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UK dietary exposure to PCDD/Fs, PCBs, PBDD/Fs, PBBs and PBDEs: Comparison of results from - 24 hour duplicate diets and total diet studies

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UK dietary exposure to PCDD/Fs, PCBs, PBDD/Fs, PBBs and PBDEs: Comparison of results from a 24-hour duplicate diet and total diet studies

Chemicals in food are monitored to check for compliance with regulatory limits and to evaluate trends in dietary exposures, among other reasons. This study compared two different methods for estimating human dietary exposure to lipophilic persistent organic pollutants (POPs) during the period 2011/12: (a) the 2012 Total Diet Study (TDS) conducted by the UK Food Standards Agency and (b) a 24- hour duplicate diet (DD) study of 20 adults from the North East of England. The equivalence of the two approaches was assessed, anything less than an order of magnitude could be considered reasonable and within 3-fold (equivalent to half a log) as good. Adult dietary exposure estimates derived from the DD study for both average and high level (97.5th percentile) consumers compared well with those from the TDS. Estimates from the DD study when compared with those from the TDS were within 10% for P97.5 for total PCDD/F/PCB with divergence increasing to a factor of 3.4 for average BDE-209. Most estimates derived from the TDS were slightly higher than those derived from the DD. Comparison with earlier UK TDS data over the last 30 years or so, confirmed a gradual decline in levels of PCDD/F/PCBs in food. Such comparisons also indicated peaks in dietary exposure to ∑PBDE (excluding BDE-209) between 2000 and 2005. Exposure estimates for all measured compounds using both TDS and DD data were found to be within recommended tolerable daily intake values where available or within acceptable margins of exposure.

Keywords: duplicate diet study; total diet study; validation; risk characterisation; environmental contaminants

Introduction

Chlorinated dioxins and furans (PCDD/Fs) and polychlorinated biphenyls (PCBs) are recognised persistent environmental contaminants that have been regulated within the EU since 2002 (Council Regulation 2375/2001). These regulations were introduced following the 'Belgian dioxins crisis' in 1999 when PCDD/Fs and PCBs were introduced into the food chain via PCB contaminated animal feed. This resulted in high levels of PCDD/Fs and PCBs in meat products and eggs from Belgian, French and Dutch farms (Bernard et al. 1999) where the feed had been used, and in foods that used products from these sources as ingredients. PCDD/Fs and PCBs accumulate in the food chain, concentrating in the fatty tissue of animals. Diet is the major route of human exposure to PCDD/Fs and PCBs for most individuals without specific occupational exposure. In 2004 an international environmental treaty, 'The Stockholm Convention', came into force with the aim of eliminating production, use and unintentional release of persistent organic pollutants (POPs) in signatory countries (Stockholm Convention on POPs, 2001). PCBs and PCDD/Fs were included in the first ratification of the Convention, listed in the initial 'dirty dozen' of POPs. In Europe PCDD/Fs and PCBs are regulated in food through Commission Regulation 1881/2006 which sets maximum levels for certain contaminants in foodstuffs. This regulation has been subject to a large number of amendments, some of which relate to limits for dioxins and PCBs (Commission Regulations 565/2008, 420/2011, 594/2012, 1067/2013 and 2015/704). A key amendment has been Commission Regulation 1259/2011 which introduced limits for non-dioxin-like PCBs and updated limits for PCDD/Fs and dioxin-like PCBs using 2005 WHO-TEFs.

Brominated dioxins and furans (PBDD/Fs) have similar physicochemical and toxicological properties to their chlorinated analogues (Van den Berg et al. 2013). They originate from

similar anthropogenic sources as PCDD/Fs, such as incineration, particularly of bromine-containing waste, or chemical manufacture. PBDDs may also have biogenic origin such as photochemical formation from hydroxylated PBDEs (Arnoldsson et al. 2012). Polybrominated biphenyl (PBB) flame retardants are similar to PCBs in structure, manufacture, contamination pathways and toxicological impact on human health, and have some similarities in their use. The use of PBBs as textile flame retardants was phased out from the 1970s onwards and they have not been used or manufactured in the EU since 1996 (D'Silva et al. 2004). PCDD/Fs and dioxin-like compounds bind to the Ah receptor and are widely understood to cause damage to the immune system, to affect the endocrine system, to give rise to reproductive and developmental problems, and may cause cancer (EFSA 2012).

Polybrominated diphenyl ethers (PBDEs) are a class of flame retardant that have been used to meet fire safety regulations for fabrics, furnishings, electronics and vehicles since the 1970s, when they were first used as a replacement for PBBs. During the use and lifetime of a product containing PBDEs, they can be released into indoor air and dusts (Sjödin et al. 2003) and into the wider environment where they are now ubiquitous (Harrad et al. 2010). PBDEs are persistent, undergo long range transportation and are found throughout environments and food chains across the globe (Harrad and Diamond 2006). Two commercial PBDE products, Penta-BDE and Octa-BDE, were added to the Stockholm Convention's list of POPs for elimination in 2009 (Stockholm Convention on POPs, 2009).

Governments and international organisations monitor chemicals in food to evaluate dietary exposures and to protect consumers by ensuring that products entering the food chain are compliant with any applicable regulatory limits (Rose 2015). Total Diet studies (TDS) can provide initial exposure estimates for food constituents, such as contaminants, which act as a baseline for any future measures aimed at reducing exposure at the population level. TDS allow exposure time trends to be monitored and in some cases can be used to determine

the effectiveness of regulatory controls for different food types e.g. to assess the impact of pollution control measures on levels of PCDD/Fs in food. An overview of population and population subgroups' exposures to contaminants can be gained using TDS data in which samples of a wide variety of food and beverage types are selected from various retailers across the target area (EFSA et al. 2011).

Items are purchased, prepared as if for consumption and combined into groups of similar foods for analysis (EFSA et al. 2011, Rose 2015). The food group contaminant concentrations are combined with dietary consumption data to estimate exposure. There are limited historic examples of TDS across the globe, although the approach is gaining popularity. The long term use of TDS in the UK provides a valuable historic perspective.

Duplicate diet (DD) or duplicate portion studies are useful to provide realistic estimates of an individual's dietary intake over defined periods. Participants collect a duplicate of the food (and sometimes drink) that they consume throughout the defined period, providing a snapshot of their daily diet. The food collected is used to form a composite sample that can be used for analysis. A high degree of cooperation is required from participants. Although the overall composition of the samples will be known, duplicate diets do not attribute exposures to different food groups. Duplicate diet contents may be influenced by the individual's preferences during the period of collection and subject to anomalies arising where the participant consumes food that is not a regular part of their normal diet. Effects of local contamination and geology or food habits may be noticeable.

The aims of this study were: (i) to investigate dietary exposure to PCDD/F, PCB, PBDD/F, PBB and PBDE for a group of volunteers in the North East of England; (ii) to compare the resulting estimates with those made using the UK Food Standard Agency (FSA) TDS 2012 (Fernandes et al. 2012, Mortimer 2013) and (iii) to consider risk to human health as a result of the estimated dietary exposures.

Materials and Methods

Sample collection

Total Diet Study (TDS)

The TDS was carried out on foods that represent the average UK diet as estimated by the UK's Department of Environment, Food and Rural Affairs (Defra) Expenditure and Food Survey (2011) and trade statistics. Between 1 November 2011 and 31 March 2012, a total of 986 retail food samples were purchased from a range of national supermarkets (50%), symbol retailers (independent retailers that are members of a larger organisation e.g. Spar) (25%) and independent retailers (25%) in twelve locations around the UK. These samples were split into 20 representative food groups (see Table 1) and each food group analysed for a range of contaminants (Henderson et al. 2002). All food groups were analysed except for beverages, which have negligible fat content and therefore have low importance for lipophilic POPs. A wider range of samples was obtained for the animal product food groups, because these are more important sources of POPs in the diet (Fernandes et al. 2012). Table 1 shows the sample numbers for each group. Each individual sample was prepared as though for consumption, using a variety of methods of cooking where appropriate. Samples were homogenized, put into their respective food groups in relative quantities, as determined by national consumption data (PHE 2014), and thoroughly re-homogenized. Aliquots were freeze dried prior to analysis. For intake estimations, total consumption for each food group was derived from four day food diaries kept by approximately 500 adult participants (78% aged 19-64 years and 22% aged ≥ 65 years) in the Department of Health's National Diet and Nutrition Survey 2011-2012 (PHE 2014).

Duplicate Diet (DD) Study

24 hour DD samples were collected by 20 volunteers (10 males and 10 females, aged 26-43 years, weight range 62-101 kg) living in the North East of England as part of a wider in-depth study into potential human exposure sources and uptake of PBDE and emerging brominated contaminants from food and indoor dusts (Bramwell et al. 2014). The wider study matched serum and human milk samples with the 24 hour DD samples as well as samples of dust from the volunteers' indoor environments. Two of the volunteer couples subsequently repeated the study providing some validation for the method. The study aimed to recruit individuals with a range of diets to potentially reflect low, medium and high levels of exposure to PBDEs, by selecting participants who were oily fish eaters and vegetarians, and those with possible occupational exposure. A short pre-screening questionnaire identified volunteers who would provide a divergent range of exposures. One of the female participants was a vegetarian, one had a strong dairy intolerance, one was nursing an infant with a dairy intolerance, two participants ate mainly organic food, and one participant did not eat beef.

The DD samples were collected between 1 April 2011 and 28 February 2012. Whatever food was eaten by volunteers throughout the day, an equal amount was placed into a contaminant free (this was verified by tests carried out prior to sampling) lidded polypropylene container. Water and water based drinks were not included. For teas and coffees, the equivalent portion of milk was added. Samples were collected at the end of the day, homogenized immediately and stored frozen in chemically clean (dichloromethane rinsed) glass jars until analysis.

Volunteers gave written informed consent prior to participation. Ethical approval for the study was provided by the NHS National Research Ethics Committee North East, Durham and Tees Valley, the Newcastle upon Tyne Hospitals NHS Foundation Trust, Newcastle University's Research Ethics Committee and the Food and Environment Research Agency's Ethics Committee.

Laboratory Analysis

Laboratory analysis for both the DD and TDS samples was undertaken by the Food and Environment Research Agency (Fera), Sand Hutton, York, UK, and details of the methods used for sample preparation, extraction, clean up and analysis of PBDEs, PBBs and PBDD/Fs by high resolution gas chromatography - high resolution mass spectroscopy analysis are described elsewhere (Fernandes et al. 2008, Fernandes 2004). Methods for the analysis of PCDD/Fs and PCBs have also been previously reported (Fernandes 2004). The performance characteristics of the methodology, including quality assurance parameters such as limits of detection (LODs), precision, linear range of measurement, recoveries etc. are included in the previous reports (Fernandes et al. 2008, Fernandes 2004). Further confidence in the data is provided by regular and successful participation in laboratory proficiency testing and inter-comparison schemes such as POPs in Food 2011 and 2012 (Bruun Bremnes et al. 2012).

The following congeners were measured in both TDS and DD samples: the seventeen 2,3,7,8-Cl substituted PCDD/Fs; dioxin-like (i.e. non-ortho substituted and mono-ortho) PCBs with IUPAC (Favre and Powell 2013) numbers 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, and 189; non dioxin-like (i.e. ortho substituted) PCBs with IUPAC numbers 18, 28, 31, 47, 49, 51, 52, 99, 101, 128, 138, 153, 180; ten tetra- to hepta-, 2,3,7,8-Br substituted PBDD/Fs as well as 2,3,7-TriBDD, 2,3,8-TriBDF; dioxin-like PBBs with IUPAC numbers 77, 126 and 169; non-dioxin-like PBB-209 and PBDEs with IUPAC numbers 17, 28, 47, 49, 66, 71, 77, 85, 99, 100, 119, 126, 138, 153, 154, 183 and 209. The congeners selected for analysis are those for which reference standards are available. LODs for all measured analytes were estimated dynamically during the specific period of analysis and were dependent on parameters such as sample weight, type of matrix and instrument

performance at the time of measurement. Typical LODs were 0.01 to 0.05 ng kg^{-1} lipid for PCDD/Fs and non-ortho substituted PCBs; 10 ng kg^{-1} lipid for ortho-PCBs; 0.02 to 0.08 ng kg^{-1} lipid for PBDD/Fs; and 1 to 20 ng kg^{-1} lipid for PBDEs and PBBs.

Data treatment and statistics

Dietary exposure assessments for the TDS were carried out using the Intake 2 Programme, bespoke software developed for the FSA. Dietary exposures for average and high-level (97.5th percentile, P97.5) consumers were estimated from the distribution of calculated exposures across all participants. TDS findings for adult average and high-level consumers are used here for comparison with the DD study.

DD daily exposure estimates were calculated from the whole weight (ww) concentration of contaminants in an individual's diet sample multiplied by the mass of sample collected. Individuals' body weights were used to calculate their exposure on a body weight (bw) basis. Where participants repeated the study, only data from their first set of results were included in the statistical analysis. Data for the repeat 24 hour DD is included in the Supplementary Information Table 1. For comparison with the TDS exposure estimates the average and P97.5 are presented for DD exposure estimates, although the P97.5 is not robust for 20 individuals.

Where the analytes are PCDD/Fs or are known to show dioxin-like toxicity, i.e. PCDD/F, PBDD/F, non-ortho and mono-ortho substituted PCBs and PBBs, the PCDD/F like toxicity of the samples has been reported as toxic equivalence (TEQ) using toxic equivalency factors (TEFs) which express the toxicity of each compound relative to 2,3,7,8-TCDD (where 2,3,7,8-TCDD =1). The most recent, updated WHO 2005-TEQ (Van den Berg et al. 2006) as well as the WHO 1998-TEQ predecessors (Van den Berg et al. 1998) are both used here to allow for direct inter-study comparison. Although derived for PCDD/Fs and dioxin-like

compounds, the WHO 2005-TEQ are also used for their brominated analogues (Van den Berg et al. 2013). This is a commonly used (Fernandes et al. 2012, Pratt et al. 2013) interim measure until experimental TEF values for all of the brominated congeners that show dioxin-like toxicity become available (COT 2006). For monitoring and regulation of non-dioxin-like PCBs, the International Council for the Exploration of the Sea (ICES) selected six commonly measured 'indicator' non-dioxin-like (ortho) PCBs 28, 52, 101, 138, 153 and 180 (ICES-6 PCBs) (Webster et al. 2013) and the sum of these is presented here.

Lower bound (LB) results assume values at less than the limit of detection (<LOD) are zero whereas upper bound (UB) results assume values <LOD are equal to the LOD. Summary exposure estimates are presented as both LB and UB contaminant concentrations on a body weight basis. Improvements in measurement sensitivity have led to (i) an increase in LB estimates, (ii) a decrease in UB estimates based on lower limits of quantification and (iii) convergence of LB and UB estimates. EU analytical regulations for foodstuffs require the difference between UB and LB values to be less than 20% for confirmations of regulatory maximum exceedances (Commission Regulation 589/2014). Summary analyte concentrations discussed in the text use UB values, and are thus precautionary, 'worst case' estimates.

Findings are discussed for both lipid weight (lw) and whole weight (ww) contaminant concentrations. The laboratory results are presented as lw data so these values are relevant to the measured fat/lipid content of the sample. The measured fat/ lipid content is also provided for each sample for simple conversion to ww where required. Ww values reflect the sample as received whole or 'wet' and is the usual manner of expressing consumption and exposure data. Dietary exposure to POPs from the 'fish and seafood' group is monitored and regulated using ww measurements. Ww measurements provide a more realistic reflection of dietary exposure as the fish group contains many different species of both oily (high lipid content) and white fish (low lipid content). Liver ('offal' group) is also regulated using ww data (EEC

2013) as POPs in liver are also bound to proteins (Huwe 2012). In contrast, foods such as beef or lamb ('carcass meat' group) where different parts of the animal would contain different amounts of fat, and dairy items are monitored and regulated by their lw contaminant concentrations.

Human health risk characterisation

The sum of dietary exposure to PCDD/Fs and dioxin-like PCBs, PBDD/Fs, and dioxin-like PBBs from the TDS and DD was compared with the tolerable intake value of 2 pg WHO-TEQ kg^{-1} bw day^{-1} (COT 2001) as set by the UK Committee on Toxicology of Chemicals in Food, Consumer Products and the Environment (COT) and in line with current tolerable intakes derived by the WHO Food and Agriculture Organisation of the United Nations Joint Expert Committee on Food Additives (JEFCA 2001). It should be noted that the COT TDI was set based on PCDD/Fs and dioxin-like PCBs only and did not include PBDD/Fs, and dioxin-like PBBs. Health based guidance values are not available for non-dioxin-like PCBs and PBBs.

Potential health risks from dietary intake of PBDEs were determined using the margin of exposure (MOE) approach applied by the European Food Safety Authority (EFSA). The EFSA Panel on Contaminants in the food chain (EFSA 2011) identified effects on neurodevelopment as the critical endpoint. Chronic human intakes, associated with body burdens at the $BMDL_{10}$ for BDEs-47, -99, -153 and -209, were estimated to be 172, 4.2, 9.6 and 1,700,000 ng kg^{-1} bw day^{-1} respectively. Average and P97.5 human dietary intakes as estimated by the DD and TDS methods were compared with EFSA's chronic human daily dietary intake estimations to determine the MOEs. For PBDEs, EFSA consider that an MOE

above 2.5 indicates that a health concern is unlikely, with risk decreasing as the MOE increases (EFSA 2011).

Results

POPs concentrations in food samples

Detailed results from the 2012 TDS for PCDD/Fs, PBDD/Fs, PCBs, PBBs and PBDEs are provided in Fernandes et al. (2012) and summarised in Table 1. Concentrations for individual congeners in each DD sample, lipid content in each sample and food items making up each sample are presented in Supplementary Information Tables 1-3. Lipid content in the DD samples was median 5% range 2-13%. The DD samples with low lipid % had cups of tea added rather than just the milk. A summary of exposure estimations for the DD samples is presented in Table 2.

PCDD/F and PCB measurements

The TDS food group 'fish and seafood' demonstrated the highest lw levels of all PCDD/F and PCB groups and also the highest ww levels except for sum PCDD/F and sum non-dioxin-like PCBs where the 'offal' and 'fats and oils' groups respectively demonstrated the highest ww concentrations. Comparison of LB sum of PCDD/Fs, PBDD/Fs and dioxin-like compounds measured indicated that the chlorinated analogues were more abundant than the brominated analogues in the higher lipid content food groups containing meats, fish, dairy, eggs and oils (see Table 1). PCDD/Fs were measureable in all DD and TDS samples. The most abundant PCDD/F was OCDD although, due to the low TEF, this was not as important in terms of contribution to the TEQ. The most abundant non-dioxin-like PCB in the DD and TDS samples was CB-153. Of the four non-ortho substituted PCBs, CB-77 was the most

abundant in the DD samples and most of the TDS groups except those containing milk where PCB-126 was the most abundant. Concentration ranges (pg kg^{-1} ww) and detection rates for the ICES 6 indicator PCBs in the DD samples were CB-28: <LOD-7.27 (85%); CB-52: <LOD-16.28 (95%); CB-101: <LOD-23.36 (95%); CB-138: 4.98 – 29.03 (100%); CB-153: 4.89-31.15 (100%); and CB-180: 1.51-9.67 (100%).

PBDD/F and PBB measurements

LB sum PBDD/Fs concentrations in lower lipid content TDS food groups including 'bread', 'cereal', 'potatoes' and 'fresh fruit' were higher than concentrations in their chlorinated analogues (see Table 1). The PBDD/F analysis comprised only 12 congeners, including 2 tri-substituted PBDD/Fs, due to the availability of reference standards. Measuring fewer brominated than chlorinated congeners may influence the relative sum pg WHO-2005 TEQ kg^{-1} ww reported, though the PBDD/Fs measured were mainly those with the higher TEFs. The most abundant PBDD/F in the DD samples was 1,2,3,4,6,7,8-HeptabromoBDF, measured above the LOD in all but one of the DD samples (median 2,400, range 810 – 39,000 pg kg^{-1} lw; median 126, range 51-680 pg kg^{-1} ww). These concentrations were higher than those for OCDD, the most abundant PCDD/F in the DD samples. 1,2,3,4,7,8-HexaBDF was the next most abundant PBDD/F though at concentrations over 10 times less.

Non-ortho substituted PBBs -77, -126 and -169 and the deca BB-209 were the only PBB congeners measured in the DD samples. These were all below the LOD (average <2.7 pg kg^{-1} lw) except for BB-209, which was detected in only 15% of samples. Concentrations and detection rates for PBBs were low and measurable in a only a few of the TDS food groups. Ortho-PBBs -15, -49, -52, -80, -101 and -153 were analysed in the TDS samples only. The TDS food group 'fish and seafood' demonstrated some low but measureable concentrations.

BB-153 was identifiable in the ‘milk and dairy’, ‘poultry’, ‘meat products’ and ‘carcass meat’ groups. PBBs would not be expected to be found in UK diet samples as evidence indicates European environmental background levels to be low (EFSA 2010).

PBDE measurements

The food groups ‘sugar and preserves’, and then ‘fish and seafood’ demonstrated the highest lipid weight sum PBDE concentrations. An atypically high sum PBDE concentration in an individual sample in the composite ‘sugar and preserves’ group is the most likely explanation for the groups raised sum PBDE result. The raised PBDEs in the ‘sugar and preserves’ group BDEs 47, 99, 100 and 153 were quantified in all DD samples and BDE-209 in 90%. The highest TDS ww concentrations for BDEs-47, -153, and -99 and -209 were in the ‘fish and seafood’, ‘fats and oils’ and ‘sugar and preserves’ groups respectively.

Dietary exposure estimates for contaminants

TDS exposure estimates for the dioxin-like POP groups and individual PBDE congeners are summarised in Mortimer et al. (2013) and presented here in Table 1. A summary of daily adult dietary exposures estimated by the 24 hour DD method is provided in Table 2. Results for PBDD/Fs and PBDE congeners are included only where they were measured above the LOD in 50% or more of the samples. Dietary exposure estimates to PBB are not included in Table 2 due to their low detection rate in the DD samples (max 15% for PBB-209). DD participants had average body mass 77 and 80 kg for females and males respectively, with an average daily food intake of 1.12 kg. Individual participants’ body mass measurements and mass of individual DD samples are provided in the Supplementary Information Table 1. Details of the DD matched internal exposure/body burden data (serum

and human milk) are reported elsewhere (Bramwell et al. 2014) and matched dust data will be reported subsequently.

Adult dietary exposure estimates for average and high level (P97.5) consumers as determined by the TDS and DD studies are presented together in Table 3 for comparison. Ratios of average and P97.5 adult exposure estimates for TDS/DD are also provided in Table 3.

PCDD/F and PCB exposure estimates

Agreement between TDS and DD estimates are good when considering the DD group was much narrower than the adult range used to estimate for TDS. Neither method invalidates the other. The average adult dietary exposure to PCDD/Fs and dioxin-like compounds (PCDD/F/PCBs) was estimated to be 0.52 WHO-TEQ pg kg^{-1} bw d^{-1} when using data from the TDS and 0.27 WHO-TEQ pg kg^{-1} bw d^{-1} when using the DD data. The average adult dietary exposure to the non-dioxin-like ICES-6 PCBS was estimated to be 1.80 pg kg^{-1} bw d^{-1} by the TDS and 0.58 by the DD, the estimate derived from the TDS being over three times that derived from the DD.

PBDD/F and PBB exposure estimates

The average adult dietary exposure to PBDD/F and brominated dioxin-like compounds (PBDD/F/PBBs) was estimated to be 0.2 TEQ kg^{-1} bw d^{-1} by both the TDS and the DD. The P97.5 adult dietary exposure to PBDD/F/PBBs was estimated to be 0.51 TEQ pg kg^{-1} bw d^{-1} by the DD and 0.56 TEQ pg kg^{-1} bw d^{-1} by the TDS, these can be regarded as equal given the uncertainties involved. The maximum non-dioxin-like DD PBB-209 exposure determined was 180 pg kg^{-1} bw d^{-1}.

PBDE exposure estimates

The average adult dietary exposure to sum PBDE (for all congeners measured except BDE-209) was estimated to be 290 pg kg^{-1} bw d^{-1} using the data from the DD study and the P97.5 was estimated to be 650 pg kg^{-1} bw d^{-1}**.** BDE-209 was detected above the LOD in 90% of the DD samples, with average daily exposure estimated to be 750 pg kg^{-1} bw d^{-1} and over three times more when using data from the TDS study (2600 pg kg^{-1} bw d^{-1}). This difference probably reflects the large variation in PBDE concentrations in individual samples for the same food types. Where BDE-209 was detected in DD samples it made up a median of 73% of sum PBDE exposure. Excluding BDE-209, BDE 99 and BDE 47 accounted for just over a third of the total for all congeners measured, at 37% and 36% respectively, followed by BDE-153 (8%), BDE-100 (6%), and BDE-183 (4%). After BDE-209, BDE-47 exposure was found to be next greatest PBDE congener exposure by the TDS and BDE-99 by the DD. Average daily adult dietary exposure to BDE 47 was 92 pg kg^{-1} bw d^{-1} by DD and twice that by TDS at 200 pg kg^{-1} bw d^{-1}. Average daily adult dietary exposure to BDE-99 was 100 pg kg^{-1} bw d^{-1} by DD and 1.4 times that by TDS at 140 pg kg^{-1} bw d^{-1}. Health risk characterisation MOEs calculated for the DD and TDS exposure estimates are presented in Table 4 along with MOEs determined by EFSA (2011) summarising European dietary exposure for comparison.

Food groups having the greatest contribution to PCDD/F and PCB dietary intake such as ‘fish and seafood’, ‘meat’ and ‘milk and dairy’ generally had either no or low difference between UB and LB sum values, the greatest difference being 7% for poultry, well within the required 20% (Commission Regulation 589/2014). Food groups with lower PCDD/F and PCBs concentrations had more PCDD/F and PCB congener concentrations below the LOD and therefore greater difference between UB and LB sum values. The difference between UB and LB sum WHO 2005-TEQ concentrations of PCDD/F and PCBs in the different TDS food groups ranged from 0% to 73% with a median of 2% and average of 18%. The

differences between UB and LB sum WHO 2005-TEQ concentrations of PBDD/F and PBB ranged from 7% to 100% with median 36% and average 44%, consistent with the greater number of congener measurements below the LOD. UB and LB sum PBDE concentrations were calculated for the duplicate diet samples. Differences of 6% and 1% were observed using sum average PBDE concentrations and sum P97.5 PBDE concentrations respectively.

Discussion

Evaluation and comparison of methods

This 2011/12 study documents UK dietary exposure estimates for PCDD/Fs, PCBs, PBDD/Fs, PBBs and PBDEs and evaluates and compares the findings of two different methods of estimation. We provide estimates of adult dietary exposure for a range of UK and international TDS and DD studies to allow comparison between findings. TDS estimates were generally higher than the DD results for both average and P97.5 but the differences are not substantial considering that two very different approaches were used. With limited participant numbers and timeframes, DD studies measure a snapshot of individuals' exposures and are unlikely to have the range required to represent a general population. The small number of samples in this DD study also limited the statistical power.

For this study, estimates for individual PBDE congeners show good agreement between the TDS and DD studies, providing an element of validation for both methods e.g. combined PCDD/F and dioxin-like-PCB exposures compare well with dietary exposure estimates average 0.52 and 0.27 and high 1.10 and 0.88 pg kg^{-1} bw d^{-1} for TDS and DD respectively and a TDS /DD ratio of 1.2. Some of the difference may be accounted for by the limited number of DD participants and possibly their lower meat and dairy consumption compared to average UK diets represented by the TDS. In addition, there are known to be

behavioural changes for individuals involved with DD exercises (eat less, more health food) and these may also have an impact of reducing the DD exposure estimates (Rose 2015).

Individual BDEs -47,-99 and -153 had an average TDS /DD ratio range 1.4 – 2.2 and range 1.0 – 2.0 for P97.5. ICES-6 were higher for the TDS with ratios 3.2 (average) and 2.8 (P97.5). Variation between exposure estimates for BDE-209 (TDS/DD ratio average 3.4, P97.5 2.8) may be influenced by the high TDS result for the 'sugars and preserves' food group, accounting for 50% of total exposure, and 'milk', accounting for 25% of exposure (Mortimer 2013). The 2012 'sugar and preserves' BDE-209 concentration (2.00 μg/kg ww) was notably higher than that for 2003 (0.39 μg/kg ww). This may be due to the inclusion of a highly-contaminated sample within the composite. BDE-209 usage has been particularly high in the UK and contamination of a sample during transport or processing cannot be excluded. With 'sugar and preserves' and 'milk' results excluded the exposure estimates for the TDS and DD are close (Mortimer 2013). Where numbers of samples making the food group composite for TDS are low, distortion of results may occur where one or more samples contained atypically high contamination.

While the relative abundance of some individual PBDEs varied between diet types, e.g. BDE-47,-49, -100 and -153 were higher in the DDs containing fish, BDE-209 concentrations were consistent across DD types (lactose free/ vegetarian/ omnivore/ high meat/ high fish). We hypothesise that this indicates BDE-209 contamination may be getting into the food subsequent to the primary production stage when most contamination is assumed to occur, *e.g.* from food packaging, processing/preparation, contamination with airborne dust particles or dust via dermal contact.

Temporal trends

Concentrations of PCDD/F, PCB and PBB in our food supply have declined over the last decade (EFSA 2010, EFSA 2012). The reduction in dietary exposures to PCDD/F and PCB is

illustrated in Figure 1 with data from this study and other TDS and DD studies from across Europe. Exponential downward curves can be seen from 1982 to 2012 for both average and high consumers. It should be noted that sensitivity of analytical methods has improved over the time period depicted, allowing more congeners to be positively determined. These changes may affect comparability when assessing temporal trends. In 2011/12 the estimated high level exposure WHO 2005 TEQ total kg^{-1} bw day^{-1} for total PCDD/F and PBDD/F and dioxin-like compounds was estimated to be 1.44 and 1.59 by DD and TDS respectively. In 2001 and 1982 only PCDD/F and dioxin-like PCBS were measured so a direct comparison is not possible, but decreases are nonetheless apparent: 0.11- 0.33 WHO 2005 TEQ total kg^{-1} bw day^{-1} since 2001 and 11 WHO 2005 TEQ total kg^{-1} bw day^{-1} since 1982. When compared with UK levels reported in food groups from 2003 (FSA 2006), the LB results have generally increased whilst the UB levels have generally decreased, although the changes are relatively small in absolute terms. This is again likely to reflect improvements in analytical sensitivity rather than a temporal effect. .

Data in Table 3 indicate peaks in dietary exposure to BDEs-47 and -99 between 2000 and 2005. BDE-153 has also reduced but not quite as quickly, in keeping with its longer half-life in the environment. BDE-209 exposure may still be increasing, but usage was not phased out at the same time as the lower-substituted BDEs and was particularly high in the UK.

No temporal influence on exposure estimates would be expected to be measurable between the DD and TDS samples as they were collected in 2011 and 2012 respectively. Comparison of two DD carried out in near identical conditions at different periods would be required to investigate such effects.

Risk characterisation

PCDD/F/PCBs and PBDD/F/PBBs

Estimated dietary exposure to PCDD/Fs, PBDD/Fs and dioxin-like compounds for both TDS and DD sample sets for this study, calculated on an UB basis, were within current international recommended tolerable intake values for PCDD/F with dioxin-like PCBs (COT 2001, JEFCA 2001). The DD samples indicated an UB average dietary intake of 0.47 and P97.5 of 1.4 pg WHO 2005 TEQ kg^{-1} bw d^{-1} for PCDD/F/PCB and PBDD/F/PBB. The TDS UB intake estimates indicated an average of 0.77 and P97.5 of 1.6 pg WHO 2005 TEQ kg^{-1} bw d^{-1}. A tolerable weekly intake of 14 pg WHO-TEQ kg^{-1} bw was derived in 2001 by the Scientific Committee on Food (SCF) and a provisional monthly intake of 70 pg kg^{-1} bw was derived by JEFCA (JEFCA 2001). In November 2001, the UK Committee on Toxicology of Chemicals in Food, Consumer Products and the Environment recommended that the UK tolerable daily intake for mixtures of PCDD/Fs and dioxin-like PCBs be reduced from 10 pg WHO-TEQ kg^{-1} bw day^{-1} to 2 pg WHO-TEQ kg^{-1} bw day^{-1} (COT 2001).

Non dioxin-like PCBs and PBBs

For non-dioxin-like PCBs, EFSA were unable to derive any health-based guidance values (EFSA 2005). Their recommendation was that dietary exposure should be reduced and data from projects such as this provide a means to determine whether this is being achieved.

To determine the potential for health effects from dietary exposure to sum ortho-PBBs, EFSA use a worst case no-observed-effect level (NOEL) of 0.15 mg kg^{-1} bw for hepatocarcinogenesis in rats (EFSA 2011). This is six orders of magnitude above the maximum sum ortho PBB exposure determined by the DD study indicating no health concerns. BB-77 was the only non-ortho PBB detected above LOD in DDs for this study (20% detection rate).

PBDEs

No health concerns are expected from the levels of PBDEs measured in these adult DD and TDS studies as all had MOEs over 2.5 (EFSA 2011). BDE-99 exposures demonstrated the lowest MOEs; 16 and 17 for high UB dietary intake for DD and TDS. EFSA derived an MOE of 3.9 for adults for BDE-99 when reviewing the EU evidence (EFSA 2011). BDE-209 demonstrated the greatest MOE for dietary exposure, 2,260,000 and 664,000 for average UB dietary intake by DD and TDS respectively. These reported MOEs are for adults only, EFSA noted concern about exposure of young children (age 1-3 years) for whom EFSA derived MOEs of 1.4 and 0.7 for dietary exposure to BDE-99 for average and high consumption respectively (EFSA 2011). It should be also be observed that PBDE intake is not exclusively from the diet and inhalation of PBDEs in indoor dust and air, most notably for BDE-209, will add to total human exposure (Bramwell et al. 2016, EFSA 2011). For adults ingesting 50 mg dust per day this additional BDE-209 source is estimated to be in the range 0.045 to 7 ng kg^{-1} bw day^{-1} (EFSA 2011, Fromme et al. 2009). Dust intake is greater for young children and their additional BDE-209 intake from dust estimated to be 0.5-80 ng kg^{-1} bw day^{-1} (Bramwell et al. 2016, EFSA 2011). Both the UK DD and TDS MOEs are well within the UB MOEs determined by EFSA in their review of EU evidence of dietary PBDE exposure (EFSA 2011).

Conclusions

TDS and DD estimations for all measured compounds were found to be within recommended tolerable daily intake values where available or within acceptable margins of exposure. To the authors' knowledge, this study is the first to compare DD and TDS techniques for measuring human dietary exposure to PCDD/Fs, PCBs, PBDD/Fs, PBBs and PBDEs. TDS provide a versatile data set which can be used to estimate dietary exposure for a range of consumers. DD studies give distinct estimates of participating individual's exposures, taking into account local food sources such as farms, fish or wild food. DD are particularly useful

for interpreting associations with internal POPs exposure measurements such as serum or human milk concentration. DD studies are difficult to run on a large scale or over a prolonged period of time with issues of cost to individuals and management of sample collection and storage, and may not reflect an individual's long term exposure. The TDS data provided information on the relative levels of contamination in different food groups. When used with food consumption information, the TDS can be used to provide dietary exposure estimates for a range of age groups and eating behaviours making it a more versatile data set. This is particularly useful for establishing baseline levels of population exposure to new contaminants or monitoring temporal changes. By comparing estimates using the two contrasting approaches, both receive an element of cross-validation. There is no doubt that the DD method is suitable for estimating an individual's dietary intake for the period of the diet collection. It is reassuring to know that the UK National estimate can reasonably reflect individuals' dietary exposure.

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TDS group	Matrix	No. of sub-samples	Fat content %	PCDD/F/PCBs (pg WHO 2005 TEQ kg^{-1} ww)		PBDD/F/PBBs (pg WHO 2005 TEQ kg^{-1} ww)		BDE-47 (pg kg^{-1} ww)	BDE-99 (pg kg^{-1} ww)	BDE-153 (pg kg^{-1} ww)	BDE-209 (pg kg^{-1} ww)
				LB	UB	LB	UB				
1	Bread	29	4.14	7.0	11.5	8.2	20.7	5	6	2	<200
2	Cereals	40	9.42	5.0	12.6	23.1	34.4	6	8	2	<190
3	Carcass Meat	51	14.41	76.7	76.9	29.8	37.0	18	22	7	<130
4	Offal	85	9.92	191	191	42.0	45.9	7	9	3	<120
5	Meat Products	123	14.86	29.9	30.2	12.0	16.0	18	19	4	<140
6	Poultry	51	7.32	10.0	10.8	3.0	9.1	5	6	1	220
7	Fish & Seafood	140	9.31	326	326	10.5	16.4	134	23	7	170
8	Fats & Oils*	84	73.8	70.8	91.5	0.0	79.0	37	35	8	<390
9	Eggs	34	9.55	43.9	44.2	8.4	16.8	13	16	5	90
10	Sugar & Preserves	30	6.05	55.5	55.6	94.9	102	121	62	7	1950
11	Green Vegetables	23	0.29	4.5	4.6	3.6	6.0	2	1	0.2	50
12	Potatoes	23	5.19	8.1	9.7	9.1	12.6	5	5	1	50
13	Other Vegetables	40	5.46	52.6	52.7	4.6	10.1	5	8	1	50
14	Canned Vegetables	15	0.53	1.0	2.1	0.6	3.4	1	0	<1	20
15	Fresh Fruit	23	0.21	1.4	3.2	4.0	7.3	1	1	0.2	140
16	Fruit Products	15	0.42	6.3	7.5	12.2	16.9	1	1	0.4	30
18	Milk	44	1.97	8.2	8.3	3.5	5.1	2	2	0.5	120
19	Milk & Dairy Products	102	23.31	105	105	21.7	28.2	23	25	6	20
20	Nuts	34	41.84	5.0	18.8	3.3	34.7	6	5	1	100

*from animal and vegetable origin

Table 1. Total Diet Study (TDS) 2012: Food group compositions, PCDD/F/PCB and PBDD/F/PBB levels and PBDE congener concentrations (Mortimer 2013) LB= lower bound data, UB=upper bound data

	%	Min.		Max.	Median		Mean		P97.5	
	>LOD	LB	UB		LB	UB	LB	UB	LB	UB
WHO-TEQ Summary										
WHO 1998 TEQ pg kg^{-1} bw day^{-1}										
PCDD/F	-	0.028	0.036	0.844	0.129	0.141	0.168	0.177	0.595	0.606
non ortho-PCB	-	0.026	0.028	0.456	0.070	0.069	0.102	0.102	0.329	0.33
ortho-PCB	-	0.007	0.011	0.084	0.019	0.026	0.021	0.03	0.057	0.071
WHO 2005 TEQ pg kg^{-1} bw day^{-1}										
PCDD/F	-	0.023	0.036	0.76	0.110	0.122	0.147	0.154	0.530	0.537
non ortho-PCB	-	0.027	0.028	0.456	0.074	0.075	0.106	0.106	0.342	0.344
ortho-PCB	-	0.007	0.011	0.084	0.019	0.026	0.021	0.03	0.057	0.071
PBDD/F	-	0.004	0.007	0.615	0.060	0.141	0.121	0.199	0.477	0.558
non ortho-PBB	-	$<4x10^{-4}$	0.001	0.002	$<7x10^{-4}$	0.001	$1x10^{-6}$	0.001	$7x10^{-6}$	0.002
Sum of 2005 TEQs	-	0.061	0.083	1.92	0.263	0.265	0.395	0.49	1.41	1.51
PBDD/F Results pg kg^{-1} bw day^{-1}										
238-TriBDF	75	<0.013	0.013	0.129	0.037	0.042	0.045	0.057	0.128	0.128
2378-TetraBDF	75	<0.009	0.009	0.144	0.034	0.039	0.042	0.05	0.125	0.125
23478-PentaBDF	65	<0.017	0.017	0.339	0.078	0.078	0.087	0.105	0.3	0.3
1234678-HeptabromoBDF	95	ND	0.746	14.2	1.58	1.93	3.14	3.31	12.1	12.0
NDL PCBs pg kg^{-1} bw day^{-1} ICES-6[a]	-	0.1	0.137	2.01	0.41	0.41	0.578	0.583	1.78	1.78
PBDE Results pg kg^{-1} bw day^{-1}										
BDE-28	75	<0.001	1.42	21.2	2.63	4.26	4.84	5.76	17.0	17.0
BDE-47	100	23.4	23.4	208	73.4	73.4	91.9	91.9	204	204
BDE-49	60	<0.002	1.85	53.7	2.75	5.1	7.3	9.02	42.9	42.9
BDE-66	70	0	1.23	38.9	4.31	5.67	6.57	7.83	30.5	30.5
BDE-99	100	24.2	24.2	274	73.1	73.1	99.6	99.6	263	263
BDE-100	100	4.33	4.33	48.6	13.6	13.6	19.2	19.2	46.2	46.2
BDE153	100	5.35	5.35	57.5	14.1	14.1	19.9	19.9	53.4	53.4
BDE 154	90	<0.002	1.77	36.9	9.18	9.18	11.2	11.2	32.2	32.2
BDE-183	95	<0.002	1.92	59.8	10.9	12.0	11.3	14.3	25.6	44.2
Deca Results pg kg^{-1} bw day^{-1}										
BDE-209	90	<0.059	58.6	1850	596	652	708	751	1770	1770
BB-209	15	<0.018	17.7	185	<0.037	37	16.7	50.4	148	148
PBDE Summary pg kg^{-1} bw day^{-1}										
∑PBDEs[a]	-	154	226	2320	774	966	982	1040	2290	2310
∑PBDE(except 209)[b]	-	63.3	82.5	677	226	247	274	292	635	646
$\sum PBDE_{(6)}$[c]	-	63.3	66.8	590	218	218	253	256	574	574
$\sum PBDE_{(3)}$[d]	-	54.5	54.5	514	182	182	211	211	494	494

LOD= limit of detection, NDL = non-dioxin like, LB= lower bound data, UB=upper bound data, [a] Sum of all PBDE congeners measured, [b] Sum of all PBDE congeners measured except BDE-209, [c] Sum of BDE-47, -99, -100, -153, -154 and -183 [d] Sum of BDE-47, -99 and -153, P97.5 – 97.5th percentile

Table 2: Daily adult dietary exposure to PCDD/Fs, PCBs, PBDD/Fs, PBBs and PBDEs as determined by 24 hour duplicate diet (DD)

	Sampling year and study type	Statistical summary information		BDE-47 (pg kg^{-1} bw d^{-1})	BDE-99 (pg kg^{-1} bw d^{-1})	BDE-153 (pg kg^{-1} bw d^{-1})	BDE-209 (pg kg^{-1} bw d^{-1})	∑PBDE (except 209) (pg kg^{-1} bw d^{-1})	∑PBDE (pg kg^{-1} bw d^{-1})	PBDD/F/PBB (TEQ pg kg^{-1} bw d^{-1})	PCDD/F/PCB (TEQ pg kg^{-1} bw d^{-1})	ICES 6 (NDL PCBs)(pg kg^{-1} bw d^{-1})
UK This study	2011 DD (n=20, 24 hr)	Avg.	lower	92	100	20	708	274	982	0.2	0.27	0.58
	2012 TDS	Avg.	upper	200	140	30	2560	-	-	0.2	0.52	1.84
	2011 DD (n=20, 24 hr)	Avg.	upper	92	100	20	751	292	1040	0.2	0.27	0.58
	TDS/DD	Avg.	upper	2.2	1.4	1.5	3.4	-	-	1.0	1.9	3.2
	2011 DD (n=20, 24 hr)	P97.5	lower	204	263	53	1770	634	2290	0.56	0.88	1.77
	2012 TDS	P97.5	upper	410	250	60	5030	-	-	0.51	1.08	4.88
	2011 DD (n=20, 24 hr)	P97.5	upper	204	263	53	1770	646	2310	0.56	0.88	1.77
	TDS/DD	P97.5	upper	2.0	1.0	1.1	2.8	-	-	0.9	1.2	2.8
UK (FSA 2006)	2003/4 TDS	Avg.	lower	500	500	100	4500	-	5800	0.1	-	-
		Avg.	upper	500	500	100	4500	-	5900	0.4	-	-
		High	lower	1000	800	200	13000	-	15000	0.2	-	-
		High	upper	1000	800	200	13000	-	15000	0.8	-	-
UK (Harrad et al. 2003, 2004)	1999/2000 DD (n=10o, 5v, 14 days) 2005 DD (n=50, 7 days)	Avg. o	lower	651	683	45	-	-	-	-	-	-
		Avg. o	upper	651	694	178	-	2200	-	-	0.73*	-
		Avg. v	lower	-	-	-	-	-	-	-	1.09*	-
		Avg. v	upper	-	-	-	-	-	-	-	0.14*	-
		Max. o	upper	1150	2150	186	-	-	-	-	0.53*	-
		Max. v	upper	-	-	-	-	-	-	-	2.22*	-
		Median	medium	161	255	51	-	-	-	-	0.96*	-
Germany (Fromme et al. 2009)	2005 DD (n=50, 7 days) 2004/5 TDS	P95	medium	340	501	140	-	-	-	-	-	-
		Avg.	lower	-	-	-	-	1100	-	0	-	-
Japan (Ashizuka et al. 2007)	2004/5 TDS 2003/4 TDS	Avg.	medium	-	-	-	-	-	-	1.58	-	-
		Median	upper	400	110	-	-	-		-	-	-
Holland (Winter Sorkina et al. 2006)	2003/4 TDS 2004 DD (n=35, 24 hrs)	P97.5	upper	1100	210	-	-	-	-	-	-	-
		Avg.	n/a	770	500	-	480	-	-	-	-	-
Holland (Zeilmaker et al.2008)	2004 DD (n=35, 24 hrs)	Max.		3500	2300	-	3300	-	-	-	-	-
	1994 DD (n=10,24 hrs)	Avg.		140	610	-	-	-	-	-	-	-
	1984 DD (n=10,24 hrs)	Avg.		80	300	-	-	-	-	-	-	-
	1978 DD (n=10,24 hrs)	Avg.	n/a	570	120	-	-	-	-	-	-	-

NDL = non-dioxin like, PCDD/F, PBDD/F and dioxin-like concentrations are presented in WHO 2005 TEQ equivalencies unless indicated otherwise. *WHO 1998 TEQ (using 1998 TEFs results tend to be about 10% higher). o - omnivore, v - vegan diet

Table 3. Adult dietary exposure to PBDEs, PBDD/F/PBBs,PCCD/F/PCBs and ICES 6 PCBs for this and previous studies.

	BDE-47			BDE-99			BDE-153			BDE-209		
	DD	**TDS**	**EFSA**	**DD**	**TDS**	**EFSA**	**DD**	**TDS**	**EFSA**	**DD**	**TDS**	**EFSA**
MOE for Average LB dietary intake	1,870	-	593	42	-	38	482	-	320	2,400,000	-	-
MOE for Average UB dietary intake	**1,870**	**860**	**90**	**42**	**30**	**6.5**	**482**	**320**	**23**	**2,260,000**	**664,000**	**>97,000****
MOE for high LB dietary intake	844	-	156	16	-	14	180	-	137	960,000	-	-
MOE for high UB DD dietary intake	**844**	**420**	**38**	**16**	**17**	**3.9**	**180**	**160**	**14**	**960,000**	**338,00**	**>97,000****
EFSA estimated intake at $BMDL_{10}$ ($ng\ kg^{-1}\ bw\ day^{-1}$)	172			4.2			9.6			1,700,000		

* EFSA data is P95, ** EFSA determined MOE of 97,000 was for children (age 1-3) which are considered the most sensitive receptor, and did not determine the adult MOE for BDE 209. The adult MOE for BDE 209 can be expected to be greater than that for children.

Table 4. Comparison of margins of exposure (MOEs) for PBDEs as determined by the DD and TDS methods and European summary MOEs as determined by the EFSA review of EU evidence (2011)

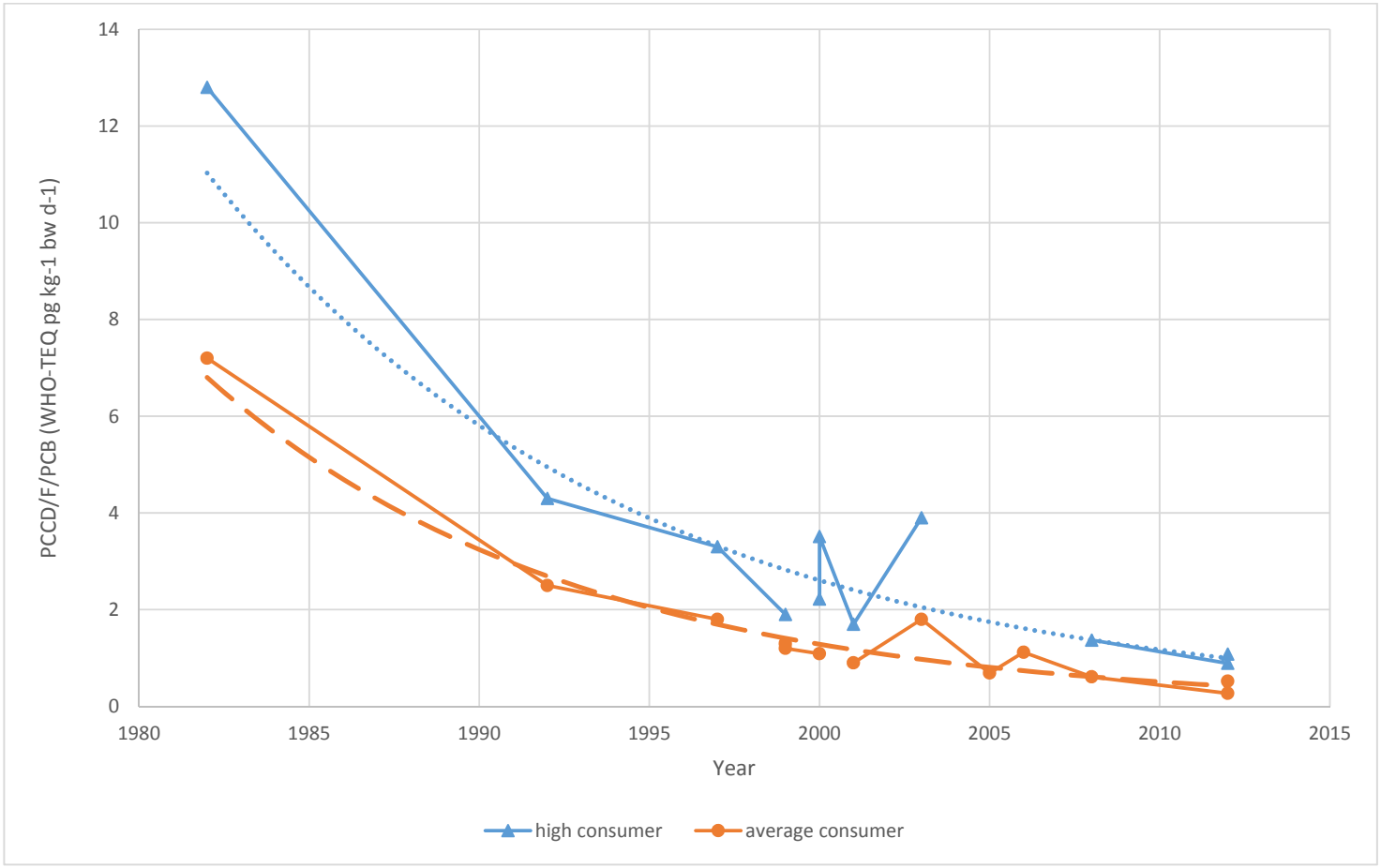

Figure 1. Decrease in adult high (95 and 97.5 percentiles) and average dietary PCCD/F/PCB exposure in Europe from 1982 to 2012. Data taken from UK TDS and DD 2011/12 (this study), UK TDS 1982, 1992 and 1997 (FSA 2003), Netherlands TDS 1999 (Baars et al. 2004), Sweden TDS 1999 and 2005 (Ankarberg et al. 2007), UK DD 1999/2000 (Harrad et al. 2003), Spain TDS 2000 and 2006 (Llobet et al. 2008), France TDS 2001-4 (Tard et al. 2006), Belgium 2008 (Windal et al. 2010). Exponential curves are fitted to the data.