IMPACT OF DUST FROM MULTIPLE MICROENVIRONMENTS AND DIET ON PENTA-BDE BODY BURDEN

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Introduction

Polybrominated diphenyl ethers (PBDEs) have been widely used as flame retardants in consumer products and are ubiquitous in residential indoor air and dust.[1] Biological measurements of PentaBDEs have been shown to be associated with concentrations in house dust and dietary intake of animal fat, at least in North America where use of this compound was highest.[2-5] However, little is known about exposure to dust in other microenvironments. For example, as fire regulations in many localities (e.g., Boston, MA USA) are stricter for public places than for residences, it is possible that exposure is greater in offices than in homes. Furthermore, while exposure estimates suggest that exposure of US adults via dust ingestion is higher than for food,[6] these estimates are based on uncertain exposure factors, particularly for dust ingestion. Therefore, our objectives were to determine relative contributions of diet and dust exposure from multiple microenvironments to PentaBDE body burden, and to explore the role of handwipes as a measure of personal exposure to PentaBDEs.[7]

Materials and methods

We collected serum, dust (office, main living area, bedroom, vehicle) and handwipe samples in 2009 from 31 adults who worked in offices in the Boston, MA area. Dust samples were collected into cellulose extraction thimbles as previously described.[8] We vacuumed each room for approximately 10 minutes, capturing dust from the entire floor surface area of the room, including accessible floor space under desks and the tops of immovable furniture. We sampled dust from vehicles by vacuuming the front and back seats. We collected handwipe samples from participants at their offices by immersing a 3 inch X 3 inch sterile gauze pad in 3ml of isopropyl alcohol and wiping the palm and back of hand from wrist to fingertips. We collected one 10ml red top Vacutainer tube of blood from each participant at the end of the work week. Tubes were allowed to coagulate at room temperature for 1-2 hours and centrifuged for 15 minutes at 1,000 x g. Dust, handwipes and serum were analyzed for PBDEs using established methods.[7-9]

We used a questionnaire to collect information on personal characteristics, work habits and diet. To assess diet we used a food frequency questionnaire used in our previous study that found associations between PentaBDE body burdens and food consumption.[2] Participants were asked about food consumption over the previous year and shown pictures of standard portion sizes to help estimate quantities. Responses to the dietary questionnaire were converted to a linear scale (servings/day). Dairy consumption was converted to dairy fat using standard data.[2]

PBDE concentrations were log-normally distributed and natural log-transformed before statistical analysis. We used Spearman correlation analysis and multiple linear regression to examine associations between outcomes (e.g., serum or handwipes) and predictors (e.g., dust concentrations, food consumption).

Results and discussion

The participants in our study were predominantly female, 90% white and had a median age of 49 years. Geometric mean concentrations of PentaBDE in dust from offices, main living areas, bedrooms, and vehicles were 2,170 ng/g, 1,690 ng/g, 1,380 ng/g, and 2,610 ng/g respectively. Dust concentrations in vehicles were similar to those reported by Lagalante et al.[10] Dust concentrations in main living areas and bedrooms were significantly correlated ($r=0.49$. $p=0.007$). The geometric mean of PentaBDEs in handwipes and serum were 70 ng and 28 ng/g lipid respectively.

PentaBDEs in handwipes—collected in the offices—were correlated with dust collected from offices (r=0.35, p=0.06) and bedrooms (r=0.39, p=0.04), but not with dust from main living areas (r=-0.05, p=0.77) or vehicles (r=0.17, p=0.47). Reported frequency of handwashing also predicted handwipe concentrations of PentaBDE.

PentaBDEs in serum were correlated with dust from main living areas (r=0.42, p=0.02) and bedrooms (r=0.49, p=0.008), but not with dust from offices (r=0.22, p=0.25) or vehicles (r=0.20, p=0.41). As reported previously, handwipes were a significant predictor of serum concentrations.[5] The final regression model included main living area dust and handwipes, and predicted 55 percent of the variation in serum PentaBDE concentrations (p=0.0004). We did not include bedroom dust in this model: Dust from bedrooms and main living area were correlated and both measure exposure in the home. Furthermore, handwipes may be at least partially an intermediate variable between bedroom dust and serum. Contrary to expectation, dietary variables were not significant predictors of PentaBDEs in serum.

Our research suggests that exposure to dust in the home environment may be the most important factor in predicting PentaBDE body burden in our population, and potential exposure pathways may involve PBDE residues on hands, potentially by hand-to-mouth behavior or dermal exposure. While PentaBDE concentrations were higher in offices than homes, participants spend more time at home; behavior likely also differs: e.g., sitting a desk at work vs. activities at home that may lead to more exposure with dust. The associations between levels on handwipes (sampled in the office) and dust concentrations from the office and bedroom, but not main living area, suggest that handwipes may partially integrate very recent exposure. The majority of our population did not commute to work by automobile, so an association with vehicle dust would not be expected. As serum concentrations were most strongly correlated with dust from residences, future studies should also collect handwipes at home. While office work is common in the USA, our results may not generalize to other populations, particularly other occupations or non-North Americans where concentrations and relative patterns of exposure to PentaBDE may differ. Further work on children is also needed.

Strengths of our study include the concurrent measurement of PentaBDEs from multiple indoor microenvironments (home, office, vehicle), as well as diet, handwipes and serum. This design theoretically allows assessment of the relative importance of each exposure source and has not, to our knowledge, been reported before. Weaknesses of our study include a relatively small sample size, limiting our statistical power. As the best method for collecting dust for use as a determinant of human exposure is not known, random error may weaken the associations we found between dust and serum.[1,8] Similarly, error in assessing food consumption is likely to weaken observed associations with dietary exposure.

While previous US studies have found associations between PentaBDE body burdens and consumption of meat and/or dairy products,[2-3] we did not observe this association in our study population even though we used a survey instrument very similar to the one we used earlier in 2004-5.[2] Our inability to find an association with diet in the current study may be due to dietary measurement error, a smaller sample size or differences in the study population, including somewhat reduced variation in food consumption. It is also possible that the diet signal has become weaker in the five years between collection of samples. Huwe recently reported a 50-80% decrease of PentaBDEs in meat and poultry between 2002 and 2008.[11]

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