RESIDENTIAL EXPOSURE TO PBDEs: FROM PRODUCT TO PERSON

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Introduction

While polybrominated diphenyl ethers (PBDEs) are found in remote regions of the globe, they are primarily urban or "indoor POPs," persistent organic pollutants that are typically found in much higher concentrations indoors than outdoors. Early work on these compounds assumed that exposure would be primarily via the food chain, by analogy with many other POPs such as PCDDs and PCDFs. However, because PBDEs are used as flame retardants in consumer products, often at concentrations in the percent range, it was suspected that indoor exposure might be important. Measurement of high levels of PBDEs in house dust suggested that incidental ingestion of dust might play a very significant role, particularly for children.[1-3] In studying exposure to PBDEs, most investigators have used the exposure factors approach: i) measure concentrations of PBDEs in various environmental media (e.g., air, dust, food), and ii) multiply them by standard exposure factors—e.g., from the USEPA's *Exposure Factors Handbook* [4]—to estimate exposure. In assessing such studies, several issues must be considered including i) are the measured media concentrations representative of the target population, ii) how well known are the exposure factors? As many studies use convenience samples, the target population is often poorly defined. Some exposure factors such as inhalation rate are relatively well known, but others such as rates of incidental dust ingestion are very uncertain. We have taken a second, complementary approach to exposure assessment that relies principally on environmental epidemiology and field methods. Our goal has been to understand how people are exposed indoors to PBDEs along the complete pathway from product to person (Figure 1). In this paper, we use this paradigm to assess progress towards this goal in the North American context, highlighting important knowledge gaps.

Materials and Methods

We briefly review methods used in our recent studies. Wu et al. measured PBDEs in breast milk collected 2-8 weeks after delivery from 46 first time mothers in the Boston area, collecting personal data (e.g., age, education level, SES) and dietary information (via a food frequency questionnaire); PBDEs were also measured in researcher-collected dust collected from the homes of a subsample of these women.[5] Allen et al. used standardized methods to collect dust from two rooms (main living area and bedroom) of twenty homes in the Boston area; to assess changes over time, the sampling was done in two rounds 6-8 months apart.[6] The participants' vacuum cleaner bags were also collected. During the first round, we actively collected air samples from the two rooms as well as personal air.[7] During the second round, we used X-ray fluorescence (XRF) to measure the bromine concentrations—a surrogate for brominated flame retardants—in furniture, electronics and other products in the two rooms.[8] Stapleton et al collected handwipes from 33 people using a standardized protocol.[9]

Results and Discussion

1) Relationship of body burdens to dust and diet

PBDE body burden provides a measure of absorbed dose integrated over all routes of exposure; the relevant time period depends on the half-life of the congener (weeks for BDE 209, years for penta-related congeners). Using data from the dietary questionnaire, we showed that penta-related BDEs in breast milk were significantly related to pre-pregnancy consumption of meat and dairy products.[5] Inclusion of personal and household characteristics in the model did not substantially affect the regression coefficients for diet. PentaBDEs in breast milk were also associated with concentrations in dust from the women's homes. The association was not confounded by diet, as the concentrations in dust were not strongly associated with diet. These data indicate that both diet and the indoor environment (as measured by dust) were important determinants of body burdens in this population. In terms of Figure 1, they link internal dose of pentaBDEs to personal exposure (diet) and microenvironments (dust).

2) Inhalation Exposure

While the association between concentrations of pentaBDEs in breast milk and dust indicates exposure via the indoor

environment, it does not distinguish between the possible routes of exposure: inhalation, incidental dust ingestion, and dermal exposure. Incidental dust ingestion is hypothesized to be the dominant route based on exposure factors, but these values are highly uncertain, particularly for adults. We measured inhalation exposure within the home, using personal air monitors (personal exposure in Figure 1), comparing the results with those from stationary pumps in the main living area and bedroom (microenvironments).[7] We found that concentrations of the PBDEs were higher in personal air compared with room air, especially the heavier (less-volatile) congeners such as BDE209. This suggests a personal cloud effect due to suspension of dust by everyday activities. Although PBDE concentrations in our residential air samples were higher than those found by earlier studies (using passive air monitors), the levels do not appear high enough to make inhalation a major pathway for most people, with the possible exception of BDE209 for adults.[7]

3) Critical factors in assessing exposure to dust

Using our data on the 20 Boston homes, we were able to examine a series of methodological questions that are important in assessing exposure to dust.[6] Factor analysis of the data suggested that the PBDEs in dust derive predominantly from the three commercial sources, forming three independent log-normal distributions of penta, octa and deca-related congeners. The concentration of PBDEs per gram of dust was highly correlated with the mass of PBDEs per unit surface area; the latter may be more important for assessing exposure. Average concentrations of PBDEs in dust did not significantly change between the two rounds of sampling (6-8 months), perhaps because of minimal change in furnishings. Concentrations of PBDEs were significantly higher in the living room than the bedroom. Concentrations in dust from home vacuum cleaner bags were significantly lower than and not strongly correlated with researcher-collected dust. Although vacuum cleaner bags provide a simple method for collecting dust, they may not provide a good surrogate for researcher-collection. Different types of vacuums may have different efficiencies of capturing particles of various sizes and/or densities. Concentrations of PBDEs in dust can thus depend on both how and where in the home dust is sampled; this should be taken into account when comparing results of different studies.

4) Exposure to dust via hand to mouth activity

Because of the large uncertainty regarding exposure factors for dust ingestion, we decided to try to partially dissect this pathway. One possible mechanism is transfer of PBDEs from dust or indoor surfaces to hands, followed by hand-to-mouth activity. The latter is obvious for young children, but can also occur in adults (e.g., not washing hands before eating). We were able to detect PBDEs on handwipes (n=33 people), sometimes at high levels (1980 ng total PBDEs).[9] In this preliminary study we did not collect information on hand-to-mouth behavior, collect serum samples or collect dust samples from their homes. Thus we could not empirically assess the links between handwipes and dust (personal exposure and microenvironments) or assess the importance of handwipes as a predictor of body burdens (the link between personal exposure and internal dose). Nevertheless, handwipes may provide an improved method for assessing personal exposure to PBDEs over earlier dust ingestion approaches. Indeed, these handwipe data suggest that dust exposure may exceed earlier predictions.[9]

5) Links between product and dust concentrations

Although consumer products are thought to be the major indoor sources of PBDEs, we were unable to find significant associations of either body burdens or dust concentrations with counts of foam-containing furniture and electronics.[5,8] We hypothesized that counts may not work if there are substantial differences in PBDE concentrations between otherwise similar objects; such misclassification can attenuate associations, making them hard to detect. Since it is not generally feasible to do "biopsies" of consumer products from homes and test them for PBDEs, we used a surrogate: bromine concentrations as measured by x-ray fluorescence (XRF).[8] In a validation phase, we found that bromine measured by XRF was strongly associated with PBDE concentrations in samples of foam and electronics. In the second round of dust sampling at homes in Boston, we used XRF to measure bromine in foam-containing furniture, electronics and other objects in the mean living area and bedroom. Bromine levels were generally higher in electronics (particularly TVs) than in furniture, and quite variable within most classes of objects. We created a bromine loading index for each room, multiplying bromine concentrations by surface area (or volume) and summing across furniture and electronics separately. PentaBDE concentrations in dust were significantly associated with bromine loading in furniture but not counts. Bromine loading also increased the association between decaBDE in dust and electronics; the latter association was largely due to televisions and was increased in homes with more people, a potential indicator of

activity. These data link concentrations of PBDEs in microenvironments (dust) back to sources (products). It does not, however, tell us the mechanism by which PBDEs move from products to dust or air. Two pathways have been proposed: volatilization (perhaps increased by heating of electronics) and physical abrasion or weathering. We are currently exploring these pathways.

One of the advantages of the epidemiologic/field study approach to exposure assessment is that it brings useful tools developed in those disciplines. In particular, we can separate the notions of internal validity (i.e., how confident are we of the observed associations in the studied population?) from generalizability (how do the results apply to other populations?). While we believe our studies are valid, work in other, larger populations is needed.

We summarize our findings separately for pentaBDE and decaBDE in Figures 2 and 3 (octaBDE was a minor component of dust). For pentaBDE, our studies have been able to largely complete the pathway from indoor sources to internal dose. Although we found an association between dust and body burdens, more work is needed to understand the links between dust and personal exposure (suggested by the dashed arrow between these compartments). The mechanism of release of pentaBDE also needs further study. Our knowledge of decaBDE exposure is less complete. In particular, we do not yet know the associations between body burdens of BDE209 and either dust concentrations or diet. The mechanism of release of relatively non-volatile BDE209 from products to air and dust remains a major research question. Additional work is needed on exposure to PBDEs in non-residential environments such as offices and cars. Finally, our indoor exposure paradigm treats diet as an exogenous source. However, the production and disposal of products, and releases of PBDEs from them during use, will lead to emissions to the outdoor environment and contamination of food (Figure 4). Indeed as the bolus of PBDEs currently indoors are discarded, they may become more like a conventional POP.[10] This set of pathways—the urban environment as source to the wider environment—needs further exploration.

Acknowledgements
Thanks to colleagues who worked on some of the projects described here, particularly Dr. Olaf Päpke, Dr. Robert Hale and Nerissa Wu.

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Figure 1: Pathway for Exposure from Indoor Sources to Internal Dose. The diagram could be extended to the right to include health outcomes.

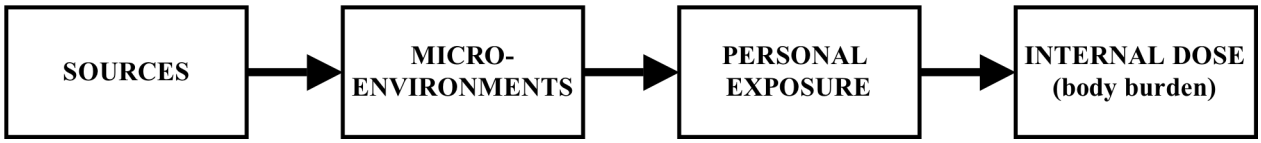

Figure 2. Pathway for Exposure from Sources to Internal Dose for PentaBDE. The details of the pathway from dust to personal exposure are still being worked out.

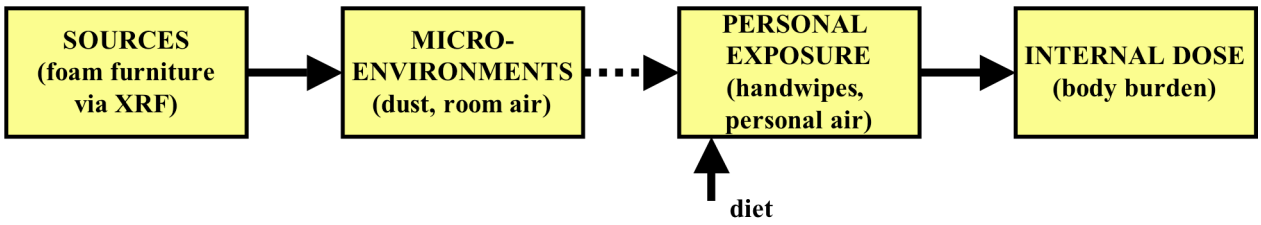

Figure 3. Pathway for Exposure from Sources to Internal Dose for DecaBDE. Numerous links remain poorly understood.

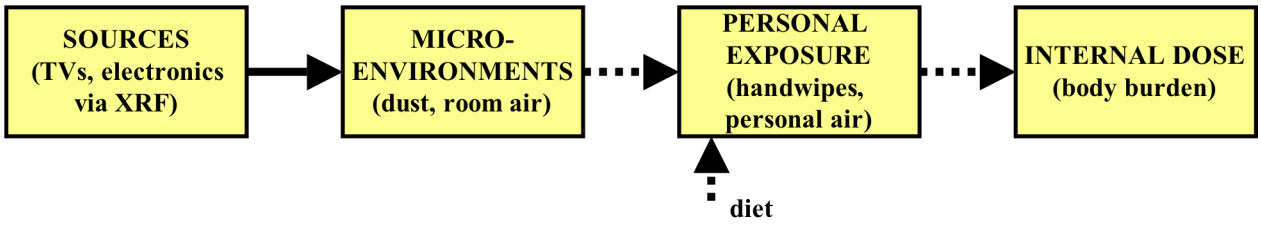

Figure 4. Pathways for Contamination of Food Remain Largely Unexplored.