TRANSFER MECHANISMS OF POLYBROMINATED DIPHENYL ETHERS AND HUMAN EXPOSURE PATHWAYS: FOCUS ON INDOOR AND OTHER MICROENVIRONMENTS

Dongli Wang and Jianwen She
California Department of Public Health, Environmental Health Laboratory Branch, 850 Marina Bay Parkway, G365, Richmond, CA 94804, USA

Introduction

Polybrominated diphenyl ethers (PBDEs), used as brominated flame retardants, are incorporated into plastics, textiles, foams, and electronic equipment in houses, workplaces, and vehicle interiors, and have been associated with rapidly increasing and elevated levels of these pollutants in indoor environments, biota, and people. Serum levels are approximately 10-fold higher in North American residents than in Europeans and Asians (1). Potential external exposure sources include indoor air, house dust, and direct contact with treated products, and contaminated foods (2). Studies on diet, indoor air, house dust and biomonitoring in Germany suggested that dietary exposure is the dominant intake pathway (3). However, overall PBDE exposure assessment showed that food consumption may be a minor exposure pathway, while indoor environment is a primary route of exposure for the American population (4-6). Additionally, PBDEs were also found in air and dust samples in other microenvironment such as offices (7), school classrooms (8), and from vehicles (9-11).

Although indoor and other microenvironment are main exposure sources of PBDEs, transfer mechanisms of PBDEs from household items to indoor air and dust, and thus to human body have not been clearly understood. This study reviewed the recent published studies on human exposure to PBDEs in indoor and other microenvironments. The objectives are to summarize current knowledge and research status, and supply further insight into the routes of human exposure to different PBDE congeners, and into linkage of levels of PBDEs in microenvironment with body burdens.

Human exposure to PBDEs in house dust

House dust has been identified as the primary route of PBDE exposure (12). Incidental ingestion and dermal contact with house dust may cover 82 % in overall human exposure by an EPA review (5). Total PBDE levels in breast milk from 11 U.S. women were correlated with their house dust concentrations (6). BDE 47, 99, and 100 in house dust from 50 homes were correlated with those in serum of male-female couples from 12 of the homes. Dust PBDE concentrations may be used as a marker for exposure to BDE 47, 99, and 100 (13). However, dust and serum levels of BDE 153 were not correlated, and the correlation of dust and serum levels of BDE 209 could not be evaluated due to low detection rates of BDE 209 in serum (13).

The primary indoor emission sources for PBDEs have been home or office fabric products and electronic and electrical appliances (14). Higher human body burden of PBDEs in Californians may result from the state's strict furniture flammability standards (15, 16). Studies on emissions of PBDE from U.S. houses and garage demonstrated that PBDE release total about 4 μg/h per house or 20 ng/m^3, and US houses and garage collectively release about 4100 kg/year. Most of these emissions are settled in floor dust, but about 20% are released directly to the ambient environment via airborne vapor and particulate matter (17).

However, studies are limited on how PBDEs are transferred to house dust from household products. Takigami et al. (18) performed microenvironment experiments in TV sets to confirm that PBDEs are migrated to dust from the TV set components (18). The possible mechanism may involve some physical processes such as abrasion or weathering (19), vaporization, migration and adsorption of PBDEs in heterogeneous phase media (such as indoor air, dust, and polymeric materials) (18).

Inadvertent ingestion of house dust is the major contributor to PBDE exposure of toddlers through to adults and is thus the main exposure pathway for all life stages. The infant may also uptake PBDEs from the mother's milk (4). Particularly, exposure to PBDEs via ingestion of indoor dust makes children at a greater risk. In 19 of 20 families

studied, children had higher ∑PBDE concentrations than their mothers. BDE 209 was also found in 13 children and 9 mothers (20). BDE 209 was found the dominant congener in the indoor dust, while BDEs 47 and 99 predominated in human milk and blood as well as food (21). Dust samples containing PBDEs were administered to male rats and found that retention of PBDEs in tissues ranged from <5% of the dose for BDE-209 to 70% for BDEs-47, 100, and 153 (22). This study showed that BDE 209 has fairly lower bioavailability. Children's increased hand-to-mouth activity, dietary preferences, and exposures from breast milk may result in greater ingestion of PBDEs than adults (20). However, adults have fewer opportunities to ingest dust than children, and the findings on correlation between adult PBDE body burden with those in indoor dust drive us to consider other human exposure pathways such as inhalation of indoor air and body direct contact with household items.

Human exposure to PBDEs in indoor air

In the indoor and other microenvironments, PBDEs coexist in household product materials, the air (including the gas and the particle phase), and house dust. The partitioning and deposition of PBDE congeners among heterogeneous phase media (such as gas, dust, and polymeric materials) are largely controlled by chemical and physical properties of PBDE congeners, such as their vapor pressure and by the prevailing environmental conditions, such as the micro-environmental temperature. The portion of the compounds in the indoor air can also be important concern for determining human exposure (23). Studies have showed that PBDEs were present in both the particle and gas phases. About 80% was in the gas phase for BDE 47, and about 55-65% was in the gas phase for BDE 100 and BDE 99, and for BDE 154 and BDE 153 only about 30% was in the gas phase (24). Allen et al. (25) used personal air samplers to measure indoor air exposure to PBDEs for 20 residents of the Greater Boston Area, and found that total personal air concentrations (particulate + vapor) were 469 pg/m^3 for non-209 BDEs and 174 pg/m^3 for BDE 209. Inhalation may account for up to 22% of the total BDE 209 exposure in U.S. adults. As the level of bromination of PBDEs congeners increased, the accumulation level in human milk and serum decreased (Table 1). Due to high vapor pressure, low brominated congeners may be partitioned from polymeric materials directly into both air and house dust. Human might be exposed to these low brominated congeners directly from indoor air. She et al. (26) used principle compound analysis (PCA) on the relative congener profiles to identify the exposure sources for various population groups. The congeners in the PCA analysis included BDE 28, 47, 99, 100, 153, 154 and 183. The results demonstrated that the exposure of the general population in the US is closely related to pentaBDEs, and the PBDE in indoor air (gas phase) in the US is highly correlated with the PBDE burden in the general population in the US, indicating a major exposure pathway (Figure 1, 26). The higher brominated congeners such as BDE 209 were detected in house dust in most situation with high concentration, but were rarely detected in human specimen in general populations (Table 1) except for occupational population in China (26) and occupational population in Sweden (32).

Human exposure to PBDEs via direct contact

Most individuals are spending time a day in close proximity to their cell phones, computers, car seats, sofas, pillows and bedding in offices and homes. PBDEs were found in air and dust samples from offices and other microenvironment. Compared with those from residences, concentration of PBDEs in offices and cars air samples may be elevated by three times and 1.7 times, respectively (27). In vacuum cleaner dust collected from one square meter of carpet in residents, offices and upholstered car seats, the average concentrations of tri- to hexaBDE was 3.2 higher in offices and 30-fold higher in cars (9, 28). Mandalakis et al. (10) also reported high levels of PBDEs in the interior of vehicles. BDE 209 was the dominant congeners in all the microenvironment, and especially in cars and residences. PBDEs in pillows and automotive seats are released by off-gassing or breakdown of polyurethane foam and fabrics, resulting human exposure (29). However, little is known whether and to what degree we are uptaking PBDEs from these items via direct contact.

To better understand human body burdens, further research is required into phase partitioning, the sources and exposure pathways of PBDEs and metabolic differences influencing an individual’s response to exposure. In

addition, temporal trend analysis is necessary with continued monitoring of PBDEs in the human populations as well as in the suggested exposure matrices of food, dust and air.

Table 1. PBDEs levels in paired house dust, indoor air and serum levels.

BDE congeners	dust (ng/g)	air (ng/PUF or pg/m^3)	serum (ng/g lipid)	vapor pressure (Pa)
17	nd	1.35	nd	
28	nd	2.03	2.01	
47	520 (91)	15.17 (55)	19.11 (4.4)	2.19×10^{-5}
49	nd	2	nd	
66	nd	2.7	nd	
85	nd	3.68	1.95	
99	614 (184)	2.79 (36)	4.06 (0.9)	1.26×10^{-5}
100	120 (38)	1.25 (6.4)	2.77 (1.2)	1.44×10^{-5}
153	73 (23)	3.55 (nd)	4.53 (1.4)	0.257×10^{-5}
154	51	nd	nd	0.447×10^{-5}
183	102	nd	0.3	
206	51	nd	nd	
207	32	nd	nd	
209	1398 (377)	5.48 (nd)	nd (0.3)	1.26×10^{-12}

Note: human serum data from Imm et al. (29), and air level expressed as ng/PUF; for congeners 47, 99, 100, 153 and 209, human milk data in parenthesis cited from the study by Toms et al. (30), and air level as pg/m^3. Vapor pressure data was from the study by Strandberg et al. (31).

References

1. Hites RA. (2004); *Environ Sci Technol*. 38(4):945-956
2. Frederiksen M, Vorkamp K, Thomsen M, Knudsen LE. (2009); *Int J Hyg Environ Health.*12(2):109-134
3. Fromme H, Körner W, Shahin N, Wanner A, Albrecht M, Boehmer S, Parlar H, Mayer R, Liebl B, Bolte G. (2009); *Environ Int*. 35(8): 1125-1135
4. Jones-Otazo H, Clarke J, Diamond M, Archbold J, Fergison G, Harner T. (2005); *Environ Sci Technol*. 39: 5121-5130
5. Lober M. (2008); *J Exp Sci Environ Epidemic* 18: 2-19
6. Wu N, Herrmann T, Paepke O, Tickner J, Hale R, Harvey LE, La Guardia M, McClean MD, Webster TF. (2007); *Environ Sci Technol*. 41(5):1584-1589
7. Batterman S, Godwin C, Chernyak S, Jia C, Charles S. (2010); *Environ Int*. 36(6): 548-556
8. Harrad S, Goosey E, Desborough J, Abdallah MA, Roosens L, Covaci A. (2010); *Environ Sci Technol*. 44(11): 4198-4202
9. Lagalante AF, Oswald TD, Calvosa FC. (2009); *Environ Int*. 35(3):539-344
10. Mandalakis M, Stephanou EG, Horii Y, Kannan K. (2008); *Environ Sci Technol*. 42(17):6431-6436
11. Stuart H, Ibarra C, Abdallah MA, Boon R, Neels H, Covaci A. (2008); *Environ Int*. 34(8):1170-1175
12. Wilford BH, Shoeib M, Harner T, Zhu J, Jones KC. (2005); *Environ Sci Technol*. 39(18): 7027-7035
13. Johnson PI, Stapleton HM, Sjodin A, Meeker JD. (2010); *Environ. Sci. Technol*. DOI: 10.1021/es100697q
14. Chen SJ, Ma YJ, Wang J, Tian M, Luo XJ, Chen D, Mai BX. *J Hazard Mater*. (2010); 176(1-3): 979-984

15. She J, Petreas M, Winkler J, Visita P, McKinney M, Kopec D. (2002); *Chemosphere*. 46(5): 697-707
16. Zota AR, Rudel RA, Morello-Frosch RA, Brody JG. (2008); *Environ Sci Technol*. 42(21): 8158-8164
17. Betterman S, Chernyak S, Jia C, Godwin C, Charles S. (2008); *Environ Sci Technol*. 43(8): 2693-2700
18. Takigami H, Suzuki G, Hirai Y, Sakai S. http://bfr2010.com/abstract-download/2007/P079.pdf
19. Webster TF, Harrad S, Millette JR, Holbrook RD, Davis JM, Stapleton HM, Allen JG, McClean MD, Ibarra C, Abdallah MA, Covaci A. (2009); *Environ Sci Technol*. 43(9): 3067-3072
20. Lunder S, Hovander L, Athanassiadis I, Bergman A. (2010); *Environ Sci Technol*. 44 (13): 5256–5262
21. Schecter A, Päpke O, Tung KC, Joseph J, Harris TR, Dahlgren J. (2005); *J Occup Environ Med.* 47(3):199-211
22. Huwe JK, Hakk H, Smith DJ, Diliberto JJ, Richardson V, Stapleton HM. Birnbaum LS. (2008); *Environ. Sci. Technol*. 42 (7): 2694–2700
23. Sjödin A, Hagmar L, klasson-Wehler E, Kronholm-Diab K, Jakobsson E, Bergman A. (1999); *Environ Health Perspec*. 107: 643-648
24. Harner T, Bidleman TF. (1998); *Environ Sci Technol*. 32: 1494-1502
25. Allen JG, McClean MD, Stapleton HM, Nelson JW, Webster TF. (2007); *Environ Sci Technol*. 41(13): 4574-4579
26. She J, She, Y, Song W. (2010); *Sci China* 53: 995-1002
27. Harrad S, Hazrati S, Ibarra C. (2006); *Environ Sci Technol*. 40(15): 4633-4638
28. Harrad S, Ibarra C, Diamond M, Melymuk L, Robson M, Douwes J, Roosens L, Dirtu AC, Covaci A. (2008); *Environ Int*. 34(2): 232-238
29. Imm P, Knobeloch L, Buelow C, Anderson HA. (2009); *Environ Health Perspect*. 117(12): 1890–1895
30. Toms L, Hearn L, Kennedy K, Harden F, Bartkow M, Temme C, Mueller JF. (2009); *Environ Int*. 35: 864-869
31. Strandberg B, Dodder NG, Hites R. (2001); *Environ Sci Technol.* 35: 1078-1083
32. Sjödin A, Carlsson H, Thuresson K, Sjolin S, Bergman A and Österman C (2001), Environ. Sci. Technol., 35, 448-454

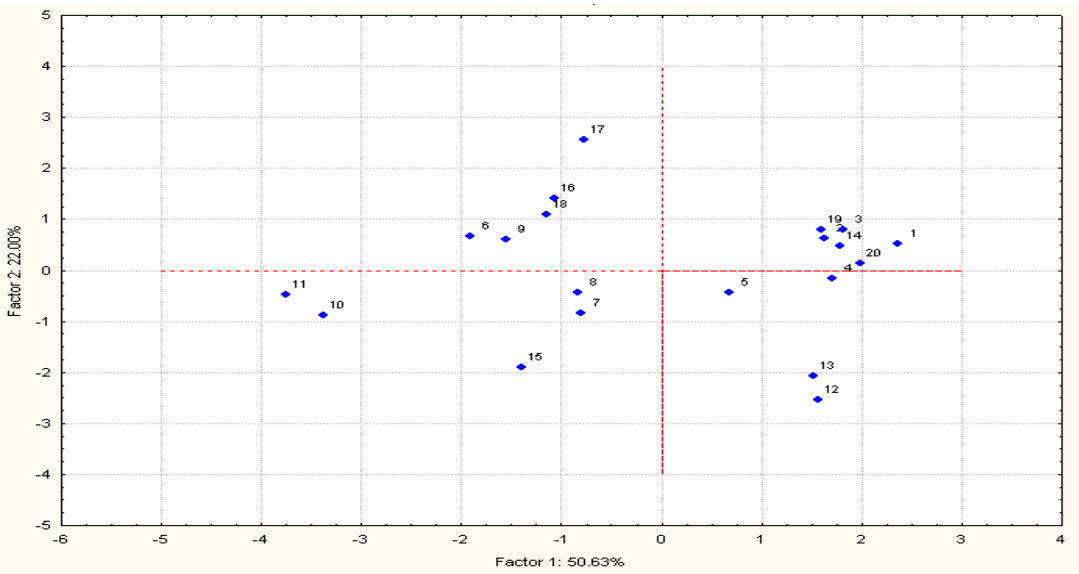

Figure 1. The score plot of PBDE exposure in China and the US (the first two PCs explain 78 % of the variance). Indiana blood (1, 5), California blood (2), USA blood (3), Washington blood (4), the USA indoor gas phase (14); Pacific northwest milk (19); and Texas Milk (20).