBCDDs in UK human milk exceeded the predicted body burdens obtained when using the estimated dietary intake values of HBCDDs from this study. This may be attributable to our focus on meat-related food samples in our study while HBCDDs showed highest levels in vegetable, fruit and milk in previous UK studies compared with meat and fish (Driffield et al., 2008; UK Food Standards Agency, 2006).
- This is the first attempt to predict the body burden of EFRs in human milk. In general, the predicted adult body burdens were in agreement with the observed levels in human milk. As these predicted body burdens were based on assumed half-lives and absorption efficiencies of EFRs extrapolated from known values for PBDEs, the good agreement between predicted and observed body burdens indicates our target EFRs possess similar physicochemical properties to PBDEs. However, more study is critical to obtain empirical values of the bioaccessibility via various exposure routes and human half-lives for EFRs.

## 7.8 Research gaps and future perspectives

Following the phase-out of PBDEs and HBCDDs due to health concerns, there is increasing demands for EFRs, some of which potentially show PBT characteristics and may pose health risks to human and wildlife, especially those produced and used in high volumes. Therefore, further investigations are needed to:

- Enhance communication with EFR producers and end-users in order to identify EFRs likely to be present in the environment.
- Gather more data on concentrations of EFRs, PBDEs and HBCDDs in samples relevant to human exposure including indoor air, dust and diet to investigate the relative importance of various exposure pathways in future and further examine the impacts of recent controls on the use and production of EFRs and "legacy" FRs. More data are also required to identify the levels of EFRs in outdoor air.
- Further understanding of the transfer mechanisms of EFRs from commercial products (e.g. foam chair, sofa, TV, computer, printer and fridge) to indoor air and dust as well as the causes of variability (e.g. temperature, ventilation rates, room size and the number of appliance) in EFR levels in indoor environments such as office and home.
- Investigate the human bioaccessibility of EFRs via different human exposure pathways like dust ingestion, dermal contact and air inhalation as well as pharmacokinetic data (e.g. half-lives) of those compounds to better predict the body burdens of those compounds in different human tissues.
- Improve knowledge of the potential adverse health effects/toxicity of EFRs to facilitate risk assessment of EFRs for human beings and enable the determination of a tolerable daily intake for EFRs.

- Better understanding the degradation pathways and rates of EFRs in environment and biota and the factors such as temperature, exposure concentrations and chemical structure affecting the generation of metabolites and degradation rates.
- Elucidate the relationship between the intake of EFRs through different human exposure pathways for different age groups and the resultant body burdens to assess the relative significance of each exposure route. More biomonitoring studies based on human milk and serum are desirable as well as studies based on less invasive media such as hair, saliva, and faeces specifically for infants and children.

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# Appendix: List of Publications

1. **Tao, F.**, Matsukami, H., Suzuki, G., Tue, N.M., Viet, P.H., Takigami, H. and Harrad, S.J., 2016. Emerging halogenated flame retardants and hexabromocyclododecanes in food samples from an e-waste processing area in Vietnam. *Environmental Science: Processes & Impacts,* 18, pp.361-370.
2. Van den Eede, N., Tomy, G., **Tao, F.**, Halldorson, T., Harrad, S., Neels, H. and Covaci, A., 2016. Kinetics of tris (1-chloro-2-propyl) phosphate (TCIPP) metabolism in human liver microsomes and serum. Chemosphere, 144, pp.1299-1305.
3. Labunska, I., Abdallah, M.A.E., Eulaers, I., Covaci, A., **Tao, F.**, Wang, M., Santillo, D., Johnston, P. and Harrad, S., 2015. Human dietary intake of organohalogen contaminants at e-waste recycling sites in Eastern China. Environment international, 74, pp.209-220.
4. Liu, W., Li, H., **Tao, F.**, Li, S., Tian, Z. and Xie, H., 2013. Formation and contamination of PCDD/Fs, PCBs, PeCBz, HxCBz and polychlorophenols in the production of 2, 4-D products. Chemosphere, 92(3), pp.304-308.
5. Liu, W., **Tao, F.**, Zhang, W., Li, S. and Zheng, M., 2012. Contamination and emission factors of PCDD/Fs, unintentional PCBs, HxCBz, PeCBz and polychlorophenols in chloranil in China. Chemosphere, 86(3), pp.248-251.