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Brominated flame retardant concentrations in sera from the Canadian Health Measures Survey (CHMS) from 2007 to 2009☆

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ABSTRACT

Pooling of surplus serum from individual samples, collected between 2007 and 2009 during Cycle 1 of the Canadian Health Measures Survey (CHMS), was performed to develop a national baseline estimate of brominated flame retardants in Canadians. Serum samples were categorized by sex and distributed by five age groups ranging from 6 to 79 years. Nearly 5000 (4583) serum samples were used to form 59 composite pools. Serum pools were created to ensure a high detection frequency of these analytes in serum because low volume samples had previously resulted in non-detectable concentrations. The analytes of interest in these serum pools included 23 polybrominated diphenyl ethers (PBDEs) and three hexabromocyclododecane (HBCD) isomers (α-, β- and γ-HBCD). PBDEs were observed in all samples tested and total PBDE concentrations ranged from 27 ng g^{-1} lipid to 130 ng g^{-1} lipid (geometric mean [GM] 46 ng g^{-1} lipid). ∑PBDE concentrations were significantly elevated in samples representing the 6–11 year old age group (GM 65 ng g^{-1} lipid) relative to ages above 40 years, although no difference in concentration was observed between the sexes. PBDE concentrations in Canadian sera from the general population were higher than reported in Europe and Asia, but a little lower than observed in the US. PBDE 47 was the greatest contributor to ∑PBDE concentrations and the GM concentration for this congener was 22 ng g^{-1} lipid. The other dominant contributors to ∑PBDE concentrations were in descending order: 153 [GM 9.4 ng g^{-1} lipid] > 99 [GM 4.6 ng g^{-1} lipid] ≅ 100 [GM 4.1 ng g^{-1} lipid] > 209 [GM 1.1 ng g^{-1} lipid] and 183 [GM 0.42 ng g^{-1} lipid]. ∑HBCD was detected in all samples analysed, although most samples were observed at concentrations < 1 ng g^{-1} lipid, similar to global concentrations. α-HBCD was the dominant contributor to ∑HBCD concentrations in Canadians although β- and γ-HBCD were detected in 23% and 35% of the samples, respectively. No differences in ∑HBCD concentration were associated with age or sex. This dataset represents the first national data describing HBCD isomers and some PBDEs (e.g., 183, 209) in Canadians.

1. Introduction

The source of greatest human exposure (>90%) to the traditional persistent organic pollutants (POPs), including polychlorinated biphenyls (PCBs) and polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) is dietary intake (Liem et al., 2000). Flame retardants such as the polybrominated diphenyl ethers (PBDEs) and hexabromocyclododecane (HBCD) have multiple important human exposure pathways including diet, dust ingestion and oral contact with consumer products containing flame retardants, such as toys or textiles, while air inhalation has been suggested by some authors (Besis and Samara, 2012; Environment Canada and Health Canada, 2011; Fraser et al., 2009; Health Canada, 2012; Meeker et al., 2009; Roosens et al., 2009a; Shoeib et al., 2012; Sjödin et al., 2003; Trudel et al., 2011). Occupational exposure to workers in industrial and recycling plants also is subject to increased exposure to flame retardants (Eguchi et al., 2012; Sjödin et al., 1999; Stapleton et al., 2008; Thomsen et al., 2007). Both of these classes of flame retardants are added to the product rather than chemically bonded to them and,

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as such, are subject to loss from the consumer product throughout the product's entire lifecycle including the manufacture of the end product as well as its use and disposal (Covaci et al., 2006; Hale et al., 2003; Shoeib et al., 2012).

With the greater usage of PBDEs in North America than in European countries, elevated concentrations have been observed in North American food and environmental compartments, resulting in an increased exposure to people (De Wit, 2002; Domingo, 2012; Hites, 2004; Tomy et al., 2009). In contrast, HBCD usage in Europe, which was greater than in North America, has resulted in a corresponding relative increase in concentrations observed in European environmental and biological samples relative to North America (Covaci et al., 2006; Johnson-Restrepo et al., 2008).

Studies using a variety of organisms to determine the effects of PBDE and HBCD exposure at the present levels have shown that they are not of concern in terms of acute toxicity (Darnerud, 2003). Delayed pubertal progression has been observed in male rats exposed to PBDE formulations where preputial separation delays and reduced ventral prostate and seminal vesicle weights were reported (Korenbrot et al., 1977; Stoker et al., 2004, 2005). Reduced sperm counts also have been reported in male rats exposed to PBDE congeners (Kuriyama et al., 2005). PBDE exposure has additionally been associated with decreased thyroxine (T4) levels in female rats and mice even at low doses (Ellis-Hutchings et al., 2006; Fowles et al., 1994; Kuriyama et al., 2007; Richardson et al., 2006). PBDEs also have been associated with endocrine disruption and developmental neurotoxicity in humans, with prenatal exposure to PBDEs having resulted in decreased fine motor skills and attention (Costa et al., 2008; Eriksson et al., 1998; Legler and Brouwer, 2003; Roze et al., 2009). Linkages have been made between increased PBDE exposure and implications to human reproductive systems such as reduced sperm quality, increased cryptorchidism in newborn boys and reduction in age of menarche in girls (Akutsu et al., 2008; Chen et al., 2011; Main et al., 2007). No relationship was found between PBDE concentrations in adipose tissue and breast cancer risk in a small study in the US (Hurley et al., 2011). Although less toxicological data are available for HBCD in the literature, exposure has been linked to drug-metabolising enzyme induction in both rats and fish (Germer et al., 2006; Ronisz et al., 2004). HBCD exposure has resulted in decreased fertility and thyroid effects in rat models (Ema et al., 2008). Behavioural effects also have been observed in mice (Eriksson et al., 2006). Malignant transformation of mammalian cells also has been reported for HBCD by some authors, although Health Canada concluded that HBCD lacks significant genotoxicity potential (Environment Canada and Health Canada, 2011; Shaw et al., 2010).

Human biomonitoring allows direct measurement of systemic exposure levels of environmental contaminants in contrast to estimates based on the rate of uptake or route of exposure. Biomonitoring provides a clear measurement of how much of a given chemical an individual has retained in the body at the time of sample collection. Biomonitoring studies to determine human exposure to PBDEs and HBCD via targeted surveys are available in the literature, however, few are representative of entire populations and are, generally, limited to comparisons between specific sub-groups within a population (Croes et al., 2012; Eskenazi et al., 2011; Fraser et al., 2009; Kalantzi et al., 2011; Leijs et al., 2008; Roosens et al., 2010; Zota et al., 2011). Prenatal exposure to brominated flame retardants has been confirmed through the study of paired maternal and foetal serum samples and foetal liver and placental tissue paired sampling (Doucet et al., 2009; Mazdai et al., 2003; Schecter et al., 2007). Additionally, analysis of human milk has provided a measure of maternal exposure to POPs and has allowed for dietary exposure estimates to infants and young children through ingestion of maternal milk (Croes et al., 2012; Inoue et al., 2006; Roosens et al., 2010). In the US, the Centers for Disease Control and Prevention (CDC) has conducted an ongoing study, known as the National Health and Nutrition Examination Survey (NHANES) to determine the nutritional and health status of its population annually (Curtin et al., 2013). As part of that national survey, serum and urine have been collected and tested to measure environmental contaminant concentrations (Axelrad et al., 2009; Chen et al., 2011).

The Canadian Health Measures Survey (CHMS), initially undertaken as a one-time survey to determine national baseline estimates of the health status of Canadians between the age of 6 and 79 years, has evolved into an ongoing survey with different collection periods identified by Cycle number (Giroux, 2007). Samples collected from adults aged 20–79 years as part of Cycle 1 of the CHMS included small volumes of plasma (2.7 mL) for PCB and PBDE analysis (Health Canada, 2010; Rawn et al., 2012). Sample collection for POP residue analysis in the initial CHMS Cycle 1 study was restricted to adults (i.e., individuals aged 20 to 79 years) for several reasons, which included avoiding increased rate of refusal to participate and due to the limits on the quantity of blood which can ethically be taken from a child as part of a study. Serum PBDE concentrations measured during the Cycle 1 study were frequently below the limit of detection in the individual samples analysed (Health Canada, 2010). Only PBDEs 47, 99, 100 and 153 were detected in the serum samples studied as part of the CHMS Cycle 1 with concentrations reported at the 50th or 75th percentile (Health Canada, 2010).

In an effort to increase the number of samples with concentrations above the detection limits for a number of classes of POPs, including the PBDEs and HBCD, the present study was undertaken to prepare larger volume pooled samples using the surplus serum from testing in Cycle 1 of the CHMS. Individual leftover serum samples from individuals ranging in age from 6 to 79 years were pooled following grouping by age and sex to develop ~25 mL samples for analysis, using gas chromatography — high resolution mass spectrometry and high pressure liquid chromatography coupled to tandem mass spectrometry, to determine Canadian national baseline PBDE and HBCD concentration estimates.

2. Materials and methods

2.1. Blood collection

The CHMS Cycle 1 sampling was established to represent the Canadian national population and included individuals living in one of the 10 provinces or three territories, but excluded individuals living on First Nations reserves, institutional residents and Canadian Forces full-time members (Statistics Canada, 2010). Sampling required a minimum of 5000 participants over two years and distributed over five regions to ensure that sampling was representative of the national population (Statistics Canada, 2010). As described by Rawn et al. (2012), blood ranging in volume from <28 mL to <80 mL, depending on the age of the donor (Statistics Canada, 2010), was collected during Cycle 1 of CHMS at mobile clinics across Canada, centrifuged, and serum was retained in 2 mL cryogenic vials within 2 h of collection and frozen. Serum samples were shipped weekly to the laboratory on dry ice and stored at −20 °C until taken for pooling. The serum left over after testing for the initially planned analyses was then re-frozen at −20 °C and stored prior to development of the pools.

2.2. Methodology for the creation of pooled serum samples

Statistics Canada developed the protocol for the creation of serum pools from the individual samples, representative of 96.3% of the Canadian population from age 6 to 79 years and was approved by the Health Canada Research Ethics Board prior to initiation of the present study (Verret and Giroux, 2010). The serum pools were formed using sampling weights, which were calculated from a model that was used in creating the original CHMS survey design. The sampling weights correspond to how many individuals of the population that was being studied were represented in a given sample.

Left over serum (350 μL) from individual participants in Cycle 1 of the CHMS was used to create 59 serum pools with approximately 80 individuals per pool, rather than the minimum of 71 individuals needed to obtain the desired 25 mL volume of serum. Individual samples that did