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Predictors of human PBDE body burdens for a UK cohort

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1 Predictors of human PBDE body burdens
2 for a UK cohort

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16 Abstract

17 Human exposure to polybrominated diphenyl ethers (PBDEs) was investigated in a
18 cohort of 20 UK adults along with their anthropometric covariates and relevant
19 properties such as room surveys, lifestyle, diet and activity details. Selected PBDE
20 congeners were measured in matched samples of indoor dust, (n=41), vehicles (n=8),
21 duplicate diet (n=24), serum (n=24) and breast milk (n=6).
22 Combined exposure estimates via dust and diet revealed total PBDE intakes of 104 to
23 1,440 $\text{pg kg}^{-1} \text{bw d}^{-1}$ for ΣBDE_{3-7} and 1,170 to 17,000 $\text{pg kg}^{-1} \text{bw d}^{-1}$ for BDE-209. These
24 adult intakes are well within health reference doses suggested by the European Food
25 Safety Authority (EFSA) and the US EPA. Diet was the primary source of intake of BDE₃₋₇
26 congeners for the majority of the cohort, with dust the primary source of BDE-209.
27 Primary sources of PBDE exposure vary between countries and regions with differing
28 fire prevention regulations. Estimated infant exposures (ages 1.5 to 4.5 years) showed
29 that BDE-99 intake for one of the households did not meet EFSA's recommended
30 margin of exposure, a further two households were borderline for high level dust and
31 diet intake.
32 Males and those having a lower body fat mass had higher serum BDE-153. Higher meat
33 consumption was significantly correlated with higher BDE₃₋₇ in serum. A reduction in
34 dietary BDE₃₋₇ would therefore result in the greatest reduction in BDE-99 exposure.
35 Rooms containing PUF sofas or armchairs over 20 years old had higher BDE₃₋₇ in their
36 dust, and rooms with carpets or rugs of that age had higher dust BDE-209. Dusting
37 rooms more frequently resulted in significantly lower concentrations of all major
38 congeners in their dust. Correlation between BDE-209 body burden and dust or diet
39 exposure was limited by its low bioaccessibility. Although vehicle dust contained the
40 highest concentrations of BDE₃₋₇ and BDE-209, serum BDE₃₋₇ correlated most strongly
41 with bedroom dust.

42

43

44 1 Introduction

45 UK residents are still exposed to a class of potentially harmful brominated flame
46 retardants, polybrominated diphenyl ethers (PBDEs), even though European
47 Union regulations restricting their manufacture, use and importation came into
48 force in 2004 and 2008. Since the 1970s PBDEs have been incorporated into
49 fabrics, foam cushioning and plastics used in everyday items such as vehicles, soft
50 furnishings and electronics. PBDEs slow the rate of ignition and fire growth in
51 petroleum based polymers and resins. PBDEs are not chemically bonded to these
52 materials and are emitted into indoor dust and air through use and volatilisation
53 (Rauert and Harrad, 2015; Sjödin et al., 2003). They can then move into the wider
54 environment where they have been found in sewage sludge, soils and river and
55 lake sediments (Allchin et al., 1999; De Boer et al., 2003; Eljarrat et al., 2008;
56 Harrad et al., 2009). They are persistent organic pollutants as defined by the
57 United Nations Environment Programme's Stockholm Convention and have an
58 environmental half-life of several years. They can travel long distances in the
59 atmosphere and are lipophilic, concentrating in animal and marine fats. These
60 qualities and their wide usage have led them to permeate environments and food
61 chains around the world (Fromme et al., 2016).

62 A recent systematic review of human health consequences of exposure to PBDEs
63 concluded health effects may include thyroid disorders, reproductive health
64 effects, and neurobehavioral and developmental disorders (Kim et al., 2014).
65 Evidence of these effects has been seen in animal and *in vitro* research, where the
66 mechanism appears to be altered hormone regulation (endocrine disruption)
67 (Linares et al., 2015; Marchesini et al., 2008; Meerts et al., 2000; Viberg et al.,
68 2006). Exposure during key developmental stages in infancy is most damaging as
69 this is the time when altered hormone regulation will have the greatest impact.
70 Recent estimates of the economic cost of just the intelligence quotient (IQ) points
71 loss and intellectual disability due to PBDE exposure was \$266 billion in the USA
72 and \$12.6 billion in the EU (Attina et al., 2016). These figures must be balanced

73 against amounts saved due to fire prevention resulting from furnishing
74 flammability standards e.g. £140 million annual savings in the UK estimated by
75 prevention of death, injury and damage to property as a result of Furniture and
76 Furnishings Fire Safety Regulations 1988 that require use of flame retardant
77 chemicals. (BIS, 2009). PBDEs were only one group of flame retardant chemicals
78 from the several BFR groups commonly used to meet such regulations.

79 In 2004, use of two commercial PBDE products, Penta-BDE and Octa-BDE, were
80 restricted within the EU (European Council Directive 2003/11/EC) and voluntarily
81 phased out in the USA. In 2009, they were added to the Stockholm Convention list
82 of POPs for elimination. Penta-BDE had been primarily used in polyurethane foam
83 (PUF) in soft furnishings, vehicles and printed circuit boards, in greatest amounts
84 in the USA. Furnishings could contain one to four percent Penta-BDE to comply
85 with fire safety regulations (Hammel et al., 2017). The Octa-BDE commercial
86 product has been produced and used less widely than Penta-BDE. Its major use
87 has been in acrylonitrile-butadiene-styrene (ABS) plastics, such as electronics and
88 resin casings of office equipment. The Deca-BDE commercial product has been
89 added to furnishing textiles, and in high impact polystyrene (HIPS) for cables,
90 sockets, mobile phones, fridges and TV housings.

91 Concentrations of BDE-209 are higher in UK indoor dusts than in dusts from
92 mainland Europe (Frederiksen et al., 2009; Harrad et al., 2008b) as a result of the
93 UK's more stringent fire safety regulations (Furniture and Furnishings Fire Safety
94 Regulations 1988/1989, 1993 and 2010). Deca-BDE has been restricted from use
95 in electrical and electronic equipment in the EU since 2008 and was added to
96 Annex A of the Stockholm Convention list of POPs in 2017. Both diet and contact
97 with indoor dust constitute important exposure pathways for PBDEs (Abdallah and
98 Harrad, 2014). Foods from higher up the food chain, of animal origin, with a
99 higher fat content (i.e. fish), meat and dairy have higher PBDE concentrations
100 (EFSA, 2011). PBDEs will be circulating in our food chains for many years to come
101 (Harrad and Diamond, 2006), and will be re-circulated back into homes as a result
102 of plastics recycling (Samsonik and Puype, 2013) .

103 Whether dust or diet is the primary exposure source for an individual depends on
104 a number of factors; loading of PBDE in dust or food items and the amounts
105 ingested, whether and when PBDE technical products have been phased out in
106 that country and on the age of the individual (Bramwell et al., 2016a). PBDE intake
107 via ingestion and inhalation of dust is the major exposure route for young children
108 in the USA that have frequent hand to mouth behaviours and spend lots of time
109 on floors and carpets (Stapleton et al., 2012). Foetal exposure in the womb and
110 transfer of PBDEs from mother to child during breastfeeding are key exposures for
111 children during important developmental periods. For countries outside of the US
112 and Canada, the largest contribution to tri-hepta BDE body burden is thought to
113 be from diet, especially in regions where Penta-BDE use has been restricted for
114 longer. Dust is likely to be most important contributor to exposure to higher
115 brominated congeners in all regions (Sahlström et al., 2015).

116 The aim of this study was to determine the major dust and diet sources of PBDEs
117 for a north east England cohort and to consider any potential health risks. The
118 five specific objectives were: (a) to measure PBDE concentrations in dust from
119 homes, work places and vehicles, (b) to calculate relative intake of PBDE via dust
120 in the microenvironments, (c) to evaluate the relative importance of PBDE
121 exposure via indoor dust versus dietary PBDE exposure, (d) to compare intake
122 estimates with reference health values, (e) to investigate relationships between
123 matched environmental and biomonitoring data, and (f) to determine the most
124 effective means of reducing PBDE exposure for the cohort.

125

126 2 Materials and Methods

127 We used a cross sectional and purposive sampling strategy to provide a snap shot
128 of PBDE exposures and body burdens for individuals with expected high, average
129 and low exposures. By comparing individuals with expected divergent exposures,
130 we aimed to reveal the factors influencing body burdens.

131 2.1 Volunteer recruitment

132 We targeted individuals with a range of occupations and diets; such as workers in
133 electronics, soft furnishings, transport, office workers, outdoor workers, oily fish
134 eaters, omnivores and vegetarians. In 2010/11, following ethical approval for the
135 study, volunteers over 18 years of age and with six months or more of domestic
136 and occupational stability were recruited via local authorities, universities,
137 businesses, hospitals, playgroups and breast-feeding groups. A short pre-
138 screening questionnaire was used to identify volunteers that could provide the
139 optimum range of exposures. 79 couples completed the pre-screening
140 questionnaires, 10 couples were invited, and agreed, to participate in the full
141 study week. Further description of the cohort is provided in the Supplementary
142 Information. Volunteers gave written informed consent prior to participation.

143 **2.2 Timing of sample collection**

144 Participants undertook a 'sampling week' during which they completed an
145 exposure and food frequency questionnaire (FFQ), food- and activity-diaries, room
146 surveys including contents, usage and cleaning information and they were asked
147 not to vacuum or dust their home. We adapted the validated WHO-IARC EPIC
148 semi-quantitative dietary questionnaire for the study. On the seventh day of their
149 sampling week, participants collected their duplicate diet samples (DD), and the
150 researcher visited that evening to collect the DD samples, home and vehicle dust
151 samples, questionnaires and surveys. The participants then fasted until their blood
152 sample collection appointment the following morning where anthropometric
153 measurements were also taken. Two couples repeated the full sampling week,
154 with sampling points 6.5 and 7.5 months apart. This provided a longitudinal
155 dimension to the study and an element of validation. All sampling weeks took
156 place between April 1st 2011 and 28th February 2012.

157

158 **2.3 Serum, breast milk and duplicate diets**

159 Study participants collected an equal amount of whatever food they ate
160 throughout the day in a contaminant free (verified by tests carried out prior to
161 sampling) lidded polypropylene container for the 24 hour duplicate diet collection.

162 The next day they provided a fasted 60 ml blood sample at the Clinical Research
163 Facility of the Royal Victoria Infirmary in Newcastle. 50 ml breastmilk samples
164 were collected by either pump or manual expression up to 12 h before and 24 h
165 after provision of the blood sample and kept in pre-cleaned Nalgene containers.
166 Samples were stored at -18°C until transfer to the laboratory for analysis. Details
167 of the serum, human milk and duplicate diet sample collection and analysis have
168 been published previously (Bramwell et al., 2014; Bramwell et al., 2017).

169 **2.4 Dust samples**

170 Participants were requested not to vacuum or dust their home or vehicle during
171 the sampling week. Dust samples from main living areas (n=11), bedrooms (n=12),
172 and vehicles (n=8) were collected by a researcher following a standard sampling
173 protocol to allow direct comparison with previous studies (Abdallah and Harrad,
174 2009; Coakley et al., 2013; Harrad et al., 2008a; Harrad et al., 2008b). Samples
175 from workplaces (n=10) were collected during the sampling week at the
176 participants' (and their employers') convenience. Dust samples were extracted
177 and analysed at the University of Birmingham, UK, using previously published
178 methods for preparation, extraction, clean up, analysis and quality control
179 (Abdallah et al., 2009; Harrad et al., 2008a; Harrad et al., 2008b). Further details of
180 the dust sample collection, preparation, extraction and analysis are provided in
181 the Supplementary Information.

182 **2.5 QA/QC**

183 For the analysis of serum, breast milk and duplicate diet samples, the
184 performance characteristics of the methodology, including quality assurance
185 parameters such as limits of detection (LODs), precision, linear range of
186 measurement, recoveries etc. are included in the previous reports (Fernandes et
187 al., 2008; Fernandes, 2004). Further confidence in the data is provided by regular
188 and successful participation in laboratory proficiency testing and inter-comparison
189 schemes such as POPs in Food 2011 and 2012. PBDEs with IUPAC numbers 17, 28,
190 47, 49, 66, 71, 77, 85, 99, 100, 119, 126, 138, 153, 154, 183 and 209 were

191 measured. The congeners selected for analysis are those for which reference
192 standards are available. Typical LODs were 1 to 20 ng kg⁻¹ lipid for PBDEs.

193 For the dust sample analysis the average blank (including field blanks) plus 3
194 standard deviations was used for the limit of detection giving an average 0.7 ng
195 g⁻¹ for BDEs₃₋₇ (range 0.2-1.7) and 52 ng g⁻¹ for BDE-209. The PBDE ¹³C labelled
196 internal standard recoveries were: ¹³C-BDE 47 = 69 ± 20%, ¹³C-BDE 99 = 70 ± 20%,
197 ¹³C-BDE 153 = 69 ± 20% and ¹³C-BDE 209 = 17 ± 6%. The low recovery for BDE-209
198 indicates uncertainties in its measurement which are presented here with that
199 caveat. Measurement of SRM NIST 2585 had range 78% (BDE-47) to 122% (BDE-
200 49) and mean 100% of the certified contents.

201 **2.6 Exposure Assessment**

202 Concentrations of the PBDEs detected in milk and serum samples were lipid-
203 adjusted to allow comparison with the literature. PBDE intake for the 24 hrs of the
204 duplicate diet collection was measured using whole weight duplicate diet PBDE
205 concentrations multiplied by the mass of DD collected and divided by the weight
206 of the participant to give pg kg⁻¹ body weight day⁻¹.

207 PBDE intakes via dust were estimated by combining measured dust PBDE
208 concentrations with occupation time for individual's various microenvironments
209 (taken from their activity diary) using both average (20 mg/ day) and high (50
210 mg/day) adult dust intake rates average and high adult dust ingestion as
211 estimated by Jones-Otazo et al. (2005). Although dust ingestion rates may differ
212 between microenvironments and activities (as well as individuals), for the purpose
213 of this study, we have assumed that that dust ingestion occurred pro-rata to the
214 proportion of time spent in each microenvironment during the study week. This
215 was considered the only practical approach in the absence of data to confirm any
216 differences (Abdallah and Harrad, 2009). For time periods when participants were
217 in their home but not in one of the microenvironments measured, the median of
218 their home dust PBDE concentration was used. For time periods when they were
219 in an indoor environment but not in their own home the median of all dusts
220 collected for the study was used. Time spent outside was not assigned a PBDE

221 concentration. Intake rates via dust were divided by the participant's weight to
222 give pg PBDE intake kg^{-1} body weight day^{-1} .

223 PBDE intakes for average and high dust intake scenarios: average 20 mg d^{-1} , high
224 50 mg d^{-1} (Jones-Otazo et al., 2005) and diet intakes determined from the 24 h
225 duplicate diet concentrations were added together for comparison with the
226 European Food Safety Authority's (EFSA) chronic human daily dietary intake
227 estimations to determine the margins of exposure (MOEs). As PBDE exposure
228 during infancy is considered to present a greater risk to health than that for
229 adults, estimated average and high exposure scenarios for infants aged 1.5 to 4.5
230 years old were developed as well. Daily average (50 mg d^{-1}) and high (200 mg d^{-1})
231 dust intake estimations (Jones-Otazo et al., 2005) per kg body weight were
232 extrapolated from individual adult intake values determined for the study. These
233 were added to average and high dietary PBDE intake estimations from the UK
234 total diet study (TDS) (2012) data for infants aged 1.5 to 4.5 years old. Risk
235 assessment for infants from PBDE in breast milks collected for the study has been
236 previously reported (Bramwell et al., 2014).

237 **2.7 Data Analysis**

238 Associations between PBDE concentrations and intakes and potential predictors
239 were explored with scatter plots, box plots and correlations using IBM SPSS
240 Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp, Minitab 17 and Excel
241 (Microsoft Office 2013). The distribution of PBDEs in the different matrices was
242 assessed using Shapiro–Wilk statistic. As the majority of distributions were not
243 normal, non-parametric Spearman's ranking correlation coefficients were
244 determined. The criteria of $\alpha = 0.05$ for statistical significance was used. A one
245 sample t test was used to compare PBDE intake of omnivorous participants as
246 determined by duplicate diet collection and similar data collected by Harrad et al.
247 (2004) to investigate any temporal trend in dietary exposure. Statistical analyses
248 were mostly descriptive and correlations do not have sufficient sample numbers
249 to be robust. Details of further statistical analyses of room survey data are
250 presented in the Supplementary Information. Where measurements were below
251 limits of detection (LOD) values of $\text{LOD} \times 0.5$ have been assumed (median bound).

252 ΣBDE_{3-7} was calculated as the sum of all BDE congeners measured except for BDE-
253 209.

254

255 **2.8 Human health risk characterisation**

256 Potential health risks were calculated from the sum of dust and dietary intake of
257 PBDEs using the margin of exposure (MOE) approach as applied by the European
258 Food Safety Authority (EFSA) for dietary exposure health risk assessment. The
259 MOE is the ratio of the dose at which a small but measurable adverse effect has
260 been reported versus the level of exposure of the population under current
261 consideration. The EFSA Panel on Contaminants in the food chain (EFSA, 2011)
262 identified effects on neurodevelopment as the critical endpoint using BMDL_{10} for
263 neurobehavioural effects in mice induced during a relevant period for brain
264 development. Chronic human intakes, associated with body burdens at the
265 BMDL_{10} for BDEs-47, -99, -153 and -209, were estimated to be 172, 4.2, 9.6 and
266 1,700,000 $\text{ng kg}^{-1} \text{bw day}^{-1}$ respectively. For PBDEs, EFSA consider that an MOE
267 ratio above 2.5 indicates that a health concern is unlikely, with risk decreasing as
268 the MOE increases (EFSA, 2011). It should be noted that although human intakes
269 of concern are presented as daily doses these represent chronic intake and as
270 such would be better represented as weekly or monthly intakes as daily intakes
271 can be exceeded on occasion without concern as long as other days have lower
272 exposures.

273

274 **3 Results and Discussion**

275 Our cohort consisted of 10 male-female cohabiting couples living in northeast
276 England in 2011/12. All participants completed full sample and data set collection.
277 Participants were recruited from as wide a pool of socio-economic class,
278 occupation, diet and location as possible, however, the small number of
279 participants and the focus on breastfeeding mothers means that results are not
280 representative of all UK residents' exposures. The benefit of the small cohort was

281 that detailed information could be collected for each individual allowing the
282 investigation to include almost all contributing factors in PBDE exposure known at
283 the time. Further details of occupations, diets, parity, breastfeeding and other
284 lifestyle and anthropometric factors are presented in Supplementary Information.
285 Previously published serum, breastmilk, and duplicate diet concentrations
286 (Bramwell et al., 2014; Bramwell et al., 2016b) have been further examined in this
287 investigation, along with new matched dust concentrations, diet and dust intake
288 estimations and exposure and food frequency questionnaire, seven day food and
289 activity diary and room survey information in order to provide as complete a
290 picture of participants' PBDE exposures as possible.

291 **3.1 Dust PBDE concentrations**

292 Dust samples were collected from 40 micro-environments frequently used by the
293 study participants. Main living areas (n=10), bedrooms (n=12) and home offices
294 (n=2) were sampled. Workplaces were sampled if access was granted by
295 employers (n=8). None of the domestic samples were from open plan homes. Four
296 of the workplace samples were from open plan indoor spaces. Vehicles were
297 sampled if participants regularly spent more than five hours each week in them
298 (n=8). We measured PBDEs in dust from all of the microenvironments sampled.
299 Individual concentrations for all PBDEs in each dust sample are presented in
300 Supplementary Information Tables SI 1-4 and summaries of the dust
301 concentrations in different rooms are presented in Table 1. Median dust \sum BDEs₃₋₇
302 concentrations were highest in vehicles (179 ng g⁻¹) followed by living rooms,
303 bedrooms then workplaces (137, 102 and 84 ng g⁻¹ respectively). Median BDE-209
304 concentrations in dust were also highest in vehicles (19,000 ng g⁻¹) then
305 bedrooms, living rooms and workplaces (3,530, 2,960, and 2,300 ng g⁻¹
306 respectively). The highest concentration of \sum BDEs₃₋₇ was measured in a bedroom
307 (7,320 ng g⁻¹ dust), the highest BDE-183 in the rear of a work van (367 ng g⁻¹) and
308 the highest BDE-209 in a car (137,000 ng g⁻¹). Summaries of dust PBDE
309 concentrations in the different microenvironments are compared with previous
310 UK and international data in Table 2. Measurements in this study were in keeping
311 with previously published UK data (Harrad et al., 2008a; Harrad et al., 2008b;

312 Pless-Mullooli et al., 2006; Sjödin et al., 2008) and in agreement with the theory
313 that BDE-209 usage was greater in the UK (Fromme et al., 2016; Harrad, 2015).
314 Results were directly comparable to studies by Harrad et al. (2008a; 2008b) as we
315 used the same sampling protocol, sampling equipment and laboratory techniques.

316 We compared room survey information such as counts and age of soft furnishings
317 and electronics and room cleaning frequencies with the concentrations of PBDEs
318 in each room. Details from individual room surveys are provided in Supplementary
319 Information Table SI5. We did not find that simple counts of soft furnishings or
320 electronics were good predictors of high or low PBDE loading. The clearest
321 association between room contents and PBDE concentrations in dust were for
322 BDE-209 if the room contained a carpet or rugs over 20 years of age (see
323 Supplementary Information Figure 2) . Counts of large PUF items over 20 years
324 old or office chairs from the USA (adhering to Californian state fire retardancy
325 regulations TB117) correlated significantly with concentrations of Penta mix BDEs
326 only, BDE-47 ($r=0.37$, $p=0.036$), -99 ($r=0.35$, $p=0.047$) and $\sum BDE_{3-7}$ ($r=0.37$,
327 $p=0.039$). Higher dusting frequency demonstrated the greatest correlation with
328 lower dust PBDE concentrations, with BDEs-47, -99, -153, -154 and -209 all with
329 correlation significant at the 0.01 level and BDE-100 with correlation significant at
330 the 0.05 level. Table SI 6 in the Supplementary Information contains further
331 correlation data. Discussion of apparent differences between repeat sampling
332 weeks' dust data is provided as Supplementary Information.

333 We found that concentrations of \sum Penta product BDEs in the bedroom were
334 significantly correlated with those in all other environments measured; living
335 rooms ($r=0.43$, $p=0.05$), workplaces ($r=0.71$, $p=0.05$) and vehicles ($r=0.90$, $p=0.02$).
336 Concentrations of \sum Penta product BDEs in living room dusts correlated strongly
337 with those in workplaces ($r=0.90$, $p=0.01$) but not vehicles ($r=0.30$, $p=0.60$). A
338 larger data set may have revealed alternative findings, particularly for workplaces
339 and vehicles. We suggest that dust particles may briefly adhere to and then be
340 shaken from skin, hair, clothing and footwear causing distribution among key
341 environments used by participants. Further correlation data is provided in
342 Supplementary Information Table SI13.

343

344 **3.2 Intake of PBDEs via dust**

345 The ranges of average (20 mg dust ingested d⁻¹) and high (50 mg dust ingested d⁻¹)
346 PBDE intakes via dust for our study participants was 13.8-1,010 and 35-2,520 pg
347 kg⁻¹ bw day⁻¹ for \sum BDEs₃₋₇, with 281 to 15,900 and 702 to 39,600 pg kg⁻¹ bw day⁻¹
348 for BDE-209 via dust. Our \sum BDEs₃₋₇ estimates were similar to previous UK and
349 German \sum BDEs₃₋₇ intake estimates (Fromme et al., 2009; Harrad et al., 2008a) and
350 an order of magnitude lower than those in the USA (Harrad et al., 2008b). In
351 contrast, our BDE-209 intakes from dust were similar to those of the USA (Harrad
352 et al., 2008b) and an order of magnitude higher than Belgian and German
353 estimates (Fromme et al., 2009; Roosens et al., 2009) (see Supplementary
354 Information Table 6). The wide range of intakes reflected the diverse PBDE
355 loadings measured in microenvironment dusts. For this cohort, the influence of
356 specific items in specific microenvironments could be reasonably speculated on a
357 case by case basis. However, although we expected our participant with
358 occupational PUF and furnishing fabric exposure to have a raised PBDE body
359 burden, their fastidious cleaning habits appear to have reduced their exposure.

360 The greatest proportion of the estimated dust intake for \sum BDEs₃₋₇, BDE-183 and
361 BDE-209 took place in the bedroom (means 43%, 38% and 33% respectively) due
362 to the greater amount of time spent in bedrooms. Workplaces and living rooms
363 were the second most important microenvironments for \sum BDEs₃₋₇ exposure (mean
364 19%, 13%) and BDE-183 (20%, 21%). Vehicles were the second most important
365 microenvironment for BDE-209 intake (20%). The relative proportions of PBDE
366 intakes in different microenvironments for individual participants is illustrated in
367 Figure 1. Our finding that the greater proportion of exposure to all congeners
368 occurs in the bedroom is in keeping with our finding of an association between
369 bedroom dust and serum concentrations of the PBDE congeners found in the
370 commercial Penta-BDE products (BDE-47, -99, -100, -153) ($r=0.42$, $p=0.04$), an
371 association that has also been reported elsewhere (Ali et al., 2014; Coakley et al.,
372 2013; Watkins et al., 2012).

373 **3.3 Relationships between PBDE in dust and body burdens**

374 We compared PBDE concentrations in dust in the different indoor environments
375 with their matched PBDE body burdens. Significant associations were noted
376 between Penta-mix BDEs in bedroom dust and serum ($r=0.45$, $p=0.04$). BDE-153 in
377 bedroom dust was significantly associated with BDEs-47 ($r=0.45$, $p=0.03$), -99
378 ($r=0.45$, $p=0.03$), -209 ($r=0.41$, $p=0.05$) and $\sum\text{BDEs}_{3-7}$ ($r=0.45$, $p=0.03$) in serum.
379 BDE-153 in serum was associated but not significantly with BDEs-153 (0.39, 0.06)
380 and $\sum\text{BDEs}_{3-7}$ (0.39, 0.06) in bedroom dust. BDE-47 was associated but not
381 significantly in living room dust and breast milk (0.77, 0.07). BDE-209 was
382 significantly correlated in serum and workplace dusts (0.72, 0.02) however this
383 was strongly influenced by one data point. Also correlated but not significantly in
384 workplace dusts were BDEs-47 (0.57, 0.07) and -99 (0.53, 0.09). Table SI 7 in
385 Supplementary Information provides further dust and body burden correlation
386 data. No significant correlations were found between vehicle dust and serum
387 despite vehicles having the highest PBDE concentrations in their dust, possibly due
388 to participants spending less time in their cars than in other environments
389 measured. The associations between bedroom dust and serum might be expected
390 due to participants spending the greatest proportion of their day in this room,
391 similarly for associations with workplace dust and serum.

392

393 **3.4 Dietary intake of PBDEs**

394 We estimated participants' PBDE intake from diet using three different methods,
395 (i) a 24 hour duplicate diet sample collected the day before taking serum and milk
396 samples, (ii) a seven day food diary completed the seven days prior to serum and
397 milk sampling and (iii) a food frequency questionnaire (FFQ) to represent longer
398 term eating habits. Concentrations of PBDEs in the 24 hour duplicate diet samples
399 summarised in Table 1. BDEs₃₋₇ were measurable in all of the duplicate diet
400 samples and BDE-209 in 79% of them. 24 hour duplicate diet PBDE concentrations
401 were converted to daily dietary intake estimates which ranged from 82 – 1,320 pg
402 kg^{-1} bw for $\sum\text{BDEs}_{3-7}$ and <0.8- 1,860 pg kg^{-1} bw for BDE-209. BDE-209 made up a
403 median of 73% of the total PBDE exposure from diet. Estimates of individuals'
404 PBDE intake via diet are provided in Supplementary Information Table SI 11. The

405 mean intake estimates of BDEs-47, -99, -100, -153 and -154 for the omnivores in
406 this study were significantly lower than those measured by Harrad et al. (2004) for
407 duplicate diet samples collected in the West Midlands of the UK in 2002 ($p=0.01$).
408 The 2002 lower bound mean intakes were within the maximum intakes estimated
409 by this study for BDEs -47, -100, -153 and -154 and upper bound intakes for BDEs -
410 47, -100, and -154. These findings indicate a reduction in dietary exposure during
411 the 10 years between the two studies, with the greatest reductions being for BDE-
412 99 then BDE-153.

413 Meat, fish and dairy portion consumption estimates compared well between the
414 FFQ and seven day food diaries. Meat portions consumed per week ranged from
415 none to 14 or 15 (FFQ and diary respectively), with median 6.3 or 8 portions. Fish
416 and seafood portions consumed per week ranged from none to 3.5 (maximum for
417 both FFQ and diary), with median 1.8 or 2 portions. Dairy portions consumed per
418 week ranged from none to 25 or 18 (FFQ and diary respectively), with median 8.0
419 or 8.5 portions. A summary of selected information from the FFQ, diary and 24h
420 duplicate diet is presented in Table 3.

421

422 **3.5 Relationships between PBDE in diet, serum and breastmilk**

423 We compared PBDE body burdens with concentrations in the duplicate diet
424 finding a significant association for $\sum\text{BDEs}_{3-7}$ in both ($r=0.41$, $p=0.05$). Serum
425 samples were collected from fasted participants in order for the serum sample to
426 represent the participants' background PBDE body burden without influence from
427 recently consumed food. Breastmilk samples were not necessarily collected in a
428 fasted state. The complex relationship between historic PBDE deposits in adipose
429 tissue, recent diet, serum and breastmilk is beyond the scope of this paper. We
430 found limited correlation between congeners in serum and breastmilk (see
431 Supplementary Information Table SI 8), possibly the result of transfer of PBDEs
432 from serum to milk varying between different congeners. Mean serum/milk ratios
433 generally increased with molecular size and hydrophobicity, e.g. 1.3, 3.1 and 6.0
434 for BDEs-47, -99 and -209. This pattern was in keeping with findings of a 2012

435 review of PBDE in matched serum and breastmilk samples (Mannetje et al., 2012).
436 BDE-153 in the body appears to follow a different pattern with a serum/milk ratio
437 of 0.4, i.e. more in milk than serum.

438

439 We found that the number of meat portions consumed in the week prior to
440 sampling had significant positive correlations with BDEs-99 ($r=0.46$, $p=0.01$) -153
441 ($r=0.44$, $p=0.03$) and $\sum\text{BDEs}_{3-7}$ ($r=0.43$, $p=0.04$) in serum. Further correlation data
442 between dietary information is provided in Supplementary Information Table S19.
443 The UK FSA 2006 TDS found meat products (followed by fish) to contribute most
444 to the PBDE intake of the general UK population (EFSA, 2011; FSA, 2006). For
445 participants in this study, meat portions consumed exceeded fish portions. Our
446 earlier review of associations between PBDE body burden, dust and diet
447 (Bramwell et al., 2016a) also found eating meat to be the most frequently
448 reported association (eating dairy and fish were next). Similarly, a nationwide
449 study in the USA found vegetarians to have 23% lower, and heavy red meat
450 consumers to have 18% higher total PBDEs in serum than omnivores (Fraser et al.,
451 2009).

452

453 **3.6 Anthropometric and questionnaire covariates of PBDE body burden**

454 As well as participants' height, weight and body fat mass measurements,
455 information on travel habits, hand to mouth behaviours, parity, numbers of
456 household members, hobbies and occupations was also collected to look for
457 indicators of higher serum and breast milk PBDE concentrations. These
458 associations are presented in Supplementary Information Table S110. We found
459 serum BDE-153 concentrations to be significantly associated with sex ($r= -0.60$,
460 $p=0.01$), percentage of body fat mass ($r=-0.49$, $p=0.02$), parity in women ($r=-0.57$,
461 $p=0.05$) and working with electronics ($r=0.59$, $p=0.01$). Males generally had higher
462 BDE-153 in serum than females, in keeping with the findings of a recent Swedish
463 study of 170 adults (Bjermo et al., 2017) and a nationwide study in the USA that
464 found males generally had higher BDE_{3-7} body burdens (Fraser et al., 2009). We

465 hypothesise there may be two factors influencing the higher serum
466 concentrations of males in this study, (i) men generally had lower BMI values;
467 seven of the females had recently been pregnant which would increase their BMI
468 and (ii) 9 of the 10 female participants in the study had undergone some
469 depuration effect during pregnancy and breast feeding which their male partners
470 had not. In a study of the breastmilk of 83 women at three and 12 months
471 postpartum, BDE-153 showed a significant increase over time (Daniels et al., 2010)
472 suggesting that BDE-153 present in adipose fat compartments from historic
473 exposures may have been mobilised during the nursing period. Storage of BDE-
474 153 in fat compartments in the body has been suggested as the reason for dilution
475 in the serum of people with higher BMI (Cequier et al., 2015; Fraser et al., 2009).
476 Why these findings for BDE-153 are not consistent with findings for other
477 congeners is not clear but it may be linked to its longer human half-life (Geyer et
478 al., 2004).

479

480 **3.7 Was diet or dust the major source of PBDE exposure for this cohort?**

481 Diet was the major source of ΣBDEs_{3-7} for this cohort making up a median of 85%
482 of the total intake when using duplicate diet data with the average dust ingestion
483 estimate of 20 mg d⁻¹. This was a somewhat lower proportion than comparable
484 previous studies estimates of 95% (UK), 96% (Belgium) and 97%, (Germany)
485 (Abdallah and Harrad, 2014; Fromme et al., 2009; Roosens et al., 2009) due to our
486 higher median ΣBDEs_{3-7} dust concentration and the notably higher concentration
487 of ΣBDEs_{3-7} in the German duplicate diets (see Table SI 6). We did not include
488 estimates of intake of PBDEs from indoor air in our totals. Previous studies have
489 found PBDE intake from air to constitute <1% of total PBDE intake (Fromme et al.,
490 2009) and a maximum of 2% (Abdallah and Harrad, 2014).

491 Considering only a cohort's average intake hides the substantial variation between
492 individuals and their exposure sources - something this study has been able to
493 demonstrate clearly (see Figure 2 and Supplementary Information Table SI 6). An
494 individual's total PBDE intake is a combination of dust concentrations in different

495 environments, time spent in them and dietary habits. For example, the proportion
496 of $\sum\text{BDEs}_{3-7}$ BDE intake provided by dust for an average dust intake rate had a
497 median 4% but ranged between 0.7% (8M) and 32% (5F). Both these participants
498 lived rurally, the former on a smallholding, the other on a farm. 8M spent the
499 most time outdoors (almost 9 hours each day), had a low Penta-BDE loading in
500 their bedroom dust and, despite a generally home-grown and organic diet, a
501 duplicate diet intake in the 3rd quartile. 5F's relatively high dust intake (32% using
502 average dust intake and 54% using high dust intake rates) was due to having the
503 room (bedroom) with the highest $\sum\text{BDEs}_{3-7}$ concentrations measured in the study.
504 Although 5F consumed a vegetarian diet their dietary $\sum\text{BDEs}_{3-7}$ intake was in the
505 top quartile.

506 Dust was the greatest source of BDE-209 for our entire cohort, with median
507 intakes making up 75% and 88% of the total BDE-209 intake for average and high
508 dust intake rates respectively, lower than previous UK estimates of 94% and 99%
509 (Abdallah and Harrad, 2014; Harrad, 2010) possibly due to declining use of Deca-
510 BDE product and differences between cohorts in the different studies. Individual
511 participants' proportion of total BDE-209 intake provided by dust for average dust
512 intake rate ranged from 14% (8M) to 100% (1Fii and 1Mii). Participant 10M had a
513 significantly greater BDE-209 concentration than their partner possibly a reflection
514 of the relatively high amount of time spent in their vehicle (23% of their time) and
515 BDE-209 concentration in their car (30,338 ng/g).

516 We found the range of individuals' intakes of $\sum\text{BDEs}_{3-7}$ from dust to be five times
517 greater than their intakes from diet. The highest total intake (using average dust
518 intake scenario) was 16 times greater than the lowest reported intake. Our data
519 agrees with previous hypotheses that the wide range in PBDE concentrations in
520 room dusts (compared with the range seen in diets) may be the reason some
521 individuals have significantly higher internal dose (Harrad et al., 2008b; Petreas et
522 al., 2003; Thomas et al., 2006; Wu et al., 2007). Dust generation, dust ingestion
523 rates, and cleaning frequencies (both microenvironments and hand washing) may
524 also be influential.

525 Our study corroborates previous studies findings that average PBDE intakes in the UK are
526 broadly similar to those in mainland Europe, where meat is the major source of Penta-
527 BDEs for the average person but dust is the major source of BDE-209 (Bramwell et al.,
528 2016a; Harrad et al., 2008b). For infants, the average contribution to total intakes from
529 diet were >90% for \sum BDEs₃₋₇ and 69% for BDE-209. At the high dust ingestion rate this
530 decreased to 35-50% for \sum BDEs₃₋₇ and 88% for BDE-209. These figures indicate similar
531 proportional intake for infants from diet to our adults, although with considerably higher
532 amounts ingested per kg body weight (see Table 3).

533

534 **3.8 Study Limitations**

535 This study involved a relatively small cohort of 20 individuals (10 UK couples). The study
536 philosophy concentrated more on the details and habits of the volunteers in order to
537 understand their individual exposures. The volume of usage of PBDE mixtures such as
538 PentaBDE, the timelines of product introduction and restriction, either voluntary or
539 regulation enforced, and the type of usage, are all variables in general population
540 exposure. For example, a far greater volume of the PentaBDE mixture was used in the
541 USA and Canada compared to Europe and this is reflected in the relatively higher
542 concentrations of related congeners measured in serum, and in house dust levels from
543 North America. Also, where we found diet to be the most important exposure pathway
544 for Penta mix BDEs, studies such as (Lorber, 2008) have shown that dust is a major
545 pathway for PentaBDE in North American populations. When personal details and habits
546 are considered, the exposure assessment is even more unique. Thus, the finding of this
547 study are not intended to be representative of the UK as a whole, or even less, other
548 regions of the world.

549

550 **3.9 Risk characterisation**

551 The most relevant congener from a health risk perspective is BDE-99 but there is
552 no agreement on a safe intake. The US-EPA suggests a reference dose 100 ng/kg
553 bw/day (US-EPA, 2006) whereas the more recent EFSA suggested health reference
554 value is 4.2 ng/kg bw/day with an MOE of 2.5 (EFSA, 2011). We investigated
555 potential health risk from our estimated PBDE intakes by comparing them with
556 both these reference values (see Table 4 and Table SI12). The combined

557 uncertainties from household types, sampling and measurement is likely be quite
558 high and should be borne in mind. No health concerns are expected from the
559 PBDE intakes estimated in this study for adults as all had MOEs over 2.5 (EFSA,
560 2011). The lowest adult MOEs were 2.8 and 3.7 for BDE-99 using a high dust
561 intake rate for household 5 with the high BDE₃₋₇ measurements in their bedroom.
562 Accordingly, estimated infant daily exposures to BDE-99 for the same home have
563 MOEs below those recommended by EFSA for chronic exposure. Using average
564 diet intake data from the 2012 UK TDS with dust exposure data from this study
565 with average dust intake rates we found the lowest MOE estimation to be 2.3
566 which is similar to the EFSA recommended MOE of 2.5 deemed to indicate a
567 potential health risk. Using high dust intake rates with dust data for this study and
568 97.5th percentile (P97.5) dietary intake estimates from the 2012 UK TDS this MOE
569 dropped to 0.7 and two additional homes indicated high infant intake MOEs
570 between 2.5 and 3. All other adult and infant MOEs using EFSA reference values
571 and all MOEs using US EPA values were comfortably above the recommended
572 MOE. Follow-up measurement of the PBDE body burdens for infants of parents
573 participating in this study could help describe associations with raised intake
574 estimations.

575

576 4 Conclusions

577 This detailed study is the first anywhere to document concentrations of PBDEs,
578 including BDE-209, in samples of indoor dust and diet with matched human serum
579 and breast milk concentrations. Our findings confirmed that both diet and dust
580 make a contribution to PBDE body burdens and provide new evidence of a wide
581 range in their relative contributions between individuals. Diet appeared to be the
582 primary source of intake of BDE₃₋₇ congeners for the majority of this cohort, and
583 meat consumption demonstrated the strongest significant positive association
584 between diet type and serum BDE₃₋₇ concentrations. Dust was the cohort's
585 primary source of BDE-209. Rooms containing a carpet or rugs over 20 years old
586 had higher BDE-209 concentrations in their dust. Rooms that were dusted more

587 frequently had less BDE-209, as well as less Penta mix PBDE congeners. Rooms
588 containing sofas or armchairs over 20 years old had higher concentrations of
589 commercial Penta mix PBDE congeners. BDE-209 concentrations in room dusts did
590 not widely correlate with BDE-209 body burdens, possibly due to the congener's
591 relatively large molecular size and low bioaccessibility. Correlations between BDE₃₋
592 ₇ congeners in serum and indoor dust were strongest in bedrooms in keeping with
593 the greater proportion of time spent there. Being male and having a lower body
594 fat mass were indicators of higher serum BDE-153 for this cohort. BDE-99 was the
595 congener demonstrating the lowest MOE (and therefore the greatest health risk)
596 and although we found a reduction in dietary exposure to this and other Penta-
597 mix PBDEs since 2002, reducing dietary exposure would still have the greatest
598 effect in reducing body burdens.

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606

607 **Conflicts of interest:** None

608

609 References

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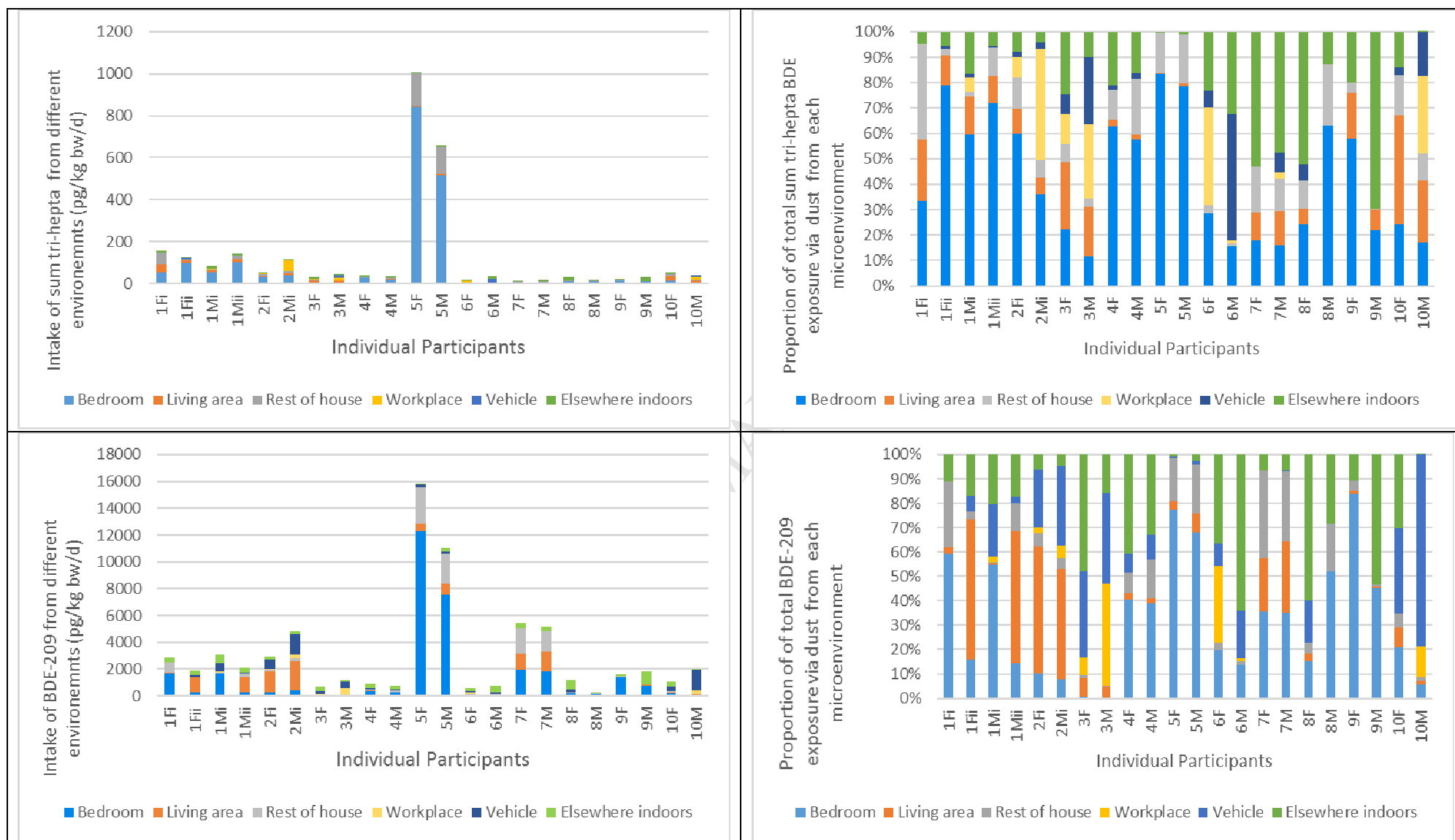


Figure 1. Individuals' proportional exposure to PBDEs via dust in different environments, calculated from dust concentrations and seven day activity diary data

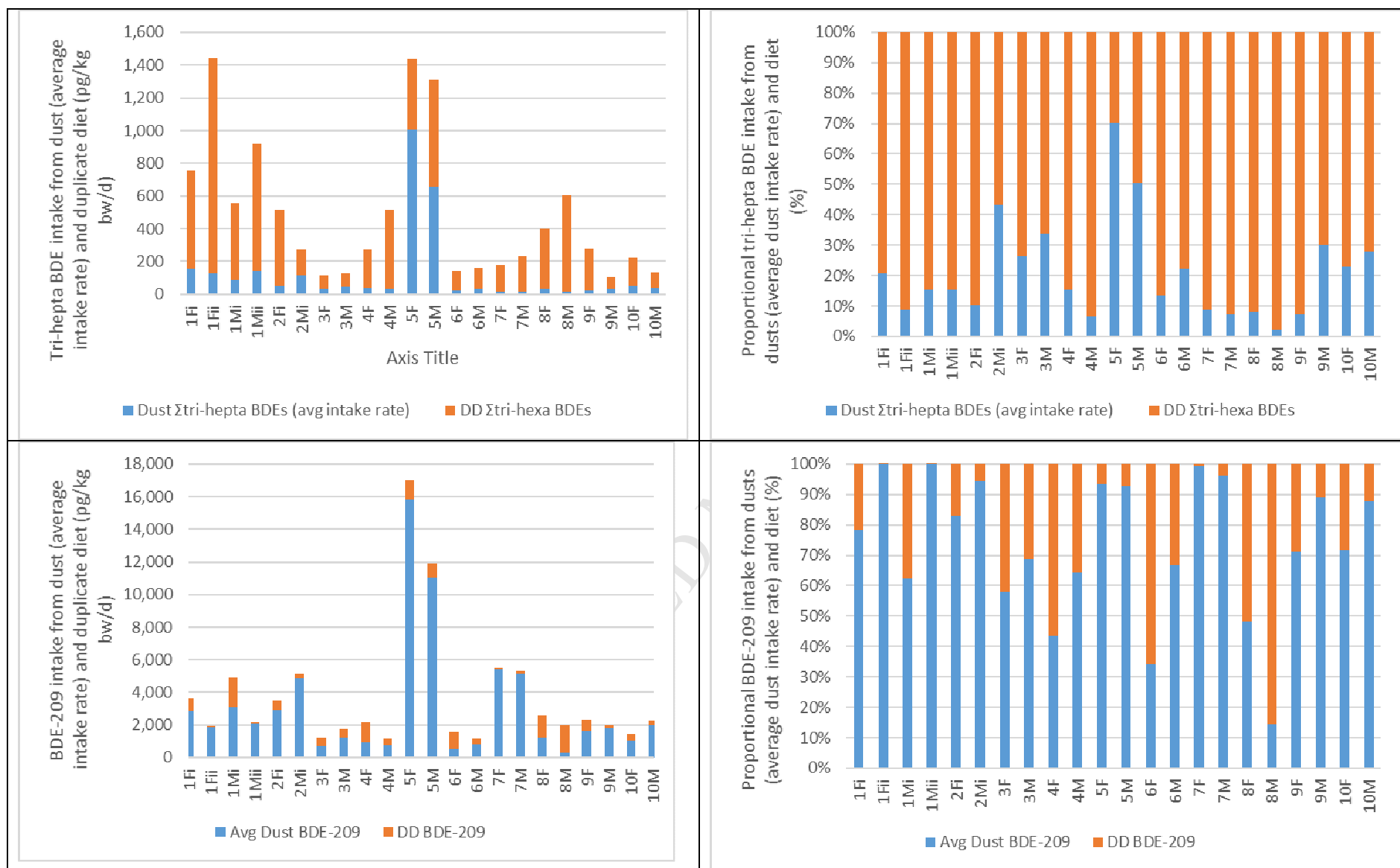


Figure 2. Comparison of individual participants' Σ tri-hepta BDE and BDE-209 intakes via dust and diet using average (20 mg day^{-1}) dust intakes and 24 h duplicate diet data ($\text{pg kg}^{-1} \text{ bw d}^{-1}$).

Highlights

- We report intake and body burdens of tri-hepta BDEs and BDE-209 for 20 UK adults
- Diet was the major source of tri-hepta BDEs, meat associated with higher exposure
- Dust was the major source of BDE-209, more frequent dusting reduced exposure
- Health concerns are indicated for infants with high PBDE intake from dust and diet