House Dust is the Main Exposure Pathway for PBDEs to Humans.

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Introduction

One of the main triggers that has sponsored legislative action regarding PBDEs is their concentrations in humant milk. Human body burdens are estimated to have a doubling time of 2-6 years in North America[1], causing urgency for action. While it is appreciated that emissions from consumer products are the main route of PBDEs into the environment, one of the puzzling questions regarding human body burdens of PBDEs is their main exposure pathway. Important exposure pathways have been hypothesized to be fatty foods (similar to PCBs), indoor air and most recently, house dust[2]. However, most exposure analyses have failed to account for the elevated body burdens seen in some individuals. Another puzzling question regarding the pattern of PBDEs in human milk[3] is reason for the lognormal distribution that spans nearly three orders of magnitude. Could the breadth of exposures to mothers account for individuals with very high milk concentrations? An alternative hypothesis is that individuals retain PBDEs to different extents where retention could be related to metabolism and/or sequestration.

The aim of this study is to assess the main pathway(s) of exposure of PBDEs to humans. In so doing, we comment on reasons that might cause the broad range of body burdens and speculate on measures that could be taken to reduce exposures at individual and societal scales. Details of the methods and results are provided by Jones-Otazo et al.[4]

Methods

Exposure to ΣPBDEs was estimated using the exposure module of a generic multimedia human health risk assessment model, MUM-FAMrisk[4]. Exposure was calculated for "average" Canadian age classes: infant (0-6 months), toddler (6 months-4 years), child (5-11 years) and teen (12-19 years), adult (20+ years), as defined by Health Canada. We also explored the range of exposures obtained from ingesting low and high concentrations of human milk (infant) and dust (toddler and adult). We used measured and modelled concentrations for environmental media, and only measured concentrations for food. Most of the data were expressed as ΣPBDE and all data sources contained BDE-47, 99, 100, 153 and 154. BDE-209 was excluded from the analysis due to lack of data in most media.

Modelled (Multimedia Urban Model or MUM-Fate) data used in the exposure assessment were ΣPBDE in outdoor soil, outdoor air (gas- and particle-phase), as well as indoor air (gas- and particle-phase) and dust. Mean and maximum measured concentrations of gas-phase ∑PBDEs in residential indoor air were taken from Wilford *et al.* (see ref 4) for 94 homes in Ottawa, Canada. ∑PBDE minimum, mean and maximum concentrations in residential house dust were also calculated from measured gas-phase indoor air concentrations using the equilibrium K_p-K_{OA} relationship, and assuming physical properties for dust as per Bennett and Furtaw (see ref 4). The modelled concentrations correspond to the range of reported values reported in the literature.

Market basket surveys conducted in two Canadian cities, Vancouver and Whitehorse were the source of arithmetic mean concentrations of ∑PBDEs in fatty foods. Concentrations in fruit, vegetables and grain were arithmetic mean

concentrations from Finland and Ottawa, Canada. ∑PBDE concentrations in Canadian human whole milk (mean, maximum and minimum) were calculated as the sum of congener-specific measurements from 98 whole milk samples collected across Canada in 2002.

Food and media intake rates for all life stages were obtained from Nutrition Canada Survey data of Health Canada (see ref 4). Primary data used to estimate mean Canadian exposures included both consumers and non-consumers (zeros in) in order to avoid. overestimating intake rates for non-consumers. We assumed bioavailability (absorption) factors of 100% and 50% from the GI tract. Time-activity data from Richardson (see ref 4) were averaged over 12 months for Canadians over the age of 11 years, while those data for children under age 11 (infants, toddlers, children), were derived from yearly average time-activity data based on the Canadian Human Activity Pattern Survey (CHAPS). To calculate Soil/dust ingestion exposures on a daily basis, we assumed that a receptor spends 92 to > 94% of time indoors where they are assumed to be exposed only to dust, and the remaining time is spent outdoors where they are exposed only to soil. The average soil/dust ingestion rates vary from 0.05 to 0.02 g/d for toddlers and teen/adults, respectively. The inhalation pathway accounted for inhaled and resuspended dust, including gas- and particle-phases.

Results and Discussion

For all age classes, the Estimated daily intake (EDI) for the Average Urban Canadian ranged from 155 ng/day for the adult to 1965 ng/day for the breast-fed infant (Fig. 1). Normalizing for body weight, this corresponds to 2 and 280 ng/kg body wt/ day for the adult and infant, respectively. As with other POPs, human milk contributed most to the infant's exposure. The intake rate via human milk for Canadian infants could vary from 24-28,680 ng/day or 3-4,100 ng/kg body wt/ day, for the low and high concentrations, respectively taken from the range in ΣPBDE concentrations for Canadian women's milk.

For all other age classes, house dust contributed most to exposures (Fig. 1). These high exposures are a product of both high concentrations in this medium and spending 22 hours per day indoors. The exposure occurs through several exposure pathways: direct inhalation of resuspended dust; ingestion of dust through direct and indirect pathways; and, dermal exposure on the hands and body (although we neglect the latter due to high uncertainties and presumed low rates of dermal transfer). Our exposure estimates for dust indicate that ingestion is the critical exposure pathway where ingestion includes that fraction of dust removed from the nasal passages and shunted to the digestive track.

Using this "average" scenario, toddlers have particularly high exposure to house dust: dust accounts for 90% of their EDI of 264 ng/day or 20ng/kg body wt/ day (Supplemental Information, Table 2). This result is not only due to the high PBDE concentration in dust and time spent indoors, but also the relatively high soil/dust ingestion rate of 0.05 g/day (see ref 4). ΣPBDE intake via dust could, however, range from 0.46-19,270 ng/day and 0.035-1482ng/kg body wt/ day if using the low and high soil/dust intakes of 0.01-0.2 g/day from the U.S. EPA, in combination with the 5th percentile and maximum estimated dust concentrations of 0.1-105 µg/g and estimates of bioavailability of 50 and 100%. The range of dust intakes accounts for 1.4-99.8% of the toddler's EDI. In comparison, the adult's range of dust intakes is 0.8-4970 ng/day or 0.02-71ng/kg body wt/ day day using Health Canada's soil/dust intake rate of 0.02 g/day and the U.S. EPA value of 0.05 g/day, minimum and maximum dust concentrations, and bioavailabilities of 50 and 100%.

The high exposure of toddlers and even infants via dust is consistent with assessments of lead exposure where children's blood lead levels have been positively correlated with lead concentrations in house dust. Toddlers are apt to ingest dust due to their frequent hand-mouth contact and "mouthing" toys and other objects in contact with floors. However, our estimated dust exposure rates contain considerable uncertainty because, although the dust intake rates used here have been well vetted, the rates are based on a limited number studies, most of which dealt with the intake of soil, not dust.

Surface dust may achieve even higher concentrations relative to indoor air, due to its proximity to near-surface sources. Characterization of the sources and rates of PBDE emissions, in particular from household items as carpet underpadding, furniture and electronics equipment, will help explain the ranges of PBDE concentrations in dust.

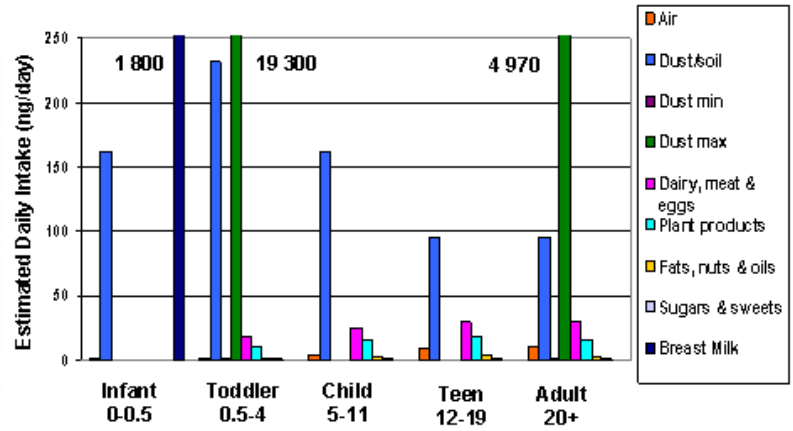

Figure 1. Estimated daily intake (EDI) of ΣPBDEs for "average" Canadian age classes and minimum and maximum intakes of dust for toddlers and adults.

Following household dust, "dairy, meat and eggs" were the next most important exposure media for the Average Urban Canadian (16% contribution): beef, pork, eggs, milk and dairy products and poultry contributed 5, 4.5, 4.3, 1.3 and 0.8% of the total daily intake of ΣPBDEs, respectively. Inhalation of indoor air contributed 6% for the Average Urban Canadian adult. Contributions from fish were only 3.6% of total exposure. Exposure contributions from foods from plants is often overlooked, since it is assumed that as for other persistent and bioaccumulative organics such as PCBs and PCDD/Fs, dietary contributions from animal products dominate exposures. We found that plant products contributed 10% of total dietary exposure, or about 64% that of dairy, meat and eggs.

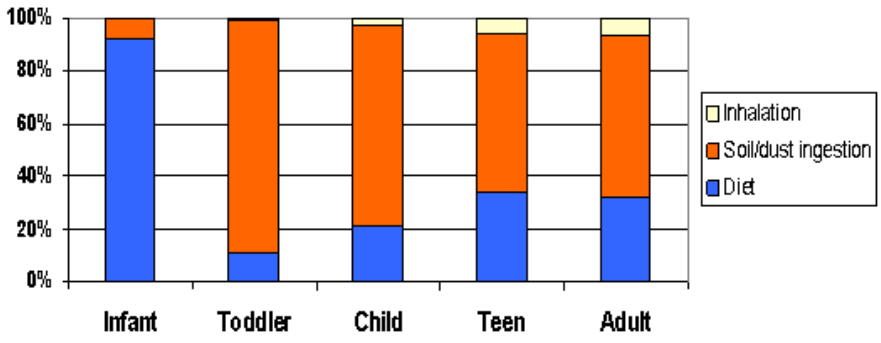

Figure 2. Percentage contributions from inhalation, soil/dust ingestion and diet to "average" Canadian exposure to ΣPBDEs.

These results give rise to optimism that we can take action to reduce our exposure to PBDEs. At the individual level, we can reduce our exposure by changing aspects of the indoor environment, such as reducing dust leves, increasing air ventilation and reducing the sources of PBDEs to the indoor environment. At the societal level, regulatory actions taken to reduce the release of PBDEs in household products will translate over time to lower indoor concentrations and hence exposures. However, if sufficient action is not taken then releases of PBDEs will, as a result of their persistence and fate processes, eventually enter the food system at which point controlling exposure is much more difficult. We have learnt this lesson with legacy POPs such as PCBs.

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