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Estimated intakes of brominated flame retardants via diet and dust compared to internal concentrations in a Swedish mother–toddler cohort

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ABSTRACT

Tri-decabrominated diphenyl ethers (tri-decaBDEs), isomer-specific hexabromocyclododecanes (HBCDs) and 14 emerging brominated flame retardants (EBFRs) were determined in Swedish market basket samples, two pooled breast milk samples and house dust collected in homes of first-time mothers. Daily dietary and dust intakes were estimated for the mothers and their toddlers and compared to previously reported levels in serum of both the mothers and toddlers and in feces of the toddlers ($n = 20$). Diet was the main contributor for intake of ΣpentaBDE and α-tetrabromoethylcyclohexane (DBE-DBCH) for both mothers and toddlers. For ΣoctaBDE, ΣHBCD and pentabromobenzene (PBBz), dietary intake was more important for mothers while house dust ingestion was more important for toddlers. House dust was the main exposure route for ΣdecaBDE, decabromodiphenyl ethane (DBDPE), 2-ethylhexyl-2,3,4,5-tetrabromobenzoate (EH-TBB), bis(2-ethylhexyl)tetrabromophthalate (BEH-TEBP), bis(2,4,6-tribromophenoxy) ethane (BTBPE) and pentabromotoluene (PBT) for both mothers and toddlers. Significant correlations (Spearmans, $\alpha < 0.05$) were found between the mothers' BDE serum concentrations and their consumption of meat and fish while no correlations were found between BFR dietary intake and serum or feces concentrations in toddlers. Octa-decaBDE congener concentrations in serum and feces of toddlers were significantly correlated to those in house dust. BDE-207 and -208 concentrations in serum of mothers were significantly correlated with the nonaBDEs in house dust. The correlations between house dust and internal concentrations and comparison of the house dust and dietary contributions to the estimated daily intakes suggest that dust exposure plays a larger role for the octa-decaBDE body burden in toddlers than in their mothers.

Introduction

Brominated flame retardants (BFRs) are chemicals that are used in different materials such as textiles, carpets, plastics in electronic housings and building insulation materials to reduce flammability of the products. Through the manufacture and use of these flame-retarded products, BFRs have become ubiquitous in the environment, and humans and wildlife are exposed to these chemicals everyday (de Wit, 2002). Technical mixtures of polybrominated diphenyl ethers (PBDEs), a group of BFRs widely used since the 1970s, have recently been banned (Penta- and OctaBDE) (UNEP, 2009) or are being phased out (DecaBDE) (USEPA, 2009) in different parts of the world. Hexabromocyclododecane (HBCD), a BFR mainly used in polystyrene insulation materials is currently being phased-out (UNEP, 2013). After these actions, levels of banned chemicals have started to decrease in the environment (de Wit et al., 2010; Law et al., 2014). Decreased levels have also been reported in human breast milk for most PBDE congeners, at least in Sweden (Lignell et al., 2009; Fängström et al., 2008). However, other brominated chemicals have been introduced onto the market to replace the restricted flame retardants. These emerging BFRs (EBFRs) have already been detected in both abiotic and biotic environments (Covaci et al., 2011).

Humans are exposed to BFRs via several different routes such as dietary intake and dust ingestion (Daso et al., 2010). Previous studies comparing dietary and dust exposure/intakes with human measures of body burden such as breast milk (Wu et al., 2007) and

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blood (Roosens et al., 2009) suggest that both exposure routes are important contributers to human body burdens of BFRs. However, other studies have found no associations between dust exposure and BFR concentrations in breast milk or serum (Toms et al., 2009a; Fromme et al., 2009).

Exposure to PBDEs and HBCD has been shown to disturb thyroid hormone homeostasis in animals (Darnerud, 2008) and prenatal and childhood exposure to PBDEs have been associated with effects on normal development of the brain and the nervous system (Eskenazi et al., 2013; Herbstman et al., 2010; Kim et al., 2014). A recent study on rats showed indications of endocrine disruptive effects caused by a flame retardant mixture (Firemaster 550) containing two EBFRs, 2-ethylhexyl-2,3,4,5-tetrabromobenzoate (EH-TBB) and bis(2-ethylhexyl) tetrabromophthalate (BEH-TEBP) (Patisaul et al., 2013). Very limited toxicological information exists on EBFRs (EFSA, 2012).

Higher serum BFR levels have been reported in children compared to adults (Ali et al., 2013; Fischer et al., 2006; Sahlström et al., 2014; Toms et al., 2009b). We recently suggested that accumulation of lower brominated BDEs from breast milk and exposure to octa-decaBDEs via house dust ingestion could explain the higher concentrations of these found in serum of toddlers compared to serum of their mothers (Sahlström et al., 2014). Several studies have suggested dust ingestion as a more important exposure route for PBDEs in toddlers compared to adults (de Wit et al., 2012; Fischer et al., 2006; Stapleton et al., 2008). Stapleton et al. (2012) found correlations between the sum of tetra-hexaBDE levels in house dust and serum of toddlers, and Wu et al. (2007) reported that levels of tetra-hexaBDEs in breast milk correlated with dust concentrations and that breast milk levels of tri-decaBDEs correlated with dietary habits. However, there are no published studies of associations between external exposure and internal concentrations of BFRs in both adults and children within the same household. The contributions from both diet and dust for intakes of HBCD and sum of tri-hexaBDEs (de Wit et al., 2012) and tetra-heptaBDEs (Fromme et al., 2009) have been estimated previously but these studies did not include octa-decaBDEs in the diet.

The aims of this study were to (1) determine the concentrations of tri-decaBDEs, isomer-specific HBCDs and EBFRs in the two most important external exposure media, diet and house dust for a mother–toddler cohort, and (2) compare internal and external exposure by studying associations between previously published concentrations in matched serum (mothers and toddlers) (Sahlström et al., 2014) and feces samples (toddlers only) (Sahlström et al., 2015) with those in the dust and diet samples. The 14 EBFRs included in this study are listed in Table 1; full names with abbreviations are listed in Table S1 in the Supplementary data. This study reports the first results of isomer-specific HBCD concentrations in Swedish market basket samples and presents estimates of both dietary and dust intakes of isomer-specific HBCDs and several EBFRs for mothers and their toddlers. It also reports the first results on contributions from both diet and dust for octa-decaBDEs and the associations between internal and external exposure to individual PBDEs in both adults and toddlers within the same households.

Methods

Sampling

Study participants were recruited among 60 first-time mothers who delivered one healthy child in Uppsala University Hospital in 2009–2010 and were included in the POPUP study (Persistent Organic Pollutants in Uppsala Primiparas) (Lignell et al., 2009) soon after delivery. When the children were about 11 months old, the mothers were re-contacted and asked to participate in a follow-up study. For those who agreed, blood samples from the mothers ($n = 24$), blood ($n = 24$) and feces samples ($n = 22$) from their toddlers and house dust samples from each home ($n = 27$) were collected on the same visit in the participants' homes. The data resulted in 20 matched sets of serum (both mother and toddler), feces (toddler) and house dust. The mothers filled in a questionnaire on individual dietary habits and their informed consent was obtained for their toddlers to participate in the study. Information about the age of the study participants, weight of the toddlers, body mass index, education level (Table S2) and dietary habits of the mothers (Table S3) are given in the Supplementary data. This study was approved by the Regional Ethics Committee in Uppsala, Sweden (Permit 2004:M-177).

The sampling and analyses of the serum and feces samples have been described in detail in previous studies (Sahlström et al., 2014, 2015). The dust samples were collected on surfaces at least 1 m above the floor in the living room, kitchen, bedroom and/or hallway, using cellulose filters in styrene-acrylonitrile holders (Krim. Teknisk Materiel AB, Bålsta, Sweden) installed in the nozzle of a vacuum cleaner. Two pooled breast milk samples were created from individual samples collected from the participants in the POPUP study from the years 2009 ($n = 30$, of which 13 donated blood in the current study) and 2010 ($n = 30$, of which 11 donated blood in the current study) (Lignell et al., 2009). Individual milk samples from the participating mothers were not available for this study.

Market basket samples comprising five different food categories (fish, meat, vegetable oils, dairy products and eggs, $n = 4$ homogenates for each food category) collected in 2010 were obtained from the Swedish National Food Agency. No other food categories were included as it has been shown that food of animal origin is the main contributor to the intake of PBDEs (Domingo et al., 2008). The sampling of the market basket samples has been described elsewhere (NFA, 2012). In short, standard food items based on per capita consumption statistics were purchased from four different stores in Uppsala in 2010. For each store, the items were divided into specific food categories (meat, dairy, eggs etc.), per capita consumption amounts were sampled from each dietary item (e.g. different types of meat) within the food category and pooled, and the total sample homogenized. Aliquots of each homogenate were then prepared and stored at −20 °C. The sampling was organized by the Swedish National Food Agency in Uppsala, Sweden.

Extraction and clean-up

Breast milk and dietary items

A 1.6–11 g homogenized food sample or breast milk was weighed into a glass test tube, 25 ng each of the ^{13}C-labeled surrogate standards α-, β-, γ-HBCD, BDE-155, BDE-197, and BDE-209 (50 ng), as well as the $^{13}C_6{}^2H_{17}$-labeled EH-TBB and BEH-TEBP (50 ng) were added and the samples were extracted according to Jensen et al. (2003); solvents used are listed in Table S4, Supplementary Data. The lipid content was determined gravimetrically. The sample extracts were cleaned-up by treatment with concentrated sulfuric acid (H_2SO_4) and the sample fractionated into three fractions on a SiO_2 SPE column according to Sahlström et al. (2012). In short, PBDEs and most of the EBFRs were eluted in Fr I with 30 mL *n*-Hx, the rest of the EBFRs (EH-TBB, BTBPE and BEH-TEBP) were eluted in Fr II with 10 mL 5% diethyl ether (DEE) in *n*-Hx, and HBCDs were eluted with 10 mL 50% DEE in *n*-Hx in Fr III. Prior to the instrumental analyses the fractions were evaporated by a gentle stream of nitrogen and transferred to appropriate vials containing a recovery standard (^{13}C-CB-180, 1050 pg for fractions I and II, and d_{18}-β-HBCD, 100 pg for fraction III) and the final volumes adjusted to 50 μL.