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1 **Polybrominated diphenyl ethers in UK human milk; Implications for**
2 **infantile exposure and relationship to external exposure**

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23

24 **Abstract**

25 Fourteen tri-deca polybrominated diphenyl ethers (PBDEs) were investigated in 35 human
26 milk samples from Birmingham, UK. While none of the hepta-nona BDEs (the main
27 components of the OctaBDE technical mixture) were above the limit of quantitation (LOQ);
28 BDE-47 (average concentration = 3.3 ng g⁻¹ lipid weight (lw)) was quantified in all samples
29 contributing 34-74% to Σtri-hexa BDEs (the principal constituents of the PentaBDE
30 commercial formulation). BDE-209 (the main congener in the DecaBDE formulation) was
31 present above the LOQ in 69% of samples (average concentration = 0.31 ng g⁻¹ lw).
32 Concentrations of Σtri-hexa BDEs ranged from 0.2-26 ng g⁻¹ lw with concentrations of BDE-
33 47 > BDE-153 > BDE-99. While concentrations of Σtri-hexa BDEs in this study (average =
34 5.95 ng g⁻¹ lw) were at the high end of those reported from other European countries,
35 concentrations of BDE-209 were lower than those reported in human milk from other
36 countries. The average exposure of a UK nursing infant to Σtri-hexa BDEs (35 ng (kg bw)⁻¹
37 day⁻¹) via breast milk exceeded the upper-bound dietary intakes of both UK adults and
38 toddlers. Using a simple one compartment pharmacokinetic model, PBDE intakes of UK
39 adults via inhalation, diet and dust ingestion were converted to predicted body burdens.
40 Predictions compared well with those observed for Σtri-hexa BDEs and BDE-209 in breast
41 milk.

42 **Keywords:** PBDEs, human milk, infant exposure, BDE 209.

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50 **Introduction**

51 Polybrominated diphenyl ethers (PBDEs) have been extensively used as flame retardants for
52 a wide range of consumer products including furniture, carpets, mattresses and casings for
53 electronic equipment (BSEF 2013). Three technical PBDE formulations were commercially
54 available: Penta (consisting primarily of BDE-47 and BDE-99 – 38-49% each, alongside
55 smaller amounts of other tri- to hepta-BDEs), Octa (a mixture of hexa- to deca-BDEs – the
56 exact congener composition varying substantially between the two principal formulations
57 marketed) and Deca (92-97% decabromodiphenyl ether – BDE 209 – plus nona- (principally)
58 and octa-BDEs) (La Guardia, et al. 2006). DecaBDE has dominated worldwide production
59 with a global market demand of 56,100 tons in 2001, compared to 7,500 and 3,790 tons for
60 PentaBDE and OctaBDE formulations respectively (BSEF 2013). Despite their utility, the
61 persistence and bioaccumulative characters of these compounds have resulted in increasing
62 concern over their potential adverse effects to human health (Frederiksen, et al. 2009; Harrad,
63 et al. 2010). Animal studies have shown PBDEs to pose potential health risks including:
64 endocrine disruption, neurodevelopmental and behavioural outcomes, hepatic abnormality
65 and possibly cancer (Birnbaum and Staskal 2004; Darnerud 2008; Hakk 2010; Wikoff and
66 Birnbaum 2011). The few data available from human epidemiological studies imply effects
67 on: male reproductive hormones (Johnson, et al. 2013; Palace, et al. 2010), semen quality
68 (Akutsu, et al. 2008), thyroid hormone homeostasis (Turyk, et al. 2008), cryptorchidism
69 (Crump, et al. 2010), behavioral factors in pregnant women (Buttke, et al. 2013), as well as
70 lower birth weight and length (Chao, et al. 2007; Lignell, et al. 2013). Such evidence has
71 contributed to complete EU bans for Penta and OctaBDE, and restrictions on the use of
72 DecaBDE in addition to other restrictions within several jurisdictions on the manufacture and
73 new use of the three commercial PBDE formulations across the world (Harrad, et al. 2010).
74 Moreover, PBDEs associated with Penta and OctaBDE have been listed under the UNEP

75 Stockholm Convention on POPs, while DecaBDE is currently under consideration for listing
76 under Annexes A, B and/or C to the convention (Stockholm convention on POPs 2009).
77 Despite such restrictions, human exposure to PBDEs is likely to continue for the foreseeable
78 future, given their persistence and ubiquity of flame-retarded consumer materials (Harrad and
79 Diamond 2006).

80 Several studies have reported different levels of PBDEs in various human tissues including
81 serum, placenta, liver, adipose tissue and breast milk from different European, Asian and
82 North American countries in the last few years (Cui, et al. 2012; Frederiksen, et al. 2009).
83 These biomonitoring data provide a direct measurement of the human body burden of BFRs
84 resulting from various external exposure pathways (e.g. inhalation, ingestion of dust, diet and
85 water) and contribute to the risk assessment of such compounds. However, the only available
86 information on BFRs in UK human samples is for tri- to hexa-BDEs (major components of
87 the PentaBDE commercial product) where the median concentrations for Σ tri-to hexa-BDEs
88 in human milk and serum samples collected in 2003 were 6.3 and 4.18 ng g⁻¹ lipid weight
89 (lw) respectively (Kalantzi, et al. 2004). In addition, BDE-209 was detected in 11 out of 153
90 serum samples at concentrations from 0.015-0.240 ng g⁻¹ lw) (Thomas, et al. 2006).

91 Current understanding is that non-occupational human exposure to PBDEs occurs mainly via
92 a combination of diet, air and indoor dust (either via ingestion or dermal contact)
93 (Frederiksen, et al. 2009; Lorber 2008; Trudel, et al. 2011). However, the extent to which the
94 known contamination of indoor environments with PBDEs influences human body burdens
95 remains unclear. While some studies have managed to establish significant positive
96 correlations between the levels of PBDEs in food or indoor dust and their concentrations in
97 human milk or serum (Dunn, et al. 2010; Thomsen, et al. 2008; Wu, et al. 2007); such
98 correlations could not be established in other studies (Roosens, et al. 2009; Wang, et al.
99 2013). An alternative approach involved application of a simple pharmacokinetic model to

100 predict the body burdens of PBDEs in American adults using intake data from different
101 exposure pathways. The predicted body burdens were then compared to the reported levels of
102 PBDEs in human matrices and the relationship between external and internal exposure of
103 American adults to PBDEs was discussed (Lorber 2008).

104 To address this paucity of UK human biomonitoring data for PBDEs, this study reports
105 concentrations of Σ tri-hexa BDEs and *for the first time* BDE-209 in 35 human milk samples
106 from Birmingham, UK. These data are then used to estimate the dietary exposure of UK
107 nursing infants under different exposure scenarios. Finally, a simple, one-compartment
108 pharmacokinetic model is applied to predict the body burdens of the studied PBDEs in UK
109 adults (using indoor air and dust levels reported elsewhere by our research group for
110 Birmingham, UK (Abdallah and Harrad 2010; Harrad and Abdallah 2011; Harrad, et al.
111 2006; Harrad, et al. 2008a). The model predictions are then compared to the concentrations of
112 target compounds measured in the analyzed human milk samples (used as indicator of adult
113 female body burdens) for further understanding of the relationship between external and
114 internal human exposure to PBDEs in UK adults.

115 **Materials and Methods**

116 *Sample collection*

117 Breast milk samples (each comprising ~50 mL) were obtained from 35 adult healthy
118 primiparous volunteers via Birmingham Women's Hospital Milk Bank after the study
119 protocol was approved by Warwickshire Research Ethics Committee and the R&D
120 Department in Birmingham Women's NHS foundation trust. Informed consent was obtained
121 from all the participants before sample collection. Samples collected in 2010 were kept in
122 clean screw-capped glass containers and transferred from the Milk Bank to the laboratory in
123 special ice boxes then stored at -20°C until the time of analysis. Due to ethical regulations,

124 the samples were collected in a completely anonymous fashion with all participant
125 information kept strictly confidential. For the purpose of this study, only 1 milk sample was
126 collected from each mother during her first 6 month of lactation.

127 *Sample extraction*

128 Accurately weighted aliquots of the freeze-dried samples (~ 2 g) were loaded into pre-
129 cleaned 66 mL Accelerated Solvent Extraction (ASE 300, Dionex Inc., UK) cells containing
130 1.5 g florisil, 3 g alumina, 5 g anhydrous Na₂SO₄ and hydromatrix (Varian Inc., UK) to fill
131 the void volume of the cells, spiked with 25 ng of each of ¹³C-labelled BDE-47, BDE-99,
132 BDE-153, BDE-183, BDE-209 as internal (surrogate) standards. The ASE cells were
133 extracted with hexane:dichloromethane (1:9, v/v) at 90 °C and 1500 psi. The heating time was
134 5 minutes, static time 4 min, purge time 90 s, flush volume 50%, with three static cycles. The
135 lipid weight of the studied samples was determined gravimetrically on separate aliquots using
136 a standard procedure (The European Standard EN 1528-2, 1996; see supplementary data for
137 more details).

138

139 *Sample Clean-up*

140 The crude extracts were concentrated to 0.5 mL using a Zymark Turbovap® II (Hopkinton,
141 MA, USA) then washed with 3 mL of 98% sulfuric acid. After phase separation, the hexane
142 layer was transferred onto a florisil column topped with sodium sulfate and eluted with 25
143 mL of hexane:dichloromethane (1:1, v/v). The eluate was evaporated to dryness under a
144 gentle stream of N₂ and the dried extract reconstituted in 200 µL of ¹³C-BDE-100 (25 pg µL⁻¹
145 in methanol) used as recovery determination (or syringe) standard to determine the recoveries
146 of internal standards for QA/QC purposes.

147

148 *LC-APPI-MS/MS analysis*

149 Sample analysis was carried out using an LC-MS/MS system composed of a dual pump
150 Shimadzu LC-20AB Prominence liquid chromatograph equipped with SIL-20A autosampler,
151 a DGU-20A3 vacuum degasser coupled to a Sciex API 2000 triple quadrupole mass
152 spectrometer. Details of the multi-residue analytical methodology used for separation and
153 quantification of the studied PBDEs can be found elsewhere (Abdallah, et al. 2009). (A brief
154 description is given in the supplementary data section).

155

156 *Comparison of PBDE intake to human body burdens.*

157 We have previously estimated UK adult intake of the target PBDEs via inhalation, dust
158 ingestion and diet (Harrad and Abdallah 2011; Harrad, et al. 2006; Harrad, et al. 2008a;
159 Harrad, et al. 2008b) (A summary of the assumptions on which these estimations are based is
160 provided as supplementary data). To examine the relationship between these estimated
161 intakes and the body burdens indicated via human milk samples, a simple one-compartment,
162 first order pharmacokinetic (PK) model was used. The studied PBDEs were hypothesized to
163 accumulate in lipids (the single compartment in the model). Therefore, the change in PBDE
164 lipid concentration over time can be expressed by equation 1 (Lorber 2008).

165
$$\frac{\delta C_{PBDE}}{\delta t} = \frac{I_{PBDE}(t) \times AF_{PBDE}}{BL(t) - K_{PBDE} \times C_{PBDE}(t)} \quad (1)$$

166 Where C_{PBDE} is the compound specific concentration in lipids ($\text{ng g}^{-1} \text{lw}$); I_{PBDE} is the daily
167 intake of the target BFR (ng day^{-1}); AF_{PBDE} is the absorption fraction (unitless); BL is body
168 lipid mass (g) and K_{PBDE} is the compound specific first order dissipation rate (day^{-1}).

169 If K_{PBDE} is assumed to be constant over time then equation 1 can be solved into:

$$170 \quad C_{PBDE}(t) = C_{PBDE}(0) \times e^{(-K_{PBDE} \cdot t)} + \left[\frac{(I_{PBDE}(t) \times AF_{PBDE})}{BL(t)} \right] \times \left[\frac{(1 - e^{(-K_{PBDE} \cdot t)})}{K_{PBDE}} \right] \quad (2)$$

171 Where $C_{PBDE}(0)$ is the studied PBDE body lipid concentration at time 0 (initial concentration
172 before intake).

173 Assuming a constant dose over time at constant body lipid mass, the steady state PBDE lipid
174 concentration can be calculated from equation 3. It is stressed that the assumption of steady
175 state conditions is an inherent uncertainty with this approach.

$$176 \quad C_{PBDE} = \frac{(I_{PBDE} \times AF_{PBDE})}{BL \times K_{PBDE}} \quad (3)$$

177 *Quality assurance/Quality control*

178 Good recoveries (68-106%) of the ^{13}C -labelled internal standards were obtained for all the
179 studied compounds (table SI-4). Further evaluation of the method extraction/clean up
180 performance was achieved via spiking milk samples (n=6) with ^{13}C -BDE-154 prior to freeze
181 drying and excellent recoveries (>90%) were obtained (table SI-5).

182 No target compounds were detected in method blanks (n=5; consisting of 2 g pre-extracted
183 anhydrous sodium sulfate treated exactly as a sample) or field blanks (n=5; consisting of ~2 g
184 of broken pieces of the glass milk containers treated exactly as a sample). Therefore, there
185 was no need for blank correction of concentrations and method limits of detection (LOD) and
186 quantification (LOQ) were estimated based on 3:1 and 10:1 S:N ratios respectively.

187 The accuracy and precision of the analytical method applied for PBDE determination was
188 assessed via replicate analysis (n=10) of NIST SRM 2585. The results obtained compared
189 favourably with the reported reference values (table SI-6a).

190 **Results and discussion**

191 *Concentrations of Σ tri-hexa BDEs in UK human milk*

192 While none of the investigated hepta- to nona-BDE congeners were above LOQ, BDE-47
193 was quantified in all the analysed samples contributing 34-74% to Σ tri-hexa BDEs (Table 1).
194 The predominant BDE congeners in the studied human milk were in the order BDE-47 >
195 BDE-153 > BDE-99. These 3 congeners constituted an average of 85% of the quantified Σ tri-
196 hexa BDEs in the studied samples. This is in agreement with previous reports of PBDEs in
197 human milk from various countries (Frederiksen, et al. 2009). Interestingly, a higher average
198 level of BDE-153 (1100 pg g⁻¹ lw) than that of BDE-99 (710 pg g⁻¹ lw) was observed (Table
199 1). While this differs from the relative contribution of these 2 PBDE congeners in the
200 commercial PentaBDE formulations (La Guardia, et al. 2006), several authors have reported
201 higher levels of BDE-153 than BDE-99 in human milk (Ben Hassine, et al. 2012; Dunn, et al.
202 2010; Frederiksen, et al. 2009). In addition, a recent study has reported BDE-153 as the
203 dominant congener in 5 human breast milk samples from California (Park, et al. 2011).
204 Furthermore, a study of PBDEs in human milk from the Faroe islands also reported
205 predominance of BDE-153 (Fangstrom, et al. 2005). However, such high levels of BDE-153
206 could not be associated with high consumption of seafood diet in the studied population,
207 indicating that dietary exposure was not the reason for the elevated BDE-153 concentrations
208 in breast milk. Therefore, we hypothesize that the relatively higher contribution of BDE-153
209 to Σ tri-hexa BDEs in human milk samples than expected from the PentaBDE technical
210 mixture may be attributed to 2 main factors:

211 First, the high bioaccumulation potential of BDE-153 in lipids (as evidenced by a half-life of
212 6.5 years compared to 1.8 and 2.9 years for BDE-47 and BDE-99 respectively (Geyer, et al.
213 2004)) which indicates that over time, BDE-153 will become the predominant congener in
214 the body.

215 Second, the possible production of BDE-153 as a result of BDE-209 metabolic stepwise
216 meta-meta debromination (Roberts, et al. 2011). This stepwise debromination was previously
217 observed in peregrine falcon eggs from California, where BDE-153 was the dominant
218 congener only in eggs with high levels of BDE-209 (Holden, et al. 2009). Interestingly, while
219 concentrations of BDE-153 in this study were significantly ($r = 0.443$; $p < 0.01$) correlated
220 with those of BDE-209, no other statistically significant ($p < 0.05$) correlation was observed
221 between BDE-209 levels and any of the PBDE congeners or Σ tri-hexa BDEs in the analyzed
222 samples. This further supports the hypothesis that metabolic degradation of BDE-209 yields
223 the highly bioaccumulative BDE-153 resulting in elevated concentrations of the latter in
224 human milk.

225 While the levels of Σ tri-hexa BDEs in this study (Table 1) are slightly lower than those
226 reported in UK human milk samples collected in 2003 ($n=54$, average = $6.3 \text{ ng g}^{-1} \text{ lw}$), these
227 concentrations are still at the high end of those reported from other European, Asian, African
228 and Australasian countries (Table 2). On the other hand, Σ tri-hexa BDEs in UK human milk
229 are substantially lower than those reported from USA and Canada (Table 2) which is in
230 agreement with the far more extensive production and use of the PentaBDE technical
231 formulation in North America than elsewhere (BSEF 2013).

232

233 *Concentrations of BDE-209 in UK human milk*

234 BDE-209 was above LOQ in 69% of the studied milk samples ranging from <0.06 - 0.92 ng g^{-1}
235 lw (Table 1). To the authors' knowledge, this paper is the first to report concentrations of

236 BDE-209 in UK human milk. Interestingly, these levels are at the lower end of BDE-209
237 concentrations reported in human milk from other European countries (Table 2) despite the
238 substantially higher levels of this BFR reported in UK indoor dust compared to the rest of
239 Europe (Harrad, et al. 2010) and the reported higher usage of BDE 209 in the UK than other
240 EU countries (EU Risk Assessment Report 2002). This may indicate that while indoor dust
241 ingestion is the major pathway of external human exposure to BDE-209 (Harrad, et al. 2008a;
242 Lorber 2008), the high levels of this compound in indoor dust do not significantly contribute
243 to human body burdens. Our research group have recently reported on the very low
244 bioaccessibility (~14%) of BDE-209 in indoor dust across the human gastrointestinal tract
245 (GIT) following oral ingestion (Abdallah, et al. 2012), consistent with animal studies
246 reporting low bioavailability (4-26%) of BDE-209 (Huwe and Smith 2007; Sandholm, et al.
247 2003). Such poor uptake of BDE-209 from the GIT, combined with its very short human
248 half-life ($t_{0.5} = 7$ days, (Geyer, et al. 2004) and its preferential partitioning to serum rather
249 than milk fat (Mannetje, et al. 2012) may result in the apparently low influence of BDE-209
250 concentrations in indoor dust on UK adult body burdens.

251

252 *Nursing infants' dietary intake of PBDEs via breast milk:*

253 Breast milk is a recognized medium for direct transfer of POPs to nursing infants. To
254 estimate the nursing infants' dietary intake of the studied BFRs via breast milk, equation 4
255 was used.

256
$$Di = \frac{C_{PBDE} \times F_{lipid}}{Bw} \dots\dots\dots(4)$$

257 Where Di is the estimated dietary intake ($\text{ng kg}^{-1} \text{bw day}^{-1}$); C_{PBDE} is the concentration of
258 target PBDE in milk ($\text{ng g}^{-1} \text{lw}$); F_{lipid} is the daily lipid intake via breast milk (g day^{-1}) and Bw
259 is the body weight (4.14 kg) (U.S. EPA 2002.). The infant's daily lipid intake via breast milk

260 (F_{lipid}) was calculated based using U.S. EPA guidelines (U.S. EPA 2002.) which suggest an
261 average intake of 702 mL milk per day for a 1 month old infant weighing 4.14 kg. The
262 median lipid content of the analyzed milk samples was 3.47 g lipid per 100 mL of breast milk
263 resulting in a daily lipid intake of 24.4 g lipid day⁻¹.

264 Table 3 shows the estimated dietary intake of target PBDEs via breast milk using different
265 exposure scenarios (in which exposure factors (e.g. dust ingestion rate) were held constant
266 but using different PBDE concentrations (e.g. 25th percentile) derived from our breast milk
267 data). While the estimated average UK infant exposure to Σ tri-hexa BDEs is much lower than
268 that in North America (Park, et al. 2011), a 1 month-old infant in the UK is still more
269 exposed to Σ tri-hexa BDEs than in several other European countries via breast milk
270 (Roosens, et al. 2010). Interestingly, the average exposure of a nursing infant to Σ tri-hexa
271 BDEs via breast milk exceeded upper-bound dietary intakes of UK adults and toddlers (UK
272 Food Standards Agency 2006) (Figure 1), while for BDE-209, dietary exposure was the most
273 significant exposure pathway for toddlers.

274 The low concentrations of BDE-209 in the studied milk samples resulted in much lower
275 exposure of UK nursing infants to this contaminant than the USEPA reference daily dose
276 (RfD) of 7 μ g kg bw⁻¹ day⁻¹. Similarly, our estimated UK infant daily intakes (Table 3) are
277 lower than the USEPA reference doses for BDE-47 (100 ng kg bw⁻¹ day⁻¹ for
278 neurodevelopmental toxicity) and Σ tri-hexa BDEs (2000 ng kg bw⁻¹ day⁻¹ for liver toxicity)
279 (U.S.EPA 2008). However, the median level of Σ tri-hexa BDEs in this study (4.98 ng kg⁻¹
280 lw) is slightly higher than that associated with congenital cryptorchidism (4.16 ng kg⁻¹ lw;
281 $p < 0.01$) in Danish-Finnish newborn boys (Crump, et al. 2010) and generally in line with
282 levels associated with irregular menstruation periods in a Taiwanese population (Chao, et al.
283 2010). While this does not provide solid evidence on the potential health effects associated
284 with the reported levels of PBDEs in human milk due to the lack of relevant studies in the

285 UK, our results certainly raise concerns about potential adverse effects resulting from
286 exposure of infants and mothers to PBDEs. Although breastfeeding mothers should be
287 encouraged and supported due to the well-documented beneficial effects of breast feeding,
288 scientific studies ought to characterize and measure the contaminants in breast milk so that
289 protective measures may be provided, if necessary, to avoid any potential harmful effects on
290 the mother or the newborn.

291

292 *Comparison of PBDEs intake to human body burdens*

293 To convert daily adult intakes of BFRs via different exposure pathways to expected body
294 burdens, the bioaccessible fractions of each target compound (Abdallah, et al. 2012) were
295 used in equation 3 to substitute for AF_{PBDE} in case of exposure via dust ingestion or diet,
296 while the inhalable fraction was assumed to be 100% bioavailable. The body lipid mass was
297 estimated based on a 25% body fat for an average adult weighing 70 kg (U.S. EPA 1997).
298 Finally, K_{PBDE} was calculated as $0.693/t_{0.5}$; where $t_{0.5}$ is the half-life of the studied BFR in the
299 body lipid compartment (Geyer, et al. 2004).

300 In general, good agreement was observed between the predicted and the observed body
301 burdens of main target PBDEs (table 4) given the simplicity of the model used (e.g. only one
302 body compartment was studied), the dearth of information regarding the half-lives of
303 different PBDE congeners in various compartments of the human body, and the uncertainty
304 about the bioavailability of the studied compounds from different exposure routes.

305 In addition, the PK model used here does not estimate human exposure via routes such as
306 dermal contact and water intake. This is due to the high uncertainty and complete absence of
307 experimental data on the extent of BFR absorption via dermal contact by humans coupled
308 with the expected minimal contribution of water intake to the overall daily exposure to BFRs
309 based on the very low aqueous solubility of PBDEs.

310 Nevertheless, the good agreement between the predicted and observed results indicates that
311 the studied exposure routes are the main pathways driving UK adult body burdens of PBDEs.
312 This is in line with the findings of Lorber (Lorber 2008) who studied the exposure of
313 Americans to PBDEs and reported indoor dust ingestion as the main route of exposure
314 followed by diet and inhalation. However, more research is required for assessment of the
315 bioavailability of various PBDEs via different exposure routes and determination of $t_{0.5}$ of
316 PBDEs in various human tissues.

317

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321 manager at Birmingham Women's Hospital) for helping with the ethical issues for this
322 project.

323

324 **Supplementary data**

325 Specific details of analytical methodology, exposure estimation, QA/QC measurements and
326 concentrations of target BFRs in each sample are available as supplementary data.

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539 **Tables**

540 **Table1: Statistical summary of PBDE concentrations (ng g⁻¹ lw) in human milk samples**
541 **(n=35) from Birmingham, UK.**

	BDE-47	BDE-49	BDE-85	BDE-99	BDE-100	BDE-153	BDE-154	∑tri-hexa	BDE-209
Average	3.30	<0.05	0.08	0.71	0.45	1.10	0.30	5.95	0.31
SD*	3.25	0.08	0.15	0.67	0.39	1.05	0.30	5.35	0.30
Median	2.80	<0.05	<0.05	0.69	0.38	0.91	0.21	5.00	0.25
DF** (%)	100	20	46	94	89	97	77	100	69
LOQ	0.043	0.045	0.051	0.055	0.053	0.058	0.059	N/A [#]	0.062
Minimum	0.17	<0.05	<0.05	<0.06	<0.05	<0.06	<0.06	0.2	<0.06
25th %ile	0.78	<0.05	<0.05	0.20	0.12	0.35	0.07	1.70	<0.06
75th %ile	5.15	<0.05	0.09	0.85	0.70	1.43	0.55	9.55	0.58
Maximum	14.65	0.45	0.83	3.43	1.86	4.57	11.10	26.10	0.92

542 * Standard deviation.

543 ** Detection frequency.

544 [#] Not applicable.

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552 **Table 2: Average concentrations of PBDEs (ng g⁻¹ lw) in human milk samples from**
 553 **different countries.**

Location	year	number	∑tri-hexa BDEs	BDE- 209	Reference
UK	2009-10	35	5.9	0.3	(this study)
UK	2001-03	54	6.3	N/A*	(Kalantzi, et al. 2004)
Norway	2003-09	393	2.7	0.6	(Thomsen, et al. 2010)
Sweden	1996-2006	276	3.4	N/A	(Lignell, et al. 2011)
France	2004-06	93	2.5	1.6	(Antignac, et al. 2009)
Spain	2005	9	2.1	2.5	(Gomara, et al. 2011)
Belgium	2006	22	3.0	5.9	(Roosens, et al. 2010)
Italy	2005-07	13	1.3	N/A	(Alivernini, et al. 2011)
USA	2002	47	34.0	0.9	(Schechter, et al. 2003)
Canada	2003	10	50.4	0.4	(She, et al. 2007)
Australia	2007	10	7.6	0.3	(Toms, et al. 2009)
China	2004	19	2.5	3.0	(Sudaryanto, et al. 2008)
India	2009	45	1.1	0.4	(Devanathan, et al. 2012)
Korea	2008-09	21	2.7	N/A	(Kim, et al. 2011)
Tunisia	2010	36	8.3	N/A	(Ben Hassine, et al. 2012)

554 * N/A not analyzed

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562 **Table 3: Estimated exposure* (ng (kg bw)⁻¹ day⁻¹) of a 1 month old infant to the target**
563 **BFRs via breast milk under different scenarios**.**

	25th %ile	Average	Median	75th %ile
BDE-47	4.6	19.3	16.3	30.3
BDE-99	1.2	4.2	4.0	5.1
BDE-100	0.7	2.7	2.2	4.2
BDE-153	2.1	6.5	5.3	8.4
BDE-154	0.4	1.7	1.3	3.2
Σtri-hexa BDEs	10.0	34.9	29.4	56.4
BDE-209	<0.1	1.8	1.2	3.4

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565 * Values below LOQ were assumed to be 1/2 LOQ.

566 ** Based on an average body weight of 4.14 kg and a daily lipid intake of 24.4 g lipid day⁻¹

567 (U.S. EPA 2002.).

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575 **Table 4: Comparison of predicted adult body burdens arising from average and median**
 576 **daily exposures[#] to major target PBDEs with observed levels in human milk samples.**

	BDE-47	BDE-99	BDE-100	BDE-153	BDE-154	Σ₅BDEs	BDE-209
<i>Average intake* (ng day⁻¹)</i>							
Dust^a	1.10	1.80	0.24	0.31	0.17	3.70	4270
Diet^b	35	30	5.60	7.00	2.80	80	310
Air^c	0.90	0.60	0.14	0.05	0.03	1.70	9.40
<i>Median intake* (ng day⁻¹)</i>							
Dust^a	0.29	0.67	0.08	0.12	0.01	1.20	2975
Diet^b	35	30	5.60	7.00	2.80	80	310
Air^c	0.20	0.30	0.04	0.01	0.01	0.55	7.40
<i>Average predicted body burdens (ng g⁻¹ lw)</i>							
Dust	0.06	0.05	0.01	0.02	0.01	0.14	0.34
Diet	3.33	1.39	0.38	1.15	0.16	6.40	0.03
Air	0.11	0.05	0.01	0.01	0.01	0.20	0.01
Sum	3.49	1.49	0.40	1.19	0.18	6.74	0.38
<i>Median predicted body burdens (ng g⁻¹ lw)</i>							
Dust	0.01	0.02	0.00	0.01	0.00	0.04	0.24
Diet	3.33	1.44	0.38	1.15	0.16	6.45	0.03
Air	0.03	0.03	0.00	0.00	0.00	0.06	0.00
Sum	3.36	1.48	0.39	1.16	0.16	6.55	0.27
<i>Observed body burdens (ng g⁻¹ lw)</i>							
Average	3.28	0.71	0.45	1.09	0.28	5.92	0.31
Median	2.77	0.68	0.38	0.9	0.21	4.98	0.24

577

578 [#] Values below LOQ were assumed to be 1/2 LOQ.

579 * Based on average adult dust ingestion rate of 20 mg day⁻¹ (Jones-Otazo, et al. 2005),
 580 average inhalation rate of 20 m³ day⁻¹ (Currado and Harrad 1998) and average adult weight
 581 of 70 kg.

582 ^a Estimated from reference (Harrad, et al. 2008a); ^b Estimated from reference (UK Food
 583 Standards Agency 2006); ^c Estimated from references (Harrad, et al. 2006; Stapleton, et al.
 584 2009).

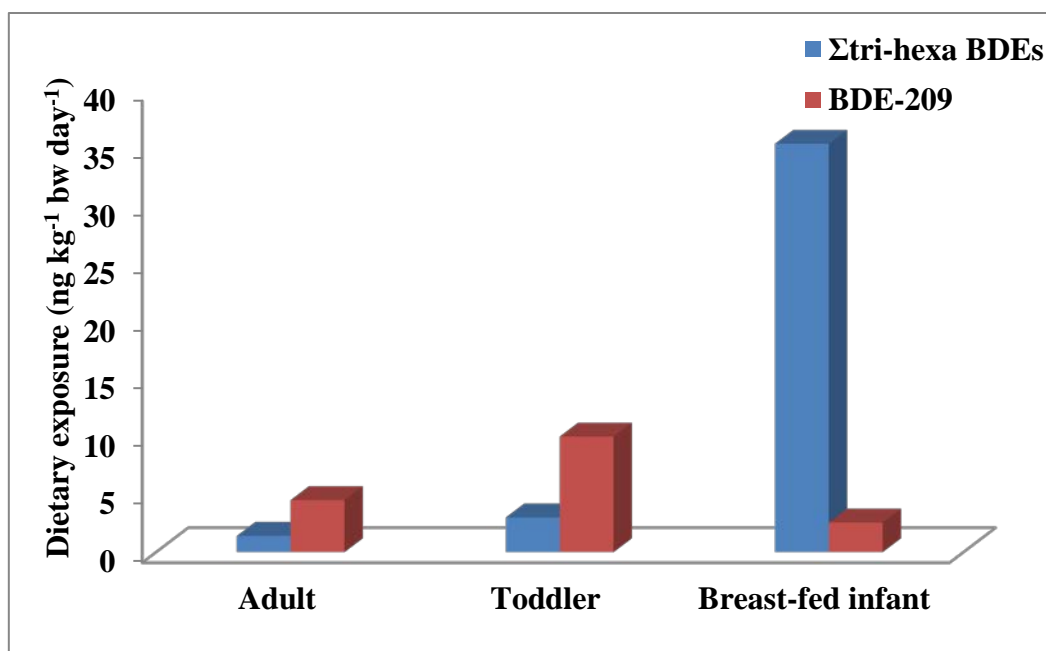
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589 **Figure 1: Average estimates of dietary exposure ($\text{ng (kg bw)}^{-1} \text{ day}^{-1}$) of UK adults*,**
590 **toddlers* and breast-fed infants** to PBDEs.**



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592 * From reference (UK Food Standards Agency 2006); ** This study.

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