An Exposure Assessment of Polybrominated Diphenyl Ethers

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ABBREVIATIONS AND ACRONYMS

(To be completed)

BDE   brominated diphenyl ether

PBDE   polybrominated diphenyl ether

PREFACE

The United States Environmental Protection Agency (U.S. EPA) has formed a working group comprised of individuals from several program offices including the Offices of Pesticides, Prevention, and Toxic Substances, the Office of Water, and the Office of Research and Development, Office of Policy, Economics, and Innovation, to study production, use, alternatives, environmental fate, exposure, and health effects of polybrominated diphenyl ethers (PBDEs). This working group issued a project plan in 2006 that outlined projects in these areas. EPA reports regularly on progress in completing the activities identified in the project plan, with the most recent status report issued in March 2008. The Web site that describes this working group, including the project plan, is http://www.epa.gov/oppt/pbde. This document addresses the exposure assessment needs identified in that project plan. It provides a comprehensive assessment of the exposure of Americans to this class of persistent organic pollutants. Individual chapters in this document address: the production, use, and lifecycle of PBDEs; environmental fate; environmental levels; and human exposure.

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EXECUTIVE SUMMARY

Polybrominated diphenyl ethers, PBDEs, are a class of brominated flame retardants that are added to plastics, polyurethane foam, textiles, and electronic equipment to reduce the likelihood of ignition and to slow the burn rate should products catch fire. Largely as a result of using fire-resistant materials in construction, consumer goods, plastics and textiles, the incidence of fires has decreased 24% over the past 10 years. PBDEs are a major class of flame retardants contributing to this benefit.

PBDEs have a common structure of a brominated diphenyl ether molecule that may have anywhere from 1 to 10 bromine atoms attached. Depending on the location and number of bromine atoms, there are a possible 209 PBDE compounds; each are termed *congener* and are assigned a specific BDE number. PBDEs have been marketed in three primary formulations: (1) the "penta" formulation, commercially known as DE-71® and Bromkal 70-5DE®; (2) the "octa" formulation, DE-79®; and (3) the "deca" formulation, DE-83R® or Saytex 102E®. The formulations differ in their composition of BDE congeners. The dominant congeners in pentaBDE (percent weight basis in parenthesis) are BDEs 99 (35–50%), 47 (25–37%), 100 (6–10 %), 153 (3–5%), and 154 (2–4%). The octa formulation is comprised of BDEs 183 (40%), 197 (21%), 203 (5–8%), 196 (8%), 208 (10%), 207 (7%), 153 (5–10%), and 154 (1–5%). The deca formulation is dominated by BDE 209 (97–99%), with the remainder being BDEs 207 and 208.

Studies have been conducted in laboratory animals to gain a better understanding of the potential health risks of PBDEs. These studies have suggested potential concerns about liver toxicity, thyroid toxicity, developmental and reproductive toxicity, and developmental neurotoxicity. These findings raise particular concerns about the potential risks to children. To date, there is no evidence of carcinogenicity of any of the PBDEs except decabromodiphenyl ether, BDE 209. In a review of toxicological studies as part of a Toxicological Review for the U.S. EPA's Integrated Risk Information System (IRIS), the U.S. EPA has found that the data supports a finding of "suggestive evidence of carcinogenic potential" according to the 2005 U.S. EPA draft *Guidelines for Carcinogenic Risk Assessment*.

Approximately 56,418 metric tons (MT) of PBDEs were produced worldwide in 2003, the latest reporting year, with between 40,000 and 67,000 MT/yr produced between

1999 and 2002. In 2001, decaBDE accounted for 83% of total worldwide production, followed by pentaBDE (11%), and octaBDE (6%). Approximately 95% of the global production of pentaBDE, 40% of octaBDE, and 44% of decaBDE were consumed in the Americas (North, South, and Central America). The penta and octa formulations were voluntarily withdrawn from the United States marketplace by their manufacturers at the end of 2004, leaving only the deca formulation currently being marketed for use in commercial products in the United States. The penta and octa formulations were banned in Europe, thus leaving the deca formulation as the only currently used formulation in Europe, too.

A limited lifecycle analysis provides some indication of the magnitude of releases of PBDEs to the outside and inside environments, based on estimates of releases from PBDE production processes, from volatilization of PBDEs in products while in use, and from the disposal of products containing PBDEs. The Toxics Release Inventory (TRI) reports a total release of 33.5 metric tons (MT) of decaBDE to the air, land, and water in 2006. According to TRI, total environmental releases peaked in 1999, with a release of 53.9 MT, and stayed at similar levels through 2002. There was drop in releases in 2003, to 36.3 MT, followed by an increase in 2004 of 44.8 MT, and then sequential declines in 2005 and 2006. Limited studies, including measurement of PBDE releases from products in experimental air chambers and measurement of the concentrations of PBDEs in consumer products, provide a starting point for estimating the releases from products in use. The European Union used an empirical equation, which was developed to approximate the volatilization rate of chemical plasticizers added to plastic films during their product use, to estimate the annual release of PBDEs in Europe. According to their calculations, the annual volatilization rate of all decaBDE, octaBDE, and pentaBDE from consumer products, expressed as a percent of the total mass of these PBDEs introduced into products during the year, is 0.038%, 0.054%, and 0.039%, respectively. Based on use data, they estimated annual releases from volatilization to be 2.6 MT/yr of decaBDE, 1.4 MT/yr of octaBDE, and 4.3 MT/yr of pentaBDE. This same empirical approach was used to estimate volatile releases from these products in the Americas. The estimates include 7.5 MT/yr of decaBDE, 0.8 MT/yr of octaBDE, and 26.6 MT/yr of pentaBDE (based on use data from 2001, when octaBDE and pentaBDE were still being marketed

for use). These estimates can, at best, be considered initial estimates that are unverifiable, but they give an order of magnitude indication of the releases of these flame retardants from products in use.

After use, releases can also occur from disposal of products. An estimate of the releases from disposal of electronic and electrical equipment (EEE) was made. In the United States, an estimated 2.4 million MT (MMT) of EEE waste was generated in 2005. Of this amount, 0.3 MMT was recovered for recycling, leaving 2.1 MMT that was either disposed of in landfills or incinerated. Losses from PBDEs in recycled products were not considered. Losses from landfills were also not considered because of the careful conditions (liners, treated leachate) required by law for hazardous waste landfills in the United States. Based on measured concentrations of decaBDE in EEE products and the destruction efficiency from municipal solid waste incinerators with a high degree of pollution control, releases of decaBDE totalled 0.046 MT/yr. Releases to water and land from effluent and sludge from sewage treatment plants were also considered. Congener-specific releases to land and water were estimated, and the total loading to water (sum of 28 congeners) was 12.8 MT/yr to land and 1.4 MT/yr to water.

Once in the environment, PBDEs persist for decades and have been detected globally in air, soils, sediments, oceans, and wildlife. They are lipophilic and persistent organic compounds having a strong propensity for bioaccumulation and biomagnifications in the aquatic and terrestrial food webs. The atmospheric transport and surface deposition of PBDEs is the primary means of distributing PBDEs over long geographical distances. Once released into the air, PBDEs partition between the vapor and particle bound phases in the atmosphere in accordance with their respective vapor pressures. Lower brominated PBDEs primarily exist in the vapor phase, while higher brominated congeners are primarily adsorbed to atmospheric particles. Photolytic degradation is the primary environmental metabolic pathway, although anaerobic degradation has been shown to occur in sediments. These abiotic degradation processes can result in debromination, which the stripping of bromine atoms from the polybrominated diphenyl ether molecule. Soils and sediments are the ultimate sinks for PBDEs released into the environment.

Several studies have shown that the higher brominated BDE congeners can undergo biotic debromination. Several recent studies have provided evidence of microbial mediated reductive debromination of decaBDE and octaBDE under laboratory conditions. For example, in one study, *Sulfurospirillum multivorans* bacterium incubated with deca-BDE has induced reductive debromination of deca-BDE in vitro to yield octa-BDE and hepta-BDE after a contact time of 2 months. The octa- and hepta-BDE did not further debrominate in the presence of the microbe—even after one year. The researcher of this study concluded that the microorganism was specific to the degradation of deca-BDE and is incapable of debrominating lower brominated PBDE compounds. In another study, BDE 209 was debrominated *in vitro* to yield BDEs 206, 207, and 208 by contact with anaerobic mesophilic microorganisms indigenous to raw sewage sludge (microbial species not identified). Methane was formed as a product of microbial respiration, and the amount of BDE 209 decreased by 30% within 238 days.

Debromination in fish, birds, and mammals have provided evidence that this process occurs *in vivo*. Laboratory studies of rainbow trout, lake trout, and carp, involving fish food spiked with pure BDE 209, have clearly shown accumulation of lower brominated BDE congeners not initially present in the feed. This evidence is suggestive of metabolic synthesis of lower brominated congeners through debromination of BDE 209. In another study, Sprague-Dawley rats were fed a commercial formulation of decaBDE, which is 98.5% BDE 209. Evidence of metabolic debromination of BDE 209 to lower congeners was observed from an apparent 160% increase in the tissue concentration of BDEs 197, 201, and 207 in the sacrificed rats as compared to levels in the feed. Additional evidence for *in vivo* metabolic debromination was found for chickens, starlings, and even house cats.

The study on house cats involved measuring the PBDE congener profile in cat fat and then also in the serum of house cats consuming dry food only, canned wet food only, and a combination of dry and wet cat food. The contamination of PBDEs in dry cat food reflected the congener profile of decaBDE, with BDE 209 representing 83-93% of total PBDE present in the food. Because of the high content of BDE 209 in dry cat food, BDE 209 dominated serum in cats only consuming the dry food. BDE 209 accounting for 4.2%, 21%, and 30% of serum PBDE levels in house cats consuming canned-, mixed-,

and dry-food, respectively. It was noted that BDE 207 was consistently present in serum in significant concentrations of the dry food eaters as compared to the consumers of the other food types. BDE 207 accounted for 4.5%, 9.8%, and 17% of the PBDE levels detected in cats consuming canned-, mixed-, and dry-food-eaters, respectively. BDE 207 was present in the dry food eaters at approximately 50% of the total concentration of BDE 209 which is uncharacteristic of the decaBDE congener profile (BDE 207 is approximately 1% of BDE congeners present in decaBDE), and was not the pattern observed in the wet food eaters. Moreover, the ratio of BDE 207 to BDE 209 in cat serum was relatively constant in all dry cat food eaters. The authors regarded these data as possible evidence for the metabolic debromination of BDE 209 to form BDE 207.

PBDEs have captured the attention of scientists and policymakers because levels in the environment and humans have increased rapidly since these chemicals came into use in the 1960s and 70s. Environmental time-trends can be observed from lake sediment core studies and archived, animal tissue samples. The sediment cores show a predominance for, and a stark rise in, BDE 209, while the animal tissue samples in general show a predominance and rise in BDE 47. The rise in PBDE concentrations in human blood and breast milk in North Americans (both from the United States and Canada) throughout the 1990s into the 2000s, coupled with the fact that North American body burdens exceed those of Europeans and others by factors of 10 or more, has served to focus attention on North American exposures to PBDEs. The penta PBDE formulation has garnered the most concern because it appears to be the major contributor to current environmental, biota, and human body levels. Even with both the penta and octa formulations having been withdrawn from the United States market, past use and possible debromination of higher brominated congeners (eg., BDE 209) by photolytic or biological mechanisms to form lower brominated congeners might result in the continued presence of lower brominated congeners in humans and the environment.

Studies measuring the concentrations of the BDE congeners in environmental and exposure media concentrations were compiled and summarized with the ultimate goal of selecting representative BDE congener concentrations in exposure media (air, dust, food, etc.) to which Americans are exposed. While the data were insufficient to derive concentrations that could be considered statistically representative of the general US

population, they were deemed adequate for conducting the exposure modeling done in this report. These exposure media concentrations were combined with exposure contact rates (air breathed, food eaten, etc.) in order to estimate an overall intake dose. Concentrations and contact rates were used to characterize background central tendency exposures. Exposure media concentrations were either the straight average or geometric mean of the concentrations found in the study selected to represent national background conditions. Contact rates were arithmetic averages for adult populations as provided in the U.S. EPA's *Exposure Factors Handbook*.

As lipophilic contaminants, PBDEs bioaccumulate in the lipids of organisms. In humans, lipid concentrations are typically measured in blood and breast milk, although adipose tissue concentrations have also been measured. Measured human concentrations of BDE congeners were compiled and representative background concentrations of BDE congeners in human blood and milk of Americans were selected. Then, a simple pharmacokinetic model was used to predict the background lipid concentrations of the BDE congeners in humans using the background intake estimates. These predictions were compared to the selected representative body burden concentrations. The model predictions matched the measurements fairly well (as discussed below), suggesting confidence in the exposure characterization. Figure E-1 displays this study approach.

This exposure exercise focused on the congeners most often studied and found in the environment. It is noted, however, that there is not a final selection of *toxic* or otherwise *most critical*, congeners as there is, for example, with dioxin-like compounds. Some studies focus on as few as four congeners while others measure over 15 congeners. The total concentrations found in the individual studies (including for discussions below) can mean the sum of *different* congeners. Many studies have focused on the penta formulation congeners and not measured the critical deca congener, BDE 209. Reasons cited for not including this congener include the historical predominance of the other congeners and the analytical difficulties associated with measuring BDE 209. The congeners selected for final modeling in this exercise include 28, 47, 99, 100, 138, 153, 154, 183, and 209. Tables E-1 and E-2 provide the results of this exposure exercise.

Table E-1 displays the final selection of congener concentrations in exposure media. Very few studies were found that characterized surface water and surface soil.

Surface water concentrations, used as a surrogate for drinking water, were measured in the San Francisco estuary. The total water concentration was 146 pg/L. A total of 33 surface soil measurements were taken in 15 states and measured for 30 BDE congeners in the single study found for the United States. Concentrations of total BDEs averaged 103 ng/g dry weight (dwt), with a geometric mean concentration of 5.3 ng/g dwt. Average soil concentrations were used in this exercise. Outdoor air was characterized by a study by the California Air Resources Board (CARB). The CARB data of 84 samples were taken in 2004 from 7 monitors on 12 dates from locations in the Bay Area and the South Coast. While the profile with a 158 pg/m^3 average might be higher than other outdoor profiles, it is still a reasonable representation of urban or suburban conditions. The indoor environment has been a focus of study because of the use of PBDEs in consumer products found in the home; a number of studies measuring PBDEs in indoor air and house dust were found. The profile in indoor house dust originated from a study from dust taken in 10 homes in 9 different states in the western part of the country. The total of 8,275 ng/g dwt in that representative profile compares with 5,811 ng/g dwt from a study of 17 homes in Washington, DC area, and 9,271 ng/g dwt from 2 samples from a computer lab in California. The geometric means of three locations (bedroom, living room, and from a household vacuum) within 20 homes in Boston of 6,332, 13,882, and 4,213 ng/g, respectively also compare well to the 8,275 ng/g dwt estimate. The indoor air profile came from this study of Boston homes in which the authors determined geometric mean concentrations for 3 locations (personal, bedroom, living room). The average of the 3 geometric means, 447 pg total BDEs/m^3, was used as the indoor air concentration.

Numerous studies on concentrations in food were summarized. A wealth of studies on fish in the wild, raised in aquaculture, and purchased in the market place showed a wide range in concentrations. Generally, concentrations in the wild were found to be higher, and, in some studies, substantially higher than farm-raised or store-bought fish. Essentially all of the representative food profiles, including the fish profiles, originated from a single study sampling food from the retail market place in Texas. While limited geographically, the market basket survey includes 62 samples, split between samples of meat, dairy, and fish, taken from several supermarkets in Dallas. Thirteen congeners, importantly including BDE 209, were measured. The average total

concentrations (not lipid weight based) used from this study include the following: 1.17 ng/g in finfish, 0.13 ng/g in beef, 0.28 ng/g in pork, 0.36 ng/g in poultry, 0.11 ng/g in dairy, and 0.09 ng/g in eggs. A total concentration of 3.6 ng/g in shellfish originated from the study in the San Francisco estuary, which also measured surface water concentrations.

Body-burden studies were compiled and reviewed. Nearly all body-burden data is from human blood and breast milk. Table E-2 shows the final selected profiles of BDE congeners in blood and human breast milk of Americans. Body burdens of Americans are higher than body burdens of individuals in other countries; these data suggest total PBDE body burdens in the range of 30 to 100 ng/g lipid weight (lwt) for Americans, while body burdens of less than 10 ng/g lwt have been found for people in other countries. Most of the data outside of the United States is from Europe. The study selected as the representative blood profile came from the National Health and Nutritional Examination Study (NHANES) from 2003/4. The geometric mean concentration of all adults from this study was 36 ng/g lwt. The predominant congener found in body burden studies is BDE 47, explaining about 50% of the total concentration. The next most abundant BDE congeners are 99 and 153, both explaining the range of 10-20% of total concentrations. Most of the studies have not measured BDE 209, including the NHANES study, but, when measured, it was found in about half the samples at low levels near 1-2 ng/g lwt, with the exception of the one case. In this case study, which entailed a family of 4 including two parents and two young children, concentrations of BDE 209 were above 100 ng/g lwt in the children. Low levels of BDE 209 have been attributed to the rapid half-life of 15 days in humans, and the higher levels in children from this study were attributed to dust exposures in the house.

Unlike human blood data, there is no nationally representative breast milk study that could be used to characterize concentrations of PBDEs. Of the several studies evaluated, perhaps three studies could be used to represent background conditions. These include one by the Environmental Working Group (EWG), which sampled 20 women from around the United States; the Northwest Environment Watch (NEW) study, which had a sample size of 40 including women residing in several states in the Northwest; and perhaps a third study of 47 samples taken in Texas. The NEW study, which importantly

includes measurements of BDE 209, was used. The median total BDE concentration of the 40 samples was 44.1 ng/g lwt.

The first-order, single-compartment pharmacokinetic model used in this exercise to convert intakes to body lipid concentrations requires the half-life elimination rates of the BDE congeners and BDE absorption fractions (i.e., fractions of BDE intakes from ingestion of dust, water, and foods, as well as inhaled BDEs, that are absorbed in the stomach or the lungs to accumulate in body lipids) as inputs. Only two studies were found that provided human half-lives, with one deriving half-lives ranging from 2.9 to 11.0 yr for BDE congeners 47, 99, 100, 138, 153, and 154. The second reference derived significantly smaller half-lives for the higher brominated congeners BDEs 183 and 209—half-lives of 0.26 and 0.041 yr, respectively. The absorption for BDEs in dust ranged from 0.04 (for BDE 209) to 0.78 (for BDE 100). These were derived from a study of the bioavailability of BDEs in dust when fed to rats. The absorption values for food and water were near 0.90 for all of the congeners. The soil-dermal contact pathway required a fraction of PBDE absorbed through the skin, set at 0.03 for all congeners, as well as parameters reflecting adherence of soil contacted by the skin, contact events, and contact surface areas.

Table E-2 provides the final results from this exposure assessment. As seen there, the exposure pathways of dust ingestion and dust-dermal contact dominated total adult exposure—explaining over 80% of the 540 ng/day total. It is also seen that the congeners BDEs 47, 99, and 209 dominated the exposure intake: each explained about 27% of total intakes, with other congeners explaining the remaining intakes. The predicted concentration of total BDEs of 35.9 ng/g lwt was similar to the NHANES human blood measurement of 36. 4 ng/g lwt and the breast milk concentration of 44.1 ng/g lwt. Predictions appear reasonably close to measurements for 7 of 9 congeners. The prediction of BDE 47 at 10.0 ng/g lwt did not appear to match the observed measurements of 20.5 ng/g lwt in blood and 26.0 ng/g lwt in milk. The cause for the underprediction of BDE 47 is not known, but it could very easily be the assumed half-life in humans. At 3.0 years, BDE-47 was eliminated nearly twice as fast as BDE 99, which was assigned a half-life of 5.4 years. Had it been assigned an elimination half-life of 10 years, the prediction would jump to greater than 30 ng/g lwt, now twice as high as BDE

99, which is in line with measurements. On the other hand, BDE 99 was predicted at 14.6 ng/g lwt, which is higher than measurements of 5.0 (in blood) and 5.4 (in breast milk) ng/g lwt. It is interesting that the calculated intake doses of BDEs 47 and 99 were comparable, but that the observed body burdens of BDE 47 were so much higher than BDE 99. In terms of modeling, BDE 99 was overpredicted while BDE 47 was underpredicted. It was suggested that perhaps this result could be due to improper assignment of elimination rates in the body, but also, this result could suggest debromination of BDE 99 in the body. In other words, the modeling may correctly determine body burdens due to intakes, but the modeling might not account for how these body burdens could change over time due to internal metabolism. However, this is much too speculative given current information on human and animal metabolism.

Exposures for key populations were also examined. These unique exposures included occupational, childhood, infant via breast feeding, and unusually high exposures at the high end of the general population. Findings from these examinations include:

1. Occupational: Limited studies from Sweden and from China suggest that PBDE concentrations are elevated in occupational groups exposed to likely sources of PBDEs. One study, which looked at incinerator workers in comparison to general population exposures, did not find a difference. The only study of occupational exposures in the United States found a significantly higher ($p < 0.05$) level of PBDEs in workers in foam recycling facilities and individuals who installed carpet padding manufactured from recycled foam. Specifically, the median total PBDE concentrations (which didn't include BDE 209) in the foam workers, carpet installers, and control were 160, 178, and 19 ng/g lipid, respectively.

2. Children: Dose intakes for children were derived in a similar manner as adults: exposure media concentrations were combined with contact rates for specific age ranges of children. The total dose for the three age ranges of children modeled were 751 ng/day for the 1-5 age range, 439 ng/day for the 6-11 range, and 536 ng/day for the 12-19 age range. On a body-weight basis, the doses are 50.1 ng/kg-day for ages 1-5 (assuming 15 kg bw), 14.6 ng/kg-d for 6-11 (30 kg), and 9.2 ng/kg-d for 12-19 (58 kg). The much higher dose for the child age 1-5 was due to the doubling of soil/dust ingestion from 50

mg/day for all other age ranges to 100 mg/day. These child exposures compare to the adult dose of 547 ng/day, or 7.8 ng/kg-day, assuming a 70 kg adult.

3. Infants Via Breast Milk: Intakes of PBDEs via breast milk were input into the pharmacokinetic model to predict infant body burdens impacts. The key assumptions for intake include 0.8 L/day breast milk ingestion, 4% fat in breast milk, and the 44.1 ng total BDE/g lipid concentration in breast milk, as described earlier. With these assumptions, the daily the total dose to infants is 1,411 ng/day. Assuming an average body weight of 10 kg for an infant during the months of breast-feeding, a dose is calculated as 141 ng/kg-day. Two assumptions on elimination half-life were used in the pharmacokinetic simulations: one a rapid half-life, on the order of weeks, and the second more typical of the half-lives used for the lower brominated BDEs in adults, on the order of years. A short half-life has been shown to be appropriate for dioxin-like compounds in infants, while adult half-lives for dioxin have been measured in the order of years, which is why these two half-lives were tested in infants. With the short half-life, lipid concentrations were modeled to rise to over 200 ng/g lwt through age 5, to then drop gradually to below 100 ng/g lwt by age 19. When an adult-like half-life of 6 years was used in this infant model, concentrations rose to nearly 200 ng/g lwt by age 1, continued to rise to about 325 ng/g lwt by age 5, to then drop to below 100 ng/g lwt by age 19. Only one study was available with which to compare these results; this study from a family in California was earlier cited because of the finding of BDE 209 in the children. Total PBDE concentrations in the 18-month old toddler rose to above 400 ng/g lwt, and concentrations in the 5 year-old were above 200 ng/g lwt. At the same time, the adult concentrations in the family were near 100 ng/g lwt. While insufficient to provide verification of either modeling assumption on half-life in infants and children, when combined, this ancillary data along with the pharmacokinetic modeling, support the conclusion that infant and childhood body burdens could very possibly be significantly higher than that of adults.

4. Unusually High Exposures in the General Population: The 2003/2004 NHANES results show that the 95th percentile is more than 10 times higher than the median, and most surprising, the maximum found in the survey is 100 times higher than the median. In contrast, the same information was sought on NHANES results of dioxin-like compounds, and it was found that the 95% is about 4 times the median, and the

maximum found is only about 12 times the median. This same trend for PBDEs was found in other blood and adipose tissue surveys, and also in indoor dust studies; that is, that levels at the high end of the distribution appear substantially higher than the median of the distribution. This suggests that unusually high body burdens in the general population could result from exposures to unusually high dust concentrations.

While predictions of adult and even infant body burdens encouragingly are close to observations, uncertainties exist in the intake dose estimates and the pharmacokinetic modeling, starting from development of dose estimates based on limited environmental measurements, to indoor contact rates with house dust, to the pharmacokinetic parameters of absorption and elimination half-life. Contact rates for food/water ingestion and inhalation are fairly well established, and the exposure media concentration summaries suggest similarities among different studies. Therefore, the dose via food/water ingestion and inhalation might be considered reasonably certain, for purposes of this discussion. However, using the pharmacokinetic model, food/water ingestion and inhalation explained less than 20% of the body burden. It was assumed that the remainder of the exposures came from house dust through the pathways of ingestion and dermal contact. Assignment of dust contact rates in combination with concentrations found in housedust lead to exposures that do result in accurate predictions of body burdens. The claim is not made that this "proves" the importance of indoor dust to overall general population exposures. However, circumstantial evidence supporting this modeling are the high concentrations found in United States house dust, particularly in comparison to house dust concentrations from other countries. Specifically, house dust concentrations in European studies were found to be lower than in the United States, by one order of magnitude or more, and one hypothesis was that the difference in European and United States body burdens (European body burdens are much lower than United States body burdens) is due to exposure of Americans to high concentrations of BDEs in house dust. Still, there was no "proof" that contact with house dust explains the majority of body burdens of Americans. Nonetheless, the overall weight of evidence of this exercise supports the finding that the bulk of United States exposures occur in the indoor environment through contact with house dust. The exercise suggests these exposures

account for between 80 and 90% of total exposures, with the remainder due primarily to food ingestion. Nonetheless, more research is recommended to verify these findings and better quantify the uncertainties that have been identified.

Table E-1. Representative exposure media concentrations

Exposure Media	Congener Number									
	28	47	99	100	138	153	154	183	209	Total
Water, pg/l	3.3	42.7	27.6	7.2	0.3	3.9	2.9	4.4	42.3	146.1
Surface soil, ng/g dwt	---	1.9	3.6	0.4	---	5.7	4.8	37.4	15.3	82.3
Indoor dust, ng/g	ND	1857	2352	911	181	243	156	60	2394	8275
Outdoor air, pg/m^3	3	53	51	13	---	4	4	1	25	158
Indoor air, pg/m^3	27	177	79	16	---	5	7	---	121	447
Shellfish, ng/g wwt	ND	3.6	1.2	0.9	ND	ND	ND	ND	ND	5.7
Finfish, ng/g wwt	0.03	0.60	0.17	0.13	0.001	0.02	0.05	0.002	0.09	1.17
Beef, ng/g wwt	0.02	0.05	0.04	0.006	0.0001	0.006	0.004	0.001	0.003	0.13
Pork, ng/g wwt	ND	0.08	0.12	0.015	0.001	0.02	0.01	0.009	0.02	0.28
Poultry, ng/g wwt	0.0002	0.06	0.12	0.03	0.002	0.02	001	002	0.12	0.36
Dairy, ng/g wwt	0.0002	0.03	0.03	0.005	<0.0001	0.004	0.002	0.002	0.04	0.11
Eggs, ng/g wwt	0.0002	0.02	0.04	0.006	0.0001	0.004	0.003	0.0001	0.01	0.09

notes:
The "totals" may include congeners other than those listed and is greater than the sum of the studied congeners; see Chapter 4 for more detail
--- means no data available; ND means data available but not detected; wwt = wet weight

Table E-2. Predicted doses and lipid-based concentrations of BDEs, with predicted concentrations compared with lipid-based measurements in blood and milk

Exposure Doses, Pharmacokinetic Results	Congener Number									TOTAL
	28	47	99	100	138	153	154	183	209	
I. Exposure Doses										
soil ingestion, ng/d	0.00	81.25	102.92	39.86	7.92	10.67	6.86	2.86	104.83	357.17
soil dermal contact, ng/d	0.00	19.50	24.70	9.57	1.90	2.56	1.65	0.69	25.16	85.73
Inhalation, ng/d	0.32	2.15	1.00	0.21	0.00	0.06	0.09	0.00	1.45	5.28
Food & water ingestion, ng/d	1.57	35.30	24.47	8.18	0.14	2.91	1.6	0.85	16.3	92.24
II. Pharmacokinetic Results										
Predicted conc. ng/g lwt	0.3	10.0	14.6	4.2	1.3	4.6	0.8	0.02	0.1	35.9
Observed blood, ng/g lwt	1.2	20.5	5.0	3.9	NA	5.7	NA	NA	NA	36.3
Observed milk, ng/g lwt	1.7	26.0	5.4	5.2	NA	4.8	0.4	0.2	0.4	44.1

Figure E-1. Approach for characterizing exposure to polybrominated diphenyl ethers in this report.
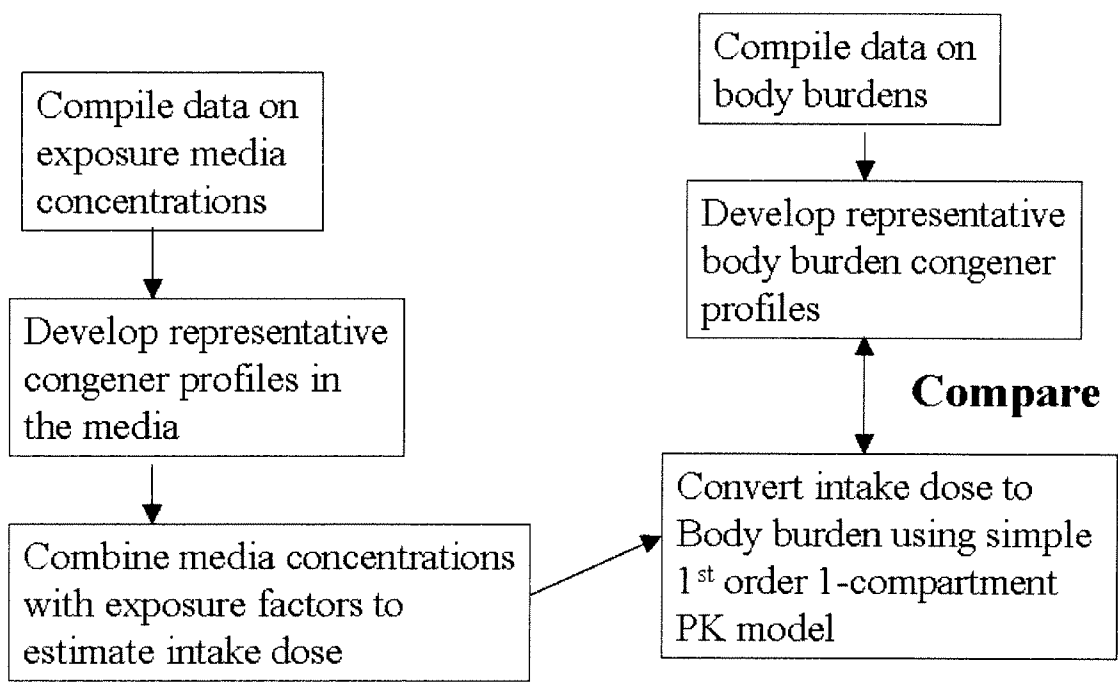
Chapter 1 Introduction

Polybrominated diphenyl ethers (PBDEs) are a class of brominated flame retardants that are added to plastics, polyurethane foam, textiles, and electronic equipment to reduce the likelihood of ignition and to slow the burn rate if the products do catch fire. PBDEs have a common structure of a brominated diphenyl ether molecule that may have anywhere from 1 to 10 bromine (Br) atoms attached. Depending on the location and number of Br atoms, there are a possible 209 PBDE compounds, each termed *congeners*. Each are assigned a specific BDE number (note: in this document, the abbreviation PBDE will be used to denote the class of brominated flame retardants, while BDE will be used in the context of PBDE congeners). For example, there are 42 tetrabromodiphenyl ether congeners (those with 4 bromine atoms), but only a few of them, specifically BDE 47 and occasionally BDE 66, are found in the product formulations and in environmental or exposure media (La Guardia, et al., 2006). Table 1-1 shows the BDE congener number and chemical composition of the most commonly studied BDE congeners.

PBDEs have been marketed in three primary formulations: the pentaBDE formulation, commercially known as DE-71 and Bromkal 70⁻5DE; the octaBDE formulation, DE-79; and the decaBDE formulation, DE-83R or Saytex 102E. The formulations differ in their composition of BDE congeners. The penta formulation is dominated (by weight) by penta congeners (50-62%) with secondary contributions by tetra (24-38%) and hexa congeners (4-12%). The octa formulation is dominated by hepta (45%) and octa congeners (33%), with secondary contributions by hexa (12%) and nona (10%) congeners. The deca formulation is composed of essentially all BDE 209 (97-99%, with 1-3% other, mainly nona, congeners), which is the congener with all ten Br positions occupied. The penta and octa formulations were voluntarily withdrawn from the United States (U.S.) marketplace by their manufacturers at the end of 2004, leaving only the deca formulation for use in commercial products. The deca formula is also the only currently used formulation in Europe because the penta and octa formulations were banned. However, Sweden banned the use of the deca formulation in August of 2006, the

ban to take effect January 1, 2007 (see:
http://www.emfacts.com/weblog/index.php?p=547).

PBDEs have captured the attention of scientists and policymakers because levels
in the environment and humans have increased rapidly since these chemicals came into
use. The rise in PBDE concentrations in blood and breast milk samples both from the
U.S. and Canada throughout the 1990s into the 2000s, coupled with the fact that North
American body burdens exceed those of Europeans and others by factors of 10 or more,
has served to focus attention on North American exposures to PBDEs. The penta PBDE
formulation has garnered the most concern because it appears to be the major contributor
to current environmental and human body levels. Even with both the penta and octa
formulations having been withdrawn from the U.S. market, past use and also the
possibility of debromination (loss of bromine atoms) of BDE 209 and other higher
brominated congeners by photolytic or biological mechanisms to form lower brominated
congeners could result in the continued presence of lower brominated congeners in the
environment.

Studies have been conducted in laboratory animals to gain a better understanding
of the potential health risks of PBDEs. These studies have suggested potential concerns
about liver toxicity, thyroid toxicity, developmental toxicity, and developmental
neurotoxicity—especially about potential risks in children. To date, there is no evidence
of carcinogenicity from exposure to any of the PBDEs except decabromodiphenyl ether,
BDE 209. In a review of toxicological studies as part of a Toxicological Review for the
U.S. Environmental Protection Agency (U.S. EPA)'s Integrated Risk Information System
(IRIS), the U.S. EPA (EPA, 2006) has found "suggestive evidence of carcinogenic
potential" for BDE 209, according to the 2005 EPA draft *Guidelines for Carcinogenic
Risk Assessment* (EPA, 2005).

The U.S. EPA has formed a working group comprised of individuals from several
program offices including the Offices of Pesticides, Prevention, and Toxic Substances,
the Office of Water, and of the Office of Research and Development, to study issues
surrounding polybrominated diphenyl ethers (see http://www.epa.gov/oppt/pbde). They
issued a project plan in 2006 that outlined projects to further study the toxicity,
environmental fate, and exposure to PBDE compounds. This document addresses the

exposure assessment needs identified in that project plan and provides a comprehensive assessment of the exposure of Americans to this class of persistent organic pollutants.

Subsequent chapters describe the historical use and composition of commercial mixtures of PBDEs (see Chapter 2), the environmental fate of PBDEs (see Chapter 3), and environmental and exposure media concentrations of PBDEs (see Chapter 4). The document concludes with a comprehensive exposure assessment addressing infant, children, and adult exposures to PBDEs (see Chapter 5).

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Table 1-1. BDE congener numbers and chemical composition of the most commonly studied BDE congeners

BDE Congener Number	Chemical Formula	BDE Congener Number	Chemical Formula
I. MonoBDE		**VI HexaBDE**	
BDE 3	4-BDE	BDE 138	2,2',3,4,4',5'-BDE
II. BiBDE		BDE 140	2,2',3,4,4',6-BDE
BDE 7	2,4-BDE	BDE 153	2,2',4,4',5,5'-BDE
BDE 8	2,4'-BDE	BDE 154	2,2',4,4',5,6'-BDE
BDE 11	3,3'-BDE	BDE-155	2,2',4,4',6,6'-BDE
BDE 12	2,6-BDE	BDE 166	2,3,4,4',5,6-BDE
BDE 13	3,4'-BDE	**VII. HeptaBDE**	
BDE 15	4,4'-BDE	BDE-181	2,2',3,4,4',5,6-BDE
III. TriBDE		BDE-183	2,2',3,4,4',5',6-BDE
BDE 17	2,2',4-BDE	BDE-190	2,3,3',4,4',5,6-BDE
BDE 25	2,3',4-BDE	**VIII. OctaBDE**	
BDE 28	2,4,4'-BDE	BDE-196	2,2',3,3',4,4',5',6-BDE
BDE 30	2,4,6-BDE	BDE-197	2,2',3,3',4,4',6,6'-BDE
BDE 32	2,4',6-BDE	BDE-203	2,2',3,4,4',5,5',6-BDE
BDE 33	2',3,4-BDE	**VIII. NonaBDE**	
BDE-35	3,3',4-BDE	BDE-206	2,2',3,3',4,4',5,5',6-BDE
BDE 37	3,4,4'-BDE	BDE-207	2,2',3,3',4,4',5,6,6'-BDE
IV. TetraBDE		BDE-208	2,2',3,3',4,5,5',6,6'-BDE
BDE 47	2,2',4,4'-BDE	**VIII. DecaBDE**	
BDE 49	2,2',4,5'-BDE	BDE-209	2,2',3,3',4,4',5,5',6,6'-BDE
BDE 66	2,3',4,4'-BDE		
BDE 71	2,3',4',6-BDE		
BDE 75	2,4,4',6-BDE		
BDE-77	3,3',4,4'-BDE		
V. PentaBDE			
BDE 85	2,2',3,4,4'-BDE		
BDE 99	2,2',4,4',5-BDE		
BDE 100	2,2',4,4',6-BDE		
BDE 105	2,3,3',4,4'-BDE		
BDE 116	2,3,4,5,6-BDE		
BDE 118	2,3',4,4',5-BDE		
BDE 119	2,3',4,4'6-BDE		
BDE 126	3,3',4,4',5-BDE		

2. PRODUCTION, USE, AND LIFECYCLE OF POLYBROMINATED DIPHENYL ETHERS

2.1 INTRODUCTION

Each year, structural and vehicle fires result in several thousand deaths and injuries and billions of dollars in property loss in the United States (U.S. Fire Administration, 2004). Fire safety and prevention is a focal point of public policy. Largely as a result of using fire-resistant materials in the construction of consumer goods, plastics, and textiles, the incidence of fires has decreased 24% over the past 10 years (U.S. Fire Administration, 2004). The use of brominated fire retardants in these materials has played a major role in fire prevention (IPCS, 1994). Brominated fire retardants are in widespread use and are now detected in the tissues of humans and wildlife, in soils, in sediments, and in air. The ubiquitous presence of brominated fire retardants in the environment has heightened concerns for adverse ecological and human health risks. This chapter describes the production, use, and lifecycle of PBDEs. The information in this chapter contributes to the understanding of the pathways leading to environmental contamination.

PBDEs are a class of aromatic compounds intentionally manufactured to retard the combustibility of treated materials. When fire occurs, the PBDE formulations utilize gas-phase chemical reactions that interfere with the combustion process, thus delaying ignition and inhibiting the spread of fire (D'Silva, 2004). These characteristics have promoted the widespread use of PBDEs in textiles, flexible polyurethane foams used in upholstery stuffing for furniture and car seats, electronic components, electrical components, and plastics used in the casings of TVs, PCs and other electronic equipment.

The purpose of this chapter is to describe the production and uses of PBDEs, and to present a basic lifecycle analysis of PBDEs involving estimates of environmental release from production, product use, waste disposal, recycling of electronics and electrical equipment (EEE) materials, and sewage treatment. Section 2.2 reviews the production of commercial PBDE formulations. Section 2.3 surveys the specific uses of PBDEs as fire retardants in a number of plastic resins and finished products. Section 2.4

presents an initial lifecycle analysis that derives estimates of environmental releases of specific BDE congeners based on production, the uses of PBDE-treated products, and the disposal of PBDE-treated products once they become functionally obsolete. Because of significant limitations in existing data, the lifecycle analysis presented in this chapter should be regarded as a *preliminary* or *initial assessment*. That being said, the lifecycle analysis does provide a platform from which distinct observations can be made regarding the potential pathways of environmental releases of PBDE formulations and their BDE congeners into the land, air and water of the United States from production through disposal of PBDE treated materials.

2.2 PRODUCTION

Commercial production of PBDEs began in 1976 (IPCS, 1994). PBDEs have been sold under various trade names but mainly consist of three commercial mixture formulations: pentaBDE, octaBDE, and decaBDE. Each commercial formulation is manufactured through the chemical reaction of bromine with diphenyl oxide and/or diphenyl ether in the presence of an inorganic catalyst (e.g., $AlCl_3$) (ATSDR, 2004). The bromine amount and the time allotted for the chemical reaction control the extent of bromination on the diphenyl ether molecule. The stepwise addition of bromine causes the formation of lower to higher chlorinated PDBE congeners until the total desired amount of bromination is obtained. Figure 2.1 displays the general structure of the PBDE compound. The molecular backbone consists of two phenyl rings interconnected by an oxygen atom. There are 10 positions whereby a bromine atom can substitute a hydrogen atom on the molecule with the possibility of ten homologue groups identified by the preface mono-, di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and decabrominated diphenyl ether. Each congener is indicated by the positional numbering of the bromine atom on the biphenyl rings. The congeners are assigned a number according to the nomenclature of the International Union of Pure and Applied Chemistry (IUPAC).

Commercial formulations are mixtures of PBDE congeners with pentaBDE, octaBDE and decaBDE having a bromine content of about 70.8%, 79%, and 83%, respectively (European Union, 2001; 2002; 2003). Although 209 PBDE congeners are theoretically possible, only a limited number have been detected in commercial flame

retardant products. Table 2.1 is a compilation of the approximate BDE congener compositions of the three commercial formulations. The dominant congeners in pentaBDE (percent weight basis in parenthesis) are BDE 99 (35–50%), BDE 47 (25–37%), BDE 100 (6–10 %), BDE 153 (3–5%), and BDE 154 (2–4%). On a homologue basis, the pentaBDE formulation is dominated by pentaBDE (50–62%) and tetra-BDE (24–38%) homologues, with hexa-BDE amounting to only 4–12% of total mass.

The BDE congeners present in octaBDE formulations are BDE 183 (40%), BDE 197 (21%), BDE 203 (5–35%), BDE 196 (8%), BDE 208 (10%), BDE 207 (7%), BDE 153 (5–10%), BDE 154 (1–5%), BDE 209 (not detected up to 3%), and BDE 190 (1%). Hexa-, hepta-, octa-, and nona-BDE homologues dominate the octaBDE commercial formulations comprising 10–12%, 43–58%, 26–35%, and 8–14% of total BDE content, respectively. With respect to decaBDE, 97–98% of the formulation is comprised of BDE 209, with the remainder being BDE 207 and BDE 208. BDE 209 is the dominant congener, and it is a signature of this formulation.

As a result of mergers and acquisitions, PBDE-fire-retardant formulations are produced by three chemical manufacturers worldwide: Albemarle, Chemtura and ICL Industrial Products. In the United States, all PBDE-fire-retardant products sold are currently decaBDE formulations. Prior to 2005, the industry was more diverse. The Dead Sea Bromine Group (DSBG), based in Israel, was a leading manufacturer of PBDE fire retardants. ICL Industrial Products acquired the DSBG in 2005. Currently the major producer of PBDE products in the United States is the Great Lakes Chemical Corporation, which sold fire retardants under the Firemaster[TM] brand name. The Great Lakes Chemical Corporation merged with Crompton Chemical to form Chemtura. Chemtura produces flame retardant products comprised of decaBDE under the brand names AZUB DB-40, AZUB DB-65, AZUB 2DA-65, and AZUB 3DA-65. Prior to 2005 the Great Lakes Chemical Corporation produced a pentaBDE based product under the brand name DE-60F. DE-60F was replaced with Firemaster-550, a phosphorous-bromine based fire retardant. Firemaster-550 is technically not enriched with PBDE, but the exact active chemical ingredients remain proprietary. Albemarle produces a decaBDE formulated fire retardant under the brand name SAYTEX 102E. Because of increased international regulatory concern regarding the potential for human health and

environmental effects, the PBDE industry voluntary ceased production of penta- and octaBDE on December 31, 2004 (BSEF, 2006a).

Approximately 56,418 metric tons (MT) of PBDEs were consumed worldwide in 2003. Global consumption for prior years was 67,440 (2001), 67,225 (2000), and 40,000 MT (1999). Two-thirds of all PBDEs produced are consumed in the Americas and Asia. In 2001, 83% of all PBDE consumed worldwide was decaBDE, followed by pentaBDE (11%), and octaBDE (6%). Within the Americas, the breakdown of the market demand for deca-, penta-, and octaBDE in 2001 was 74%, 21.5%, and 4.5%, respectively. Prior to 2004, which was when production of these PBDEs ceased; Americans used approximately 95% of the global production of pentaBDE, 40% of octaBDE, and 44% of decaBDE (BSEF, 2006a).

Concerns for environmental persistence, the potential for bioaccumulation into ecological and human food chains, and the widespread occurrence of PBDEs in the environment have precipitated regulatory and voluntary actions to further reduce the use of PBDEs. The sole United States manufacturer of pentaBDE and octaBDE phased out production of these products at the end of 2004. As well, several states enacted individual legislation banning the use of pentaBDE, octaBDE and deca formulations as summarized below.

- As of June 1, 2006 California and Maine prohibited the manufacture, distribution, and processing of products containing pentaBDE and octaBDE flame retardants (CalEPA, 2006; NCEL, 2005).
- As of January 1, 2006, Hawaii, Illinois, and Michigan prohibited the manufacture, processing, and distribution of products containing more than 0.1% of the PBDE formulations pentaBDE and octaBDE (Illinois EPA, 2006; NCEL, 2005).
- Minnesota similarly prohibited the manufacture and uses of penta and octaBDE effective January 1, 2008 (NCEL, 2007).
- Washington State banned the uses of penta and octaBDE and of decaBDE in mattresses effective January 1, 2008 (Washington State, 2007). However, the Washington State regulation does not prohibit the use of decaBDE in residential upholstered furniture, televisions, or computers with electronic enclosures until January 1, 2011.

- Beginning October 1, 2008, Maryland will prohibit the manufacture, processing, and distribution of products containing more than 0.1% of the PBDE formulations of pentaBDE and octaBDE (NCEL, 2007).
- As of August, 2004, New York prohibited the manufacture and processing, but not the sale or use, of pentaBDE and octaBDE (NCEL, 2005).

Internationally, other countries and governmental entities have banned the use of penta, octa and decaBDE. The European Union (EU) banned the marketing and use of pentaBDE and octaBDE effective February 6, 2003 with Directive 2003/11/EC of the European Parliament and Counsel (BSEF, 2006a). At the time, the EU exempted decaBDE from the ban. However, on April 1, 2008 the European Court of Justice (the EU's highest court) ruled that this decision to exempt decaBDE was flawed and ordered the EU to ban the use of the decaBDE in electrical and electronic equipment (European Court of Justice, 2008). Based on the directive of the court, the ban became effective on July 1, 2008. The ban applies to all EU member countries. Australia banned the use and import of pentaBDE effective March 6, 2007 (Australian Government, 2007). However, it is expected that the use of recycled plastics containing PBDE formulations may continue to incorporate these chemicals in the manufacture of new electronics and IT units (Morf et al., 2005).

2.3. USES OF PBDEs

Due to the phase out by the manufacturer, as well as the state-specific bans noted above, penta- and octaBDE are no longer used in products to inhibit flammability. For the most part, decaBDE is not prohibited from production and continues to be used. This section reviews the past uses of pentaBDE and octaBDE, and the current uses of decaBDE.

Prior to the end of production in 2004, approximately 95% of pentaBDE was used as an additive fire retardant in flexible polyurethane foam (FPUF) materials (European Union, 2001). The treated FPUF was used as seat cushioning and backing material for domestic furniture; in bedding mattresses and in cushioning for automobile seats and laminated automobile seat headrests (ATSDR, 2004). The remaining 5% use of

pentaBDE was in the treatment of foam-based packaging materials and carpet padding. Only 7.5% of the approximate 953,000 MT of FPUF produced annually in the United States was treated with pentaBDE (ATSDR, 2004). Typically the pentaBDE was mixed with aromatic phosphate esters in a ratio of 3:1 prior to application to FPUF. Other past uses of pentaBDE were in textile fabrics used in upholstery for furniture and automobile seat covers; epoxy resins used as protective coatings to circuit boards; unsaturated polyesters; paper laminates; flexible polyvinyl chloride used as electrical wire coatings; rubber; paints and lacquers; rigid polyurethane foam; and adhesives. However, pentaBDE was not added to acrylonitrile-butadiene-styrene (ABS)-based plastics used in the manufacture of casings to television sets, computers, hairdryers, and automotive parts (IPCS, 1994; ATSDR, 2004; Rahman et al., 2001).

Mixed with antimony trioxide, octaBDE was primarily used as an additive flame retardant to certain plastics and textiles. Approximately 95% of the use of octaBDE was as an additive flame retardant in the production of ABS-based plastics, with the remaining 5% used as an additive to high impact polystyrene (HIPS), polybutylene terephthalate (PBT), polyamide polymers, polycarbonate, nylon, polyolefin, and phenol-formaldehyde resins (ATSDR, 2004; European Union, 2003; IPCS, 1994). OctaBDE was typically added to ABS at a loading of 12–15% weight of the final product (European Union, 2003). Materials that were treated with octaBDE include ABS plastics in television sets, remote controls, facsimile machines, copiers, wire and cables, personal computers (PC), PC monitors, keyboards, scanners, audio equipment, video equipment, power adaptors, automobile parts, mobile phones, and kitchen appliances.

DecaBDE is used as a general purpose additive flame retardant to a wide array of plastics having many product applications. HIPS, polyethylene (PE), polypropylene (PP), PBT, and unsaturated polyesters (UPE) are common plastics treated with decaBDE (Alaee et al., 2003; BSEF, 2006b). A major use of decaBDE in the United States is as an additive flame retardant to HIPS. HIPS plastics are used in the manufacture of housings and back panels to televisions, in casings to audio and video equipment, mobile phones, remote controls, personal computers and PC monitors. The PE plastics are used in wire and cables to electrical equipment. The PP-based plastics are used in communication cables, capacitor films, building cables, pipes, stadium seats, lamp sockets and holders,

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and kitchen hoods. The PBT plastics are used as connectors in electrical and electronic equipment. UPE is used in building and construction materials as reinforced plastic panels. DecaBDE is also added as a flame retardant to nylon, and to upholstery textiles used in sofas, chairs, and office furniture. The decaBDE-treated nylon is used as connectors in electrical and electronic equipment, circuit breakers, and coils. DecaBDE is added to polymers in concentrations of 10–15%, by weight, and in conjunction with antimony trioxide (Directorate-General Environment, 2005). A significant use of decaBDE in the United States is the fire retardation of the back panels and the HIPS plastic casings of television sets. In 2003, approximately 17,150 MT of decaBDE was used in 28 million television sets sold in the United States (Pure Strategies, Inc, 2005).

2.4. LIFECYCLE OF PBDE

In general, the lifecycle of PBDE, from production to eventual disposal, includes activities that can cause the environmental releases of the chemical. It begins with the production of PBDE commercial formulations, then proceeds to applications to textiles and polymers, to polymer applications in consumer goods, to the use of consumer goods treated with PBDE fire retardants, and to the final disposal of PBDE treated consumer goods as products reach obsolescence. Figure 2-2 portrays the idealized lifecycle of PBDE. Each element of the lifecycle can potentially release PBDEs to environment. The scarcity of information with regard to all opportunities for environmental releases restricts the lifecycle analysis presented in this chapter, limiting what can be said about potential releases during PBDE production, the consumer use of PBDE-treated products, and the disposal of PBDE-treated products.

2.4.1. Production Releases

The EPA's Toxic Release Inventory (TRI) provides data on environmental releases from the production of decaBDE and its use to fire retard in the production of other materials for 2006 (USEPA, 2008a; the latest reporting year). A total of 33.46 MT of decaBDE was released to the air, land and water in 2006. Great Lakes Chemical, the major United States producer of decaBDE, reported a total of 18.1 MT released to the environment. Figure 2-3 displays a summary of fugitive, stack, surface water and land treatment releases for 2006 (top chart on figure). PBDE production waste was also

transferred to chemical waste landfills, bringing the total TRI reported data to 62.8 MT in 2006. However, the disposal of the PBDE waste in permitted hazardous waste landfills is not considered as an environmental release, and, therefore, this production waste was not added to Figure 2-3. Disposal in a permitted hazardous waste landfill is not considered to be an "environmental release," because of the controls in place in RCRA-permitted landfills are designed to prevent offsite migration and contamination of ground water sources. Releases of decaBDE to the air from fugitive and stack emissions accounted for the preponderance of environmental release, ~89% of total releases, from the chemical manufacture of decaBDE and its use in the production of other materials. The discharges to surface water and the off-site land treatment of the waste accounted for 6 and 5%, respectively. The bottom chart to Figure 2-3 displays total environmental releases of decaBDE from production facilities from 1997 to 2006. It appears from the chart that total environmental releases peaked in 1999 (at 53.9 MT) and remained essential the same through 2002. There was a drop in environmental releases in 2003 (to 36.3 MT), followed by an increase to 44.8 MT in 2004. Examination of total releases from 2004 through 2006 suggests that decaBDE releases decreased by about 25% over this time interval (from 44.8 MT in 2004 to 33.4 MT in 2006). It is not known if this signals a continued decrease in subsequent years of TRI reporting. The BDE congener distribution in decaBDE provides a basis for assuming the environmental loading of BDE 209, BDE 207, and BDE 208 that may be associated with United States production in 2006. Table 2-2 displays the assumed BDE congener distribution in the environmental releases associated with production of decaBDE in 2006. It should be noted that TRI data may not represent all releases of PBDEs from all sources and use activities. Therefore, the emissions of PBDE to air, water and land may possibly be underestimated (USEPA, 2008a).

2.4.2. PBDE Content of Consumer Products

The previous section reviewed current releases of decaBDE from primary production facilities in the United States. It is believed, however, that the major environmental releases occur as a function of the use and disposal of products containing PBDEs. The extent and magnitude of such releases is largely unknown, although

attempts have been made to quantify these releases. These quantifications are based on the PBDE content of the product and mathematical modeling based on these concentrations. This section reviews studies measuring the PBDE concentration in consumer products. The next section presents a procedure to quantify releases from consumer products.

An assessment of the penta and octaBDE content of various polymers was conducted by the German EPA (Kemmlein et al., 2005). A total of eight samples, each of acrylonitrile-butadiene-styrene copolymer (ABS) resin, high-impact polystyrene (HIPS), polyurethane hard foam (PUR-H), and epoxy resin (EP) were evaluated using the single-ion monitoring and a gas chromatographic/mass spectrometer system. The polymers were obtained from the manufacturers along with the percent addition of penta- and octaBDE as flame retardants. ABS samples had been treated with 1% pentaBDE and 2.95% octaBDE; PS samples with 2.96% octaBDE; and PUR-H and EP with 2% pentaBDE. Table 2-3 shows the mean and standard deviation of the concentration of penta and octaBDE detected in eight samples of each polymer.

A Swiss study published in 2003 provides a quantitative basis for assigning a plausible PDBE content of final products made from fire retarded plastic resins (Morf, et al., 2005). In this study, waste electronic products and components were sampled at an electronics recycling facility in Bern. The sampled end products included television sets (TV), video camcorders, radios, HiFi stereo systems, portable compact disk players, mobile phones, standard telephones, toasters, and vacuum cleaners. Together, these product lines represented about 90% of the total use of penta, octa, and decaBDE employed as fire retardants in electrical and electronic appliances and equipment (Morf, et al., 2005). Several samples of discarded material were taken from piles of copper cable, printed circuit boards, TV housings and rear covers, and PC housings. The samples from each product were composited, and then analyzed using either gas chromatography or electron capture detection with mass spectrometry. The mean concentrations (mg/kg) of the PBDEs found in each product are shown in Table 2-4.

In addition to the mean concentrations in electrical and electronic equipment (EEE) waste, Morf et al. (2005) determined the abundance of select PBDE congeners in the EEE materials. One sample of each of the following items were taken from the

output piles of sorted materials at the recycling plant: a plastic PC screen housing, a plastic TV-housing rear cover, a fine-grained plastic with particle size 2–5 and 5–10 mm (general EEE waste), a fine-grained metal, a fine particulate, and a printed circuit board. Samples were Soxhlet-extracted and analyzed with HRGC/HRMS. Table 2-5 summarizes the results of the analysis of these single EEE waste material samples. Because these were essentially grab samples, the results in Table 2-5 only serve as a general indication of the potential congener distribution in the EEE waste.

The Consumer Products Safety Commission reported on the chemical fire retardant content of polyurethane foams (PUFs) used in furniture in the mid-1990s (Cobb, 1995). Seven PUF types were evaluated. Three samples of each of the foam types were collected and analyzed by HRGC/HRMS. Only one PUF material contained PBDE (PBDE species not identified). An average of 3% (range 2.9 – 3.2%) PBDE was found in this one PUF. The other samples contained melamine and/or TDCP as fire retardants. Hale et al. (2002) noted in a separate study that the pentaBDE content in PUF can be as high as 30% by weight.

2.4.3. Estimates of PBDE Releases to the Air Based on Chamber Testing

PBDEs are basically fire retardant additives to materials and not bonded chemically to the matrix. This means that there exists a potential for the PBDEs to escape the matrix through the process of volatilization to the air. Thus the products treated with PBDEs may off-gas PBDE within indoor air microenvironments while the product is still in use. Volatilization is the most likely mechanism of release from the product into the surrounding air.

Only one study was found which attempted to measure this possibility of release under laboratory conditions (Kemmlein et al., 2003). Kemmlein et al. (2003) determined PBDE emission rates by placing various products into an enclosed chamber, passing an air stream over the products, and systematically sampling the chamber air over the duration of the experiment. Test chambers consisted of volumes of either 0.02 m^3 or 1 m^3, and a temperature of $23°C$ was kept constant over the experiments. The products tested represented a cross-section of materials commonly used in interior spaces: insulation and assembly foams, information technology (IT) devices, upholstered

furniture, upholstery polyurethane foams, mattresses, and circuit boards. All materials had been treated with PBDE as additive fire retardants. The treated materials were kept in the enclosed chambers for a period of 100 days or longer in order to insure that steady-state conditions were reached prior to sampling. Polyurethane foam was used to passively collect PBDE vapors that had escaped the material matrix. Additionally chamber walls were rinsed with hexane to collect PBDEs that had migrated to the walls for a complete mass balance of PBDE's emitted from the test material.

As part of this chamber experiment, two PC workstations (A and B) were tested in 1 m^3 emission test chambers under operational conditions (Kemmlein et al., 2003). Workstation A consisted of a PC monitor, a desktop computer, a keyboard, a mouse, and a printer obtained from different manufacturers. Workstation B consisted of the same array of computing devices, but the components were obtained from a single manufacturer. Stations A and B were tested in the air chamber for 93 and 150 days, respectively. In PC workstation A, BDEs 47, 100, 99, and 85 were detected in the air at concentrations less than 0.3 ng/m^3. BDEs 47, 100, and 99 were detected in the chamber air of PC workstation B at concentrations of 150, 28, and 61 ng/m^3, respectively. Additionally trace amounts of BDE 153 were detected in both experiments. Testing computer circuit boards in the test chamber yielded the following results (expressed as an emission rate in units of ng/unit/hr): BDE 17 = 0.6; BDE 28 = 1.9; BDE 47 = 14; BDE 66 = 0.4; BDE 100 = 1.3; BDE 99 = 2.6; BDE 85 = 0.1; BDE 154 = 0.1 and BDE 153 = 0.04.

The PC equipment used in these tests was manufactured after 2000. To test the hypothesis that older plastic casings may have emitted significantly more PBDEs, Kemmlein et al. (2003) tested the back panel of a television casing manufactured before 1979. The back panel had been treated with octaBDE. BDEs 28 (maximum concentration 0.5 ng/m^3), 47 (maximum concentration 8 ng/m^3), 66 (maximum concentration 0.24 ng/m^3), 100 (maximum concentration 0.27 ng/m^3), and 99 (maximum concentration 0.84 ng/m^3) were detected in the air to the test chamber. The chamber walls were rinsed with solvent, and the resulting solution was analyzed for the presence of PBDE congeners. BDE 47 and 99 were detected in the rinse, corresponding to a surface concentration of 568 ng/m^2 and 514 ng/m^2 of BDE 47 and BDE 99 respectively. It

was concluded that a significant portion of the BDE congeners 47 and 99 were adsorbed to the chamber wall; therefore, the reported air concentrations of these congeners were likely underestimated. From the analysis of the older plastic back panel an observation can be made that older treated products may continue to emit BDEs into the air after long periods of time (i.e. 20 years or more).

Hazratian and Harrad (2006) observed an apparent association between the ages of PCs used in an office and the indoor air concentrations of PBDEs. They measured PBDE levels in the indoor air of an office when an older and newer PC was used. There were three experimentally conditions: 1) Only a PC built in 1998 was used; 2) A PC built in 1998 and a PC built in 2003 were equally used by employees, and, 3) Only the PC built in 2003 was used by the office employees. In condition (1), the total PBDE concentration in indoor office air was approximately 431 pg/m^3. In condition (2), the indoor total PBDE concentration decreased to 253 pg/m^3. When only the newer PC was used, the total PBDE indoor air concentrations decreased further to 81 pg/m^3.

A study of office buildings confirmed a qualitative association between the presence of PBDE treated products and subsequent levels of PBDEs in indoor air (Harrad et al., 2003). Indoor air with the highest PBDE levels were in rooms in office buildings equipped with numerous desktop personal computers (12 - 16 per room), and numerous PUF-containing chairs (11 - 22 per room). By comparison, the lowest indoor air PBDE concentrations occurred in domestic environments that had no PUF-containing furniture (Harrad et al, 2003).

Rigid polyurethane foam (PUR-R) treated with decaBDE showed no emission of PBDE congeners after 168 day residence time in a test chamber (Kemmlein et al., 2003). PUR-R was manufactured for furniture upholstery stuffing. Thus the investigators reported no detectable brominated organic compounds within the test chamber. Direct analysis of the material showed detectable congeners of deca and nonaBDE in the product.

2.4.4. Estimates of PBDE Releases to Air from Consumer Products

Attempts have been made to estimate the annual amount of PBDE that may be volatilized to the air during the service life of a treated product. The EU has estimated

the theoretical amount of decaBDE, octaBDE, and pentaBDE that could have volatilized into the atmosphere over the European continent during the usage and product life of fire-retardant products (European Union, 2001, 2002, 2003). Equation 1 gives an approximate estimate of the percentage of PBDE that may volatilize over the product life:

$$Percentage\ loss\ due\ to\ volatilization = (1.1 \times 10^{6}) \times V_{p} \times N(\%) \qquad (1)$$

Where:

V_P = vapor pressure of the PBDE flame retardant, mmHg at 21°C
N = service life of the flame retarded product in yrs (assumed to be 10 yrs by the European Union).

Equation 1 was initially developed to approximate the volatilization rate of chemical plasticizers added to plastic films during their product use (European Union, 2002). The EU concluded that the equation should be applicable to the estimation of PBDE loss by volatilization from a solid matrix because the equation emphasizes the vapor pressure of the compound as the controlling factor. Multiplication of the annual tonnage of PBDE treated materials by the annual percent loss rate yields a rough but plausible estimate of PBDE releases to the air (kg/yr) from the treated products. In review of the EU approach, Prevedouoro et al. (2004) commented that utilizing Equation 1 should be appropriate for estimating the outgassing of PBDEs under ambient temperatures from solid matrices where they have been used as additives.

Assuming a vapor pressure of 3.47E-08 mmHg (at 21°C) and a product life of 10 yrs, a volatilization loss rate of decaBDE from the product was calculated by EU as 0.38% over 10 years (or 0.038% per year). Approximately 6,710 MT/yr of decaBDE was used in plastics for all EU countries combined. Based on this quantity, the EU estimated the total losses of decaBDE to the air during the service life of the treated plastic products to be 2.55 MT/year in Europe (6,710 * 0.00038; European Union, 2002). Using a vapor pressure of 4.9E-08 mmHg for octaBDE, the EU estimated a volatilization rate of 0.54% over 10 years (or 0.054% per year) (European Union, 2002). Assuming a 1994 consumption figure of 2,550 MT/yr, the EU estimated that approximately 1.38 MT/yr of octaBDE volatilized from product usage throughout Europe (European Union, 2002). PentaBDE was assumed to have a vapor pressure of 3.5E-07 mmHg, and this yielded an

estimated annual loss rate of 0.39% over a 10 year product life (or 0.039% per year) (European Union, 2001). The EU used a value of 1,100 MT/yr pentaBDE in polyurethane foam products to calculate an annual release rate of 4.3 MT/yr pentaBDE for Europe as a whole (European Union, 2001).

It should be noted that the product service life of 10 years was an arbitrary assumption by the EU, and, when combined with the average amount of PBDE treated materials in use each year, is thought to give a reasonable, but unverifiable, result (Prevedouoro et al., 2004).

Prevedouoro et al. (2004) estimated the volatilization flux of BDE 47 from products treated with pentaBDE and consumed in the United Kingdom. Using equation 1 and applying a high and low BDE 47 consumption rate (74 and 65 MT in the year 2000, respectively), Prevedouoro et al. (2004) calculated the annual mass flux of BDE 47 to air from the use of treated consumer products. Figure 2-4 shows these results. The peak in the air emissions of BDE 47 occurred in 1997 for both the high and low consumption values. BDE 47 emissions in 1997 were calculated to be 31 and 22.5 MT for the high and low consumption rates, respectively.

This chapter uses the EU estimation technique as a basis for calculating possible total volatilization of decaBDE, octaBDE, and pentaBDE from treated products in the Americas and Asia as well. This analysis assumes the following:

1. The market demand of the PBDE formulation for 2001 (the latest year for which statistics are available) is used as a surrogate for inferring the amount used to fire retard products
2. The product life is 10 years
3. Equation 1 is used to estimate the percent of PBDE volatilized from the treated product

In 2001, the market demand for decaBDE was approximately 24,500 MT in the Americas (BSEF, 2006a). The Americas includes all countries in North, Central, and South America. Roughly 80% of decaBDE is used to fire-retardant hard plastics (Pure Strategies, Inc., 2005), with the remaining 20% used in textiles. Therefore, with a total

demand of 24,500 MT, it is estimated that the amount of decaBDE incorporated into new plastic products in the Americas, in 2001, is 19,600 MT. A volatilization loss rate of 0.038%/yr, or 0.38% over 10 years, would result in approximately 7.5 MT decaBDE/yr volatilized from these new products made from the fire retardant plastics. Assuming no additional products were put into the market during that period, this means that approximately 75 MT of decaBDE would have volatilized from plastic products over a 10-year time period in the Americas. There is no further breakdown of decaBDE use by individual country comprising North, Central and South America. If it is assumed that the United States accounts for 80% of the total use of decaBDE produced in 2001, then 15,680 MT may have been used in the United States. It is possible that 60 MT of decaBDE may volatilize over 10 yrs (6 MT/yr) from plastic products in use in the United States. Total decaBDE market demand in Asia in 2001 was 23,000 MT (BSEF, 2006a). Asia includes the countries of China, India, and Japan. Applying the same assumptions as in the estimate for the Americas would result in a volatilization of about 7.0 MT decaBDE/yr (70 MT over 10 years) from plastic products used in Asia. The estimated global market demand for decaBDE in 2001 was 56,100 MT (BSEF, 2006a). An assumption that 80% of decaBDE is used in plastic products translates to an estimate of a global volatilization loss of 17 MT decaBDE/yr or 170 MT over 10 years.

OctaBDE has a higher vapor pressure than decaBDE; hence the rate of volatilization from treated plastic materials will be greater. Applying Equation 1, and assuming a vapor pressure of 4.9E-08 mmHg at 21°C (European Union, 2003), a calculation of annual loss due to volatilization can be made using the 2001 market demand for octaBDE. OctaBDE was primarily used as a flame retardant additive to ABS polymer used in PC casings and monitors (ATSDR, 2004). In the European Union, 95% of the use was to fire retard ABS plastic (European Union, 2003), with the remaining 5% to be used in HIPS, PBT and polyamide polymers. This assumption is used to calculate the rate of loss from ABS plastic. In 2001, approximately 1,500 MT were used in the Americas and 1,500 MT/yr also used in Asia (BSEF, 2006a). In Europe the usage figure was 610 MT. From equation 1, the loss from volatilization of octaBDE during the service life of an ABS plastic product is approximately 0.54% over 10 years (or 0.054% per year). It is estimated that Asia and the Americas might have each emitted 0.77 MT/yr

(7.7 MT over 10 years) of octaBDE into the air from volatilization from ABS plastic products, while, in Europe, approximately 0.31 MT/yr (3.1 MT in 10 years) may have been released. On a global scale, approximately 1.9 MT/yr of octaBDE (19 MT in 10 years) may have been emitted from plastics.

PentaBDE was mostly used as an additive fire retardant in flexible polyurethane foam. The vapor pressure of pentaBDE (3.7E-07 mmHg at 21°C), while low, is sufficient to induce a low rate of volatilization during the life of foam stuffing in furniture and car seats (European Union, 2001). Equation 1 predicts that the rate of loss due to the volatilization of pentaBDE during the 10-year service life of the treated flexible PUF material to be 3.9% (or 0.39% per year) (European Union, 2001). It is assumed that approximately 96% of pentaBDE was used as a fire retardant in flexible polyurethane foam, with the remainder used in rigid polyurethane elastomers for instrument casings (European Union, 2001). The annual mass flux from the flexible PUF to the air can be estimated using the market demand statistics for 2001 (BSEF, 2006b). These statistics show total usage in the Americas, Europe, and Asia to be 6,816; 144; and 144 MT, respectively. Annual emission estimates, assuming volatilization losses of 0.039%/yr, are 26.56, 0.56, and 0.56 MT/yr, (265.6, 5.6 and 5.6 MT over10 years) for the Americas, Europe, and Asia, respectively.

This analysis represents a picture of possible annual emissions from the volatilization of PBDEs from treated products based on a 10-year product life. The analysis does not take into account the introduction of new products treated with PBDEs during this timeframe, but it is consistent with the approach taken by the EU in evaluating potential human health risks associated with exposures to commercial PBDE formulations (European Union, 2003). In this context, the EU has suggested that the estimate of amount volatilized from products is likely an underestimate because the estimates are based on a single year's product use information. Therefore, the EU has indicated that the actual amounts released to air could be one order of magnitude higher than what is predicted by Equation 1 (European Union, 2001, 2002, 2003). In order to compensate for this potential underestimation due to considering only one year of use, the EU increased their calculation of air releases of PBDEs from products in European countries by a factor of 10. A similar factor of 10 could be applied to estimates made

here for the Americas, but given the uncertainties in the procedure (the validity of the empirical equation, the availability of use information before 1999, etc) as well as the fact that pentaBDE and octaBDE were taken off the market in 2004 while other new products could come into the market in future years, no adjustments are made here. In other words, an annual estimate of loss which is really 1/10 the loss over 10 full years of loss from a single year of use (2001 to be precise), will be assumed to generally represent any year – no additional losses are assumed based on introduction of new product each year.

The above calculations presented estimates of releases of penta-, octa- and deca-BDE formulations from the use of treated plastics. The BDE congener specific emissions from plastic products are estimated by multiplying these releases by the congener distributions in the various commercial PBDE formulations (as shown in Table 2-1). Table 2-6 summarizes the estimated emissions of specific congeners based on the analysis presented above. The total global release of BDE congeners (as apposed to the total global release of PBDE formulations) from plastic consumer products is approximately 4.65 MT/yr (46.5 MT over 10 yrs). Of this total, the dominant congeners are BDEs 209 released at 16.67 MT/yr or 35.9% of the total for all congeners, 99 at 28.4%, 47 at 18.7%, 100 at 4.8%, and 153 at 2.7%, with all other congeners released at less than 2% of the total.. The dominance of the congeners BDE 209, 99, 47 and 100 indicates the historical use of deca and pentaBDE in PBDE-treated products. A major caveat to this analysis is that the calculated congener profile assumes the same congener distribution as in the PBDE commercial formulation (see Table 2-1), and the congener mix is volatilized with the commercial PBDE product.

2.4.5. Estimates of the Mass Flow of PBDEs Contained in Electronic and Electrical Equipment (EEE) Waste

*Once PBDE-treated materials have reached their functional life they are discarded in landfills, in incinerators, or they are recycled. This section describes the mass-flow analysis of environmental releases of PBDEs associated with the disposal of PBDE-treated EEE waste comprised of products that have reached their end-life. There is a paucity of information on the amount of PBDE-treated products that may be discarded in any given year. There exists some information on the amount of electronic and electrical

equipment waste contained in municipal solid waste in the United States (USEPA, 2006) In the United States, an estimated 2.4 million metric tons (MMT) of EEE waste were generated in 2005 (USEPA, 2006) and incorporated into municipal solid waste. Of this amount, approximately 300,000 MT (or 12.5%) of selected consumer electronics were recovered for recycling. Selected consumer electronics subject to recycling in the United States include products such as televisions, VCRs, DVD players, video cameras, stereo systems, telephones, cell phones, hand-held electronic devices, personal computers, laptop computers, printers, fax and copy machines (USEPA, 2006). Most, but not all, of these products contain PBDE as a fire retardant (IPCS, 1994). Morf et al (2005) analyzed the EEE waste at an EEE waste recycling facility in Switzerland and found an average concentration of 510 ±35 mg/kg and 530 ±30 mg/kg of decaBDE and octaBDE, respectively. In deriving these mean concentrations in the EEE waste, Morf et al. (2005) specifically studied what they termed "small" electronic waste, including, "small household appliances (e.g., toasters and vacuum cleaners), office and communication appliances (e.g., personal computers and monitors, printers, phones, and fax and photocopy machines), entertainment electronics (e.g., television (TV) sets, videos, camcorders, radios, HiFis, and portable compact disk (CD players), and small size E&E equipment (e.g., plugs and mobile phones)." They justified this selection of EEE waste as the EEE which contained the bulk of brominated flame retardant use in EEE, and analyzed the portions of these products most likely to contain PBDEs, such as electric circuit boards and TV housings. The results from their tests are shown in Table 2.4 and 2.5, and were discussed earlier. Based on a mass balance of the total material input, including parts measured for PBDEs and parts not measured for PBDEs, they calculated total product concentrations, and the average of these total product concentrations are given as 510 and 530 mg/kg for decaBDE and octaBDE, respectively. For purposes of calculating decaBDE and octaBDE content in EEE waste in the United States, the total product concentrations of decaBDE and octaBDE found in the Morf et al (2005) study are assumed to be representative of EEE waste in the United States (as defined by USEPA, 2006). Although not perfect, the composition of EEE waste described and analyzed by Morf et al (2005) is similar enough to the composition of EEE waste in the United States (USEPA, 2006) to permit a rough estimate of the amounts of decaBDE and

octaBDE that may in present in EEE waste in the United States. Using these data, it is estimated that 1,224 MT decaBDE and 1,272 MT octaBDE are contained in 2.4 MMT of EEE waste generated in 2005 (510 mg/kg deca * 2.4MMT and 530 mg/kg octa*2.4 MMT). The recycled portion of the EEE waste is estimated to contain about 153 MT decaBDE and 159 MT octaBDE (3E+05 MT recycled EEE waste * 510 and 530 mg/kg for deca and octaBDE, respectively).

Not all of the EEE waste is recycled in the United States. The remaining 2.1 MMT of EEE material in MSW is either disposed of in landfills or municipal waste incinerators. Of the amount of total EEE waste that is not recycled, roughly 20% is combusted in MSW incinerators and 80% is landfilled (USEPA, 2006). This would mean that 0.42 MMT of EEE waste is incinerated and 1.68 MMT is sent to landfills. Using the mean concentration of decaBDE and octaBDE in EEE waste (Morf et al, 2005) that may have been incinerated or landfilled in 2005 yields the following estimates:

- 230 MT decaBDE in EEE waste incinerated in 2005.
- 239 MT octaBDE in EEE waste incinerated in 2005.
- 857 MT decaBDE in EEE waste landfilled in 2005.
- 890 MT octaBDE in EEE waste landfilled in 2005.

PBDE in landfill leachate: While it is reasonable to assume that some leaching of BDE congeners from landfilling of treated products may occur, only limited information could be found in the literature that could support any estimation of this as an environmental release. For example, Osaka et al. (2004) evaluated the untreated and treated leachate at seven landfills in Japan for the presence of BDE congeners. BDEs in the raw leachate were detected in the following ranges of concentration (pg/L) across the seven landfills: BDE 47 (not detected (ND) – 2,200); BDE 28 (ND – 970); BDE 66 (ND – 3,200); BDE 99 (ND – 1,800); BDE 153 (ND – 27); BDE 154 (ND – 1,200). In the treated landfill leachate, no PBDE congeners were detected, indicating the effectiveness of the leachate treatment process. Odusanya et al (2008) reported on the PBDE distribution in the leachate from five landfills in South Africa. BDE 28, BDE 47, BDE 66, BDE 71, BDE 75, and BDE 77 were regularly detected in raw landfill leachate samples collected from all the landfill sites. No BDE-209 could be detected in any of the

raw leachate samples. The range of concentrations of total PBDEs (pg/L) detected in each landfill were as follows: Landfill 1 (ND–2670); landfill 2 (ND–6638); landfill 3 (ND–7230); landfill 4 (41–4009), and landfill 5 (90–9793). Individual congeners across all five landfills ranged in the following concentrations (pg/L): BDE 28 (100–3,333); BDE 47 (1,469– 9,793); BDE 66 (ND– 4,020); BDE 71 (1,667– 9,459); BDE 75 (743– 7,426); BDE 77 (ND– 4,257); BDE 85 (ND– 1,240); BDE 99 (ND– 5,191); BDE 100 (ND– 2,162); BDE 119 (ND– 5,392); BDE 153 (ND– 875); BDE 154 (ND– 2,176); BDE 183 (ND– 263).

Odusanya et al (2008) and Osaka et al. (2004) give an indication of the ranges in concentration of BDE congeners that may be present in untreated landfill leachate. It should be noted, however, that these studies seem to indicate that PBDE congeners are not expected to be detected in treated landfill leachate. In the United States, MSW landfills are required by federal regulation to collect and treat landfill leachate, and to continuously monitor groundwater for an indication of leachate migration from the landfill (Code of Federal Regulations (40 CFR Part 258) Subtitle D of RCRA: Criteria for Municipal Solid Waste Landfills (MSWLFs)). Therefore, no attempt is made here to estimate the amount of PBDE potentially released into the United States environment from the migration of landfill leachate.

PBDE in incineration emissions: MSW incinerators in the United States have not been characterized for their potential stack emissions of PBDEs. PBDE congeners detected in the stack emissions to MSW incinerators is usually a consequence of not completely destroying PBDEs present in the waste during combustion (Sakai et al, 2006). MSW incinerators typically operate with a combustion efficiency of about 98% (Yang et al 2007), which means that 98% of the PBDE content of the waste is expected to be destroyed during combustion. Previously it was estimated that approximately 230 and 239 MT of deca- and octaBDE may be contained in the EEE waste subject to incineration. These amounts of deca and octaBDE present in the EEE waste should be destroyed by about 98% leaving only 4.6 and 4.8 MT of deca and octaBDE subject to stack emissions. In the United States, strict regulations have imposed highly effective air pollution control devices on MSW incinerators (Federal Register, 1995). The application of dry scrubbers combined with fabric filters on large MSW incinerators has generally

reduced the concentrations of semivolatile organics present in the combustion gases leaving the furnace by an additional 99% prior to emissions from the stack (Federal Register, 1995). The previously calculated uncontrolled emissions of deca and octaBDE would be further reduced by 99% with these air pollution control devices, leaving only 0.046 and 0.048 MT of decaBDE and octaBDE in the stack emissions from MSW incinerators in the United States (4.6 MT decaBDE * (1-0.99) and 4.8 MT octaBDE * (1-0.99).

Incineration of MSW contaminated with PBDE also forms polybrominated dibenzodioxins and dibenzofurans (PBDD and PBDF) in the combustion gases. PBDE is a direct precursor to PBDD/PBDF formation within thermal systems (Weber and Kuch, 2003). The mechanism for this is thought to consist of the intra-molecular elimination of bromine (Br_2) and/or hydrogen bromide (HBr) (Weber and Kuch, 2003). These debromination reactions are enhanced at temperatures above 500°C (Weber and Kuch, 2003), and the cleavage of the Br_2 or HBr from the molecule leads to ring closure to form PBDD and PBDF (Ebert and Bahadir, 2003). The kinetics of the conversion of PBDE to PBDD and PBDF appears to be more favorable for the lower brominated diphenyl ethers (e.g., pentaBDE) and less favorable for the decaBDE (Weber and Kuch, 2003). The destruction efficiency of the PBDE contained in the MSW by controlled incineration ranges from 90 to 99.9%, leaving enough PBDE for the emission of PBDE from the stack and for the formation of PBDD and PBDF (Weber and Kuch, 2003). Weber and Kuch (2003) and Ebert and Bahadir (2003) do not provide information on the efficiency of thermolytically converting the mass concentrations of PBDEs in the waste combusted to the mass concentrations of PBDD and PBDE formed in incinerator stack emissions. Therefore, no attempt is made here to estimate stack air releases of PBDDs and PBDFs during the incineration of MSW.

2.4.6. Estimates of the Mass Loading to Land and Water of PBDEs Present in Sewage Treatment Plant Sewage Sludge and Effluent

Chapter 3 to this report gives an overview of the levels of PBDEs in sewage treatment plant (STP) influent, effluent, and sewage sludge. Table 3-8 to Chapter 3 summarizes these data from various surveys of sewage treatment plants in various

countries. Several references in the Table 3-8 apply to STPs operating in the United States. However, the Palo Alto, California STP studied by North (2004) was chosen from the Table to approximately represent current municipal sewage treatment processes and practices in the United States. Because this was a careful mass balance study of the distribution of PBDEs in sewage sludge and the effluent, it is used here to calculate PBDE mass loadings to land and water for STPs in the United States. The focus of these calculations is the mean BDE congener mass loadings in effluent discharges to surface water, and in the sewage sludge that is applied to land as a soil amendment and fertilizer.

There are 16,519 STPs operating within the United States (USEPA, 2008b). These STPs have a combined daily sewage flow rate of approximately 1.3E+11 L/d (NAS, 2002). Approximately 93% of total daily sewage flow undergoes secondary and/or tertiary treatment before the wastewater is discharged into surface water (USEPA, 2008b), which basically matches up with the treatment processes at the Palo Alto STP studied by North (2004). Applying the factor of 93% to the total daily sewage flow from all STPs combined gives an estimated flow rate of 1.2E+05 L/d for STPs with secondary and tertiary treatment. The secondary and tertiary treatment processes remove solids from the wastewater, concentrates the solids, and generates sewage sludge. Approximately 6.3E+06 MT dw (metric tons, dry weight) of sewage sludge are used or disposed of annually in the United States, of which 3.8E+06 MT dw (60%) is land applied or commercially distributed as fertilizer (NAS, 2002; USEPA, 1999). The remaining sludge is disposed of in the following manner: 1.1 E+06 MT dw are landfilled (17%), 1.4E+06 MT dw are incinerated (22%) and 6.3E+04 MT dw (1%) have miscellaneous uses such as daily landfill cover (NAS, 2002; USEPA, 1999).

North (2004) found that just five BDE congeners accounted for approximately 86 - 90% of total PBDEs detected in effluent and sewage sludge, respectively. These five congeners are BDE 47, BDE 99, BDE 153, BDE 154, and BDE 209. The mean concentrations (μg/kg dw) of these dominant congeners in sewage sludge (Table 3-8, Chapter 3) were: BDE 47 = 757; BDE 99 = 944; BDE 153 = 88; BDE 154 = 68, and BDE 209 = 1,183. The sum of 23 BDE congeners detected in the sludge was 3,381 μg/kg. In the wastewater effluent discharged into surface water after secondary and tertiary treatment, the mean concentration of the congeners were 10.5, 11.2, 0.98, 0.78,

and 1.73 ng/L for BDE 47, BDE 99, BDE 153, BDE 154, and BDE 209, respectively.
The sum of 28 BDE congeners detected in STP effluent at the Palo Alto STP was 29.02
ng/L (North, 2004). BDE 209 dominated PBDE concentration in sewage sludge (at
approximately 35% by wt), however, BDE congeners 47 and 99 accounted for about 36%
and 39% of the total mass of the five main congeners present in treated wastewater
effluent. BDE 209 represented only 6% of the sum of the five congeners present in the
STP effluent. These analyses suggest that sewage sludge is a major sink for BDE 209,
since its concentration was high in sludge but not in effluent.

Sewage sludge is also incinerated at STPs. No inventories of PBDE emissions in
the stack gases of sewage sludge incinerators could be found in the literature. North
(2004) did not stack test the incinerator for PBDEs at the multiple hearth sewage sludge
incinerator at the Palo Alto STP under the assumption that PBDEs in the incoming sludge
would be destroyed within the incineration system by >96%, leaving all PBDE congeners
below the limit of detection. However, PBDDs and PBDFs were sampled in the
incinerator emissions with the assumption that PBDEs entrained in the combustion gas
would be thermolytically converted to PBDDs/PBDFs. Only one homologue group of
PBDFs was detected in the emissions to the incinerator, triBDF. North (2004) calculated
the mass loading of triBDF to be 2.8E-07 kg/yr based on a mean concentration of 82
$\mu g/m^3$ triBDF in the stack emissions.

The following mass loading calculations of BDE congeners from STPs to the
United States environment are made based on mean concentrations of BDE congeners
detected in the sludge and effluent at the Palo Alto STP.

- **Estimated annual loading of BDE congeners to the land from the land
  application of sewage sludge in the Unite States:**

General equation:
*Mass loading PBDE to land (MT / yr) = mean concentration PBDE in sewage sludge
x mass sludge applied to land / yr   (3)* (1)

$$BDE\ 47\ loading = 757\ \frac{ug\ BDE\ 47}{kg\ sludge} \times \frac{1 \times 10^{-9}\ kg}{ug} \times \frac{3.8 \times 10^{+6}\ MT\ sludge}{yr}$$

$$BDE\ 47\ loading = 2.88\ MT\ /\ yr\ to\ land$$

$$(2) \; BDE \;\; 99 \;\; loading \;\;\; = 944 \; \frac{ug \;\; BDE \;\; 99}{kg \;\; sludge} \; \times \; \frac{1 \times 10^{-9} \; kg}{ug} \; \times \; \frac{3.8 \times 10^{10} \; MT \;\; sludge}{yr}$$

$$BDE \;\; 99 \;\; loading \;\;\; = 3.59 \;\; MT \; / \;\; yr \;\; to \;\; land$$

$$(3) \; BDE \;\; 153 \;\; loading \;\;\; = 88 \; \frac{ug \;\; BDE \;\; 153}{kg \;\; sludge} \; \times \; \frac{1 \times 10^{-9} \; kg}{ug} \; \times \; \frac{3.8 \times 10^{-6} \; MT \;\; sludge}{yr}$$

$$BDE \;\; 153 \;\; loading \;\;\; = 0.33 \;\; MT \; / \;\; yr \;\; to \;\; land$$

$$(4) \; BDE \;\; 154 \;\; loading \;\;\; = 68 \; \frac{ug \;\; BDE \;\; 154}{kg \;\; sludge} \; \times \; \frac{1 \times 10^{-9} \; kg}{ug} \; \times \; \frac{3.8 \times 10^{-6} \; MT \;\; sludge}{yr}$$

$$BDE \;\; 154 \;\; loading \;\;\; = 0.26 \;\; MT \; / \;\; yr \;\; to \;\; land$$

$$(5) \; BDE \;\; 209 \;\; loading \;\;\; = 1,183 \; \frac{ug \;\; BDE \;\; 209}{kg \;\; sludge} \; \times \; \frac{1 \times 10^{-9} \; kg}{ug} \; \times \; \frac{3.8 \times 10^{-6} \; MT \;\; sludge}{yr}$$

$$BDE \;\; 209 \;\; loading \;\;\; = 4.5 \;\; MT \; / \;\; yr \;\; to \;\; land$$

$$(6) \; \Sigma BDE_{n-23} \;\; loading \;\;\; = 3,381 \; \frac{ug \; \sum BDE}{kg \;\; sludge} \; \times \; \frac{1 \times 10^{-9} \; kg}{ug} \; \times \; \frac{3.8 \times 10^{-6} \; MT \;\; sludge}{yr}$$

$$\Sigma BDE_{n-23} \; loading \;\;\; = 12.8 \;\; MT \; / \;\; yr \;\; to \;\; land$$

• Estimated annual loading of BDE congeners to surface waters from STP effluent in the United States:

General equation:
Mass loading PBDE to water = mean concentration PBDE in STP effluent x total STP effluent/yr (4)

(1)

$$BDE \;\; 47 \;\; loading \;\;\; = 10.5 \; \frac{ng \;\; BDE \;\; 47}{L \;\; effluent} \; \times \; \frac{1 \times 10^{-9} \; g}{ng} \times \; \frac{kg}{1 \times 10^{-3} \; g} \; \times \; \frac{1.3 \times 10^{13} \; L \;\; total \;\; effluent}{d} \; \times \; \frac{365 \;\; d}{yr}$$

$$BDE \;\; 47 \;\; loading \;\;\; = 498 \;\; kg \; / \;\; yr \; (0.498 \;\; MT/yr) \; to \;\; water$$

(2)

$$BDE \;\; 99 \;\; loading \;\;\; = 11.2 \; \frac{ng \;\; BDE \;\; 99}{L \;\; effluent} \; \times \; \frac{1 \times 10^{-9} \; g}{ng} \times \; \frac{kg}{1 \times 10^{-3} \; g} \; \times \; \frac{1.3 \times 10^{13} \; L \;\; total \;\; effluent}{d} \; \times \; \frac{365 \;\; d}{yr}$$

$$BDE \;\; 99 \;\; loading \;\;\; = 531 \;\; kg \; / \;\; yr \; (0.531 \;\; MT/yr) \; to \;\; water$$

(3)

$$BDE \;\; 153 \;\; loading \;\;\; = 0.98 \; \frac{ng \;\; BDE \;\; 153}{L \;\; effluent} \; \times \; \frac{1 \times 10^{-9} \; g}{ng} \times \; \frac{kg}{1 \times 10^{-3} \; g} \; \times \; \frac{1.3 \times 10^{13} \; L \;\; total \;\; effluent}{d} \; \times \; \frac{365 \;\; d}{yr}$$

$$BDE \;\; 153 \;\; loading \;\;\; = 46.5 \;\; kg \; / \;\; yr \; (0.0465 \;\; MT/yr) \; to \;\; water$$

(4)

$$BDE \;\; 154 \;\; loading \;\;\; = 0.78 \; \frac{ng \;\; BDE \;\; 154}{L \;\; effluent} \; \times \; \frac{1 \times 10^{-9} \; g}{ng} \times \; \frac{kg}{1 \times 10^{-3} \; g} \; \times \; \frac{1.3 \times 10^{13} \; L \;\; total \;\; effluent}{d} \; \times \; \frac{365 \;\; d}{yr}$$

$$BDE \;\; 154 \;\; loading \;\;\; = 37 \;\; kg \; / \;\; yr \; (0.037 \;\; MT/yr) \; to \;\; water$$

(5)

$$BDE\ 209\ loading = 1.73\ \frac{ng\ BDE\ 209}{L\ effluent} \times \frac{1 \times 10^{-9}\ g}{ng} \cdot \frac{kg}{1 \times 10^{-3}\ g} \times \frac{1.3 \times 10^{14}\ L\ total\ effluent}{d} \cdot \frac{365\ d}{yr}$$

$$BDE\ 209\ loading = 82.1\ kg\ /\ yr\ (0.0821\ MT\ /\ yr\ )\ to\ water$$

(6)

$$\sum BDE_{n \cdot 28}\ loading = 29.02\ \frac{ng\ \sum BDE}{L\ effluent} \times \frac{1 \times 10^{-9}\ g}{ng} \times \frac{kg}{1 \times 10^{-3}\ g} \times \frac{1.3 \times 10^{14}\ L\ total\ effluent}{d} \times \frac{365\ d}{yr}$$

$$\sum BDE_{n \cdot 28}\ loading = 1,380\ kg\ /\ yr\ (1.38\ MT\ /\ yr\ )\ to\ water$$

2.5. AN EXAMPLE LIFECYCLE ANALYSIS OF DECABDE IN THE UNITED STATES

This section presents a simplified lifecycle analysis of decaBDE used in plastics and, electronic and electrical equipment (EEE) in the United States. This lifecycle analysis is focused on the production, use, and disposal of plastics and EEE treated with decaBDE. It is noted that all quantities generated in this example are highly uncertain. This example is hypothetical and only meant to demonstrate considerations for, or a basic approach to, a lifecycle analysis. For this example, data and procedures described in previous sections are used.

1. Estimated annual decaBDE demand: 15,680 MT

In 2001 the market demand for decaBDE in the Americas was approximately 24,500 MT (BSEF, 2006a). If it is assumed that market demand is equivalent to annual production, and 80% of the market demand in the Americas was in the United States, then an estimated 15,680 MT decaBDE is annually produced in the United States.

2. Estimated annual releases of decaBDE to air, land and water from the production of decaBDE and decaBDE treated materials: 33.46 MT in 2006 (29.87 MT to air; 1.63 MT to land and 1.96 MT to surface water).

This was the estimate provided for decaBDE production as reported to TRI for the year 2006 (USEPA, 2008a) for releases to air, water, and as waste used in land farming (land application of production sludges). The year 2006 is the latest reporting year as of the date of this analysis. A total of 29.9 MT of decaBDE was released to the air, 0.65 MT released to land, and 1.95 MT discharged to surface water in 2006.

3. Estimate of amount of decaBDE volatilized from in use plastic products: 6 MT decaBDE/yr (60 MT over 10 years of product use).

Applying procedures developed by the European Union (European Union, 2002) for estimating the volatile release of decaBDE from treated plastic products gives an estimated annual release of decaBDE of about 7.5 MT/yr in the Americas. Assuming 80% of decaBDE consumed in the Americas is by the United States gives an estimate of about 6 MT decaBDE released into the air annually (or 60 MT over a 10 year product lifetime) from plastic products currently in use in the United States.

4. An estimated 1,224 MT of decaBDE may be contained in electronic and electrical equipment (EEE) waste generated annually. Of this amount of decaBDE in EEE waste, 857 MT decaBDE is landfilled; 214 MT decaBDE in EEE waste is incinerated and 153 MT decaBDE is contained in recycled EEE materials.

Approximately 2.4 MMT of electronic and electrical equipment (EEE) waste was generated in the United States in 2005 (USEPA, 2006). About12.5% or 300,000 MT of the EEE waste was recycled in 2005. Of the EEE waste not recycled, 20% (0.42 MT) is incinerated, and 80% (1.68 MT) is landfilled. The EEE waste consists of TVs, VCRs, DVD players, video cameras, stereo systems and components, telephones, cell phones, hand-held electronic devices, electronic game devices, personal computers, Wi-Fi devices, laptop computers, LCD displays, printers, and scanners. Morf et al (2005) estimated that EEE waste may contain 510 mg decaBDE per kg EEE waste. These contaminant levels were used to estimate the amount of decaBDE present in the EEE waste.

5. Estimated annual emissions of decaBDE to the air from the disposal of EEE waste in landfills and from incineration: 0.043 MT/yr from incineration.

This calculation assumes the following: MSW incinerators achieving 98% destruction efficiency of the organics in the waste (Yang et al, 2007), and a 99% additional control of PBDE stack emissions is achieved with advanced air pollution control technology (Federal Register, 2005). With these assumptions, it is estimated that 0.043 MT/yr of the 214 MT of decaBDE in the EEE waste is emitted from the stacks of MSW incinerators in the United States.

DecaBDE in EEE waste disposed of in landfills could still volatilize or leach, but no estimates of those avenues of loss could be made. It should be stated that the volatilization route is limited for landfills by the regulatory requirement that landfill operators apply daily cover material over the buried waste under the federal regulations of Subtitle D of the Resource Recovery and Conservation Act (RCRA). Although information was found regarding concentrations of various PBDE congeners in landfill leachate, no decaBDE was detected in these studies.

6. Amount of decaBDE volatilized to air from recycled EEE. No estimate is made.

The estimated amount of decaBDE that may have volatilized from the use of treated products is described in item (3) above. It is presently not known to what extent volatilization of decaBDE may continue to occur from the products comprising recycled EEE materials. Morf et al. (2005) suggested that if volatilization does occur, then it is negligible. However, no attempt to estimate volatilization from EEE waste is made here.

7. Amount of decaBDE released to land and released to water associated with Sewage Treatment Plant (STP) effluent and the disposal of sewage sludge: 0.082 MT are annually released into surface waters; 4.5 MT released to land from the land application of sewage sludge.

In the United States, there are over 16,000 STPs in operation that treat roughly 1.3E+11 L sewage/day. Approximately 93% of these facilities have advanced wastewater treatment processes to include secondary and tertiary treatment. The focus of estimating mass loadings of PBDEs to land from the land application of sewage sludge, and the effluent to surface waters in the United States. The assessment depended on the PBDE congener distribution discerned in the sludge and effluent of an advanced STP in Palo Alto, California that received and treated industrial and domestic wastewater (North, 2004). DecaBDE (represented as BDE 209) represented 35% by wt of the total mass of PBDE detected in the sewage sludge, but only 6% of total PBDEs in the STP effluent. In the United States, approximately 3.8E-10 MT of sewage sludge generated by advanced STPs are annually applied to land. Assuming a mean concentration of 1,183 µg decaBDE/kg sludge from the North (2004) study gives an estimate of 4.5 MT decaBDE

applied to land each year. In the United States, approximately 4.75E+13 L of STP effluent are discharged into United States waters each year from advanced STPs. North (2004) determined a mean decaBDE concentration in the STP effluent to be 1.73 ng/L. With this mean effluent concentration, it is estimated that 82.1 kg (0.082 MT) decaBDE are annually released into surface waters from advanced STPs.

2.6. SUMMARY

This chapter has presented information on the production, uses, and life cycle of PBDE commercial formulations. It should be noted that the production figures are highly uncertain, and no actual amounts of PBDEs used in the United States for the treatment of plastics, textiles, and flexible polyethylene foam could be found. The industry usually aggregates data on a global scale and statistics are available for the Americas, Asia, and Europe (to include the countries of the EU). The industry breakdown indicates that approximately 56,418 MT of PBDEs were consumed worldwide in 2001 (the latest reporting year) of which 83% was decaBDE. The industry voluntarily ceased production of penta- and octaBDE in December, 2004. Today, only decaBDE is being produced in the United States, and the EU countries have banned the use of decaBDE effective April 1, 2008. It is estimated that 15,680 MT decaBDE is currently consumed in the United States. Eighty percent of the decaBDE produced in the United States is used as an additive fire retardant to rigid plastics used in casings to TVs, personal computers, LCD screens and other electronic and electrical equipment (EEE). The remaining 20% are used in textiles such as fabrics used in car seats and other upholstery fabrics, but not in clothing.

The current and past use of PBDE-treated products can result in environmental releases of PBDEs. The aim of this chapter has been to evaluate the amount of PBDEs that could potentially be released from the life cycle of PBDEs, including the release during production, the release from products in use, the release from disposal and recycling of products containing PBDEs, and the releases from the land application of sewage sludge and the effluent surface water discharges from sewage treatment plants that treat industrial and domestic sewage. While there are numerous data gaps in determining these quantities for all PBDEs, this chapter has reviewed available

information on these pathways and highlighted where key information is lacking in an effort to comprehensively evaluate the life cycle of releases from production to use to end disposal. Focusing on decaBDE as an example of lifecycle analysis, the following releases to air, water and land in the United States are estimated, limited by the availability of data:

TOTAL RELEASES TO AIR: 36 MT/yr

Production: 29.9 MT/yr

Product use: 6 MT/yr

Disposal: 0.043 MT/yr (only from EEE waste incineration)

Sewage treatment: No estimate, because sewage sludge incinerators have not been adequately surveyed for decaBDE emissions.

TOTAL RELEASES TO LAND: 5.1 MT/yr

Production: 0.65 MT/yr (land application of sludges)

Product use: No estimate

Disposal: No estimate. Landfilling is not considered an environmental release, because MSW disposal in landfills is strictly regulated to prevent releases.

Recycling: No estimate.

Sewage treatment: 4.5 MT/yr (land application of sewage sludge)

TOTAL RELEASES TO SURFACE WATER: 2. MT

Production: 1.95 MT/yr

Product use: No estimate

Disposal: No estimate, because MSW disposal in landfills is strictly regulated to prevent formation of leachate. DecaBDE not detected in leachate from the few studies that could be found.

Recycling: No estimate

Sewage treatment: 0.08 MT/yr in effluent discharged into surface water from the sewage treatment plants.

TOTAL RESEASES TO THE ENVIRONMENT: 43 MT/yr

From the lifecycle analysis of decaBDE, releases to the environment from production, product use, disposal, EEE materials recycling and sewage treatment plant sludge and effluent total 43.18 MT/yr. From this analysis, approximately 83.33% of total environmental releases are to the air; 4.71% of total releases are to surface water, and 11.95% of total releases are to land (Note: the numbers in this paragraph include up to 4 significant figures. This is not to imply precision in the estimates, but is presented in this manner so that the sum adds to 100%). Sewage sludge appears to be a major sink for decaBDE. It should be noted that municipal solid waste landfills are major environmental reservoirs for decaBDE, and while this analysis presumes there are no environmental releases to air or water, landfills in the United States contain about 857 MT/yr decaBDE as EEE waste.

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Table 2.1. Approximate BDE homologue and congener-specific content of commercial PBDE formulations

Congener Identity	Commercial Formulations of Polybrominated Diphenyl Ether Flame Retardants		
	PentaBDE (Mass % Composition)	OctaBDE (Mass % Composition)	DecaBDE (Mass % Composition)
BDE 17	< 0.1%		
BDE 28	0.2%		
Total TriBDE	0 -1%		0.00001%
BDE 47	25 - 37%		0.00003%
BDE 66	< 1%		
Total TetraBDE	24 -38%		0.00003%
BDE 85	2%		
BDE 99	35% - 50%		0.002%
BDE 100	6 - 10%		
Total PentaBDE	50 – 62%		0.002%
BDE 138	0.5%		
BDE 153	3 - 5%	5 – 10%	0.001%
BDE 154	2 - 4%	1 – 5%	
Total HexaBDE	4 -12%	10 – 12%	0.001%
BDE 183		40%	
BDE 190		1%	
Total HeptaBDE		43 -58%	0.003%
BDE 196		8%	
BDE 197		21%	
BDE 203		5- 35%	
Total OctaBDE		26 - 35%	
BDE 207		7%	2.1%
BDE 208		10%	0.1%
Total NonaBDE		8 - 14%	2 - 3%
BDE 209		0 – 3%	97.8%

This table was composed from information contained in the following references: European Union (2001); European Union (2003); European Union (2002); Kemmlein, et al (2005); Peele (2004); Main Dept of Health (2005); Palm, et al. (2004); IPCS (1994); Peltala and Yla-Mononen (2000); La Guardia et al (2006).

Table 2-2. Estimated environmental releases (MT) of BDE congeners from United States decaBDE production facilities in 2006

Congener	Stack Emissions	Fugitive Emissions	Land Application	Surface Water
BDE 209	27.18	2.03	1.59	1.91
BDE 207	0.58	0.04	0.03	0.04
BDE 208	0.03	0.002	0.002	0.002

Calculations based on BDE congener distribution in decaBDE from Table 2-1.

Table 2-3. Mean concentration (ng/kg) of penta- and octaBDE in various flame retarded polymers

Polymer	PentaBDE	OctaBDE
ABS	1.187 (± 0.058)	0.528 (±0.035)
HIPS	0	1.057 (±0.061)
PUR-H	1.414 (± 0.053)	0
EP	1.414 (± 0.148)	0

Source: Kemmlein et al. (2005). ABS = acrylonitrile-butadiene-styrene-copolymer; HIPs = high-impact polystyrene; PUR-H = polyurethane hard foam; EP = epoxy resin

Table 2-4. Mean concentrations of PBDEs (mg/kg) found in electrical and electronic waste material at a recycling plant in Switzerland

Product	PentaBDE	OctaBDE	DecaBDE
Copper cable	25 (± 10)	100 (± 150)	170 (± 110)
Printed circuit boards	17 (± 7)	10 (± 1)	27 (± 19)
TV housings (wood)	10 (± 4)	10 (± 4)	20 (± 30)
TV/PC housings (plastic)	50 (± 3)	7500 (± 600)	4800 (± 400)
TV housing rear covers	50 (± 20)	7700 (± 3600)	13000 (± 9000)

Source: Morf et al. (2005).

Table 2-5. BDE congener concentrations (mg/kg) present in EEE waste components sampled at a recycling facility in 2002

BDE Congener	PC Screen Housings	Television Housings Rear Covers	Fine Particulates (Dust)	Fine-Grained Plastics (5-10 mm)	Printed Circuit Boards
	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg
BDE 28	0.32	0.32	6.5	0.47	1.3
BDE 47	3.0	2.2	39	5.1	9.6
BDE 99	6.5	3.8	52	7.5	12
BDE 100	0.65	0.28	4.4	0.78	0.72
BDE 153	598	450	31	110	4.0
BDE 154	66	38	6.9	12	1.1
BDE 183	3800	3900	150	690	12
BDE 209	7,300	13,000	760	2,500	89
PentaBDE	13	7.9	120	17	28
OctaBDE	11,000	11,000	420	2,000	34
DecaBDE	7,300	13,000	760	2,500	89

Source: Morf et al. (2005).

Table 2-6. Summary of estimated BDE congener emissions (MT/yr) to air from a 10-yr service life of fire retarded plastic products[a]

BDE Congener	Europe	Americas	Asia	Global
BDE 17	0.001	0.027	0.001	0.027
BDE 28	0.001	0.053	0.001	0.053
BDE 47	0.174	8.241	0.174	8.705
BDE 66	0.006	0.266	0.006	0.281
BDE 85	0.011	0.532	0.011	0.562
BDE 99	0.264	12.494	0.264	13.198
BDE 100	0.045	2.127	0.045	2.246
BDE 138	0.003	0.133	0.003	0.14
BDE 153	0.046	1.121	0.08	1.269
BDE 154	0.026	0.821	0.04	0.901
BDE 183	0.125	0.308	0.308	0.778
BDE 190	0.003	0.008	0.008	0.019
BDE 196	0.025	0.062	0.062	0.156
BDE 197	0.066	0.162	0.162	0.408
BDE 203	0.063	0.154	0.154	0.389
BDE 207	0.07	0.21	0.201	0.495
BDE 208	0.034	0.084	0.084	0.211
BDE 209	2.257	7.273	6.829	16.672
TOTALS	3.22	34.08	8.43	46.51

a/This analysis assumes the market demand for 2001 from BSEF. 2006a.

Figure 2-1. General structure of polybrominated diphenyl ethers.
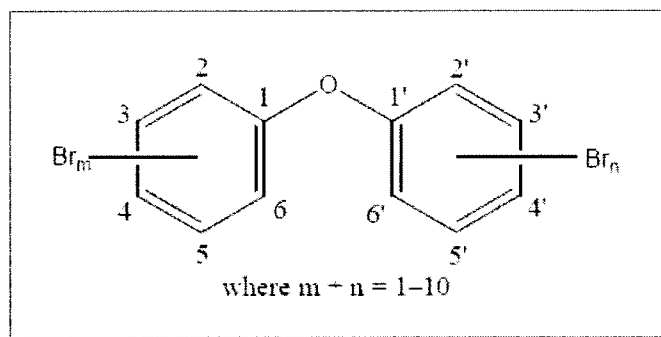
Figure 2-2. Lifecycle of polybrominated diphenyl ethers.
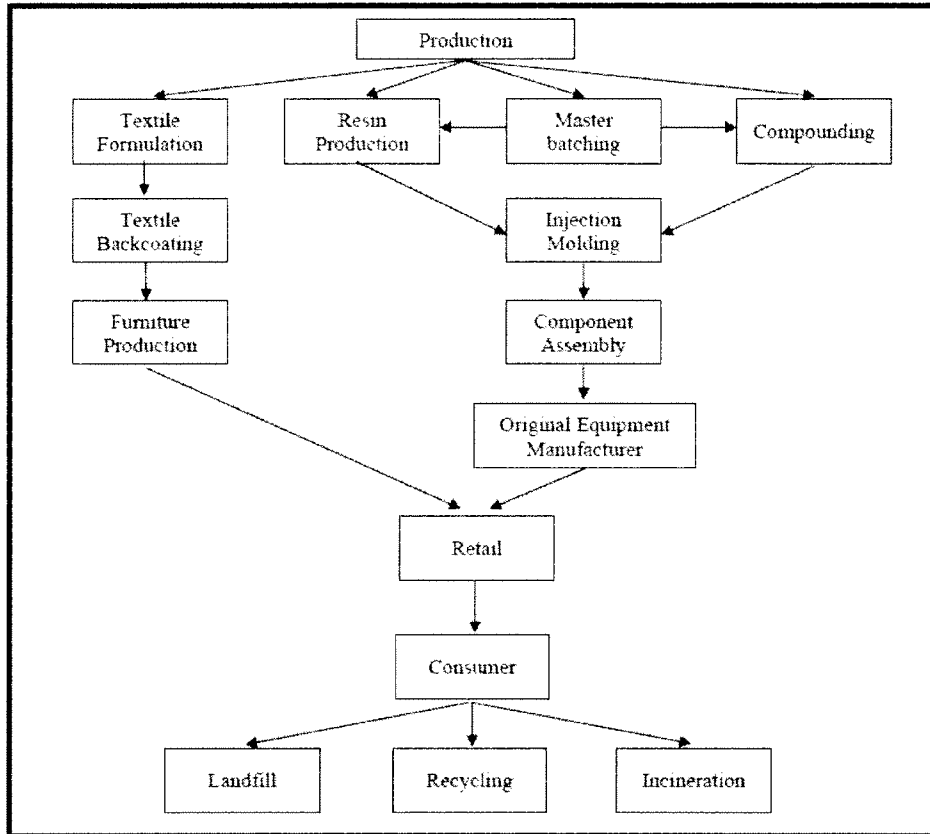
Figure 2-3. TRI data showing environmental releases (kg) of decaBDE from primary production facilities in 2006, and total environmental releases from 1997 2006 (US EPA, 2008a)
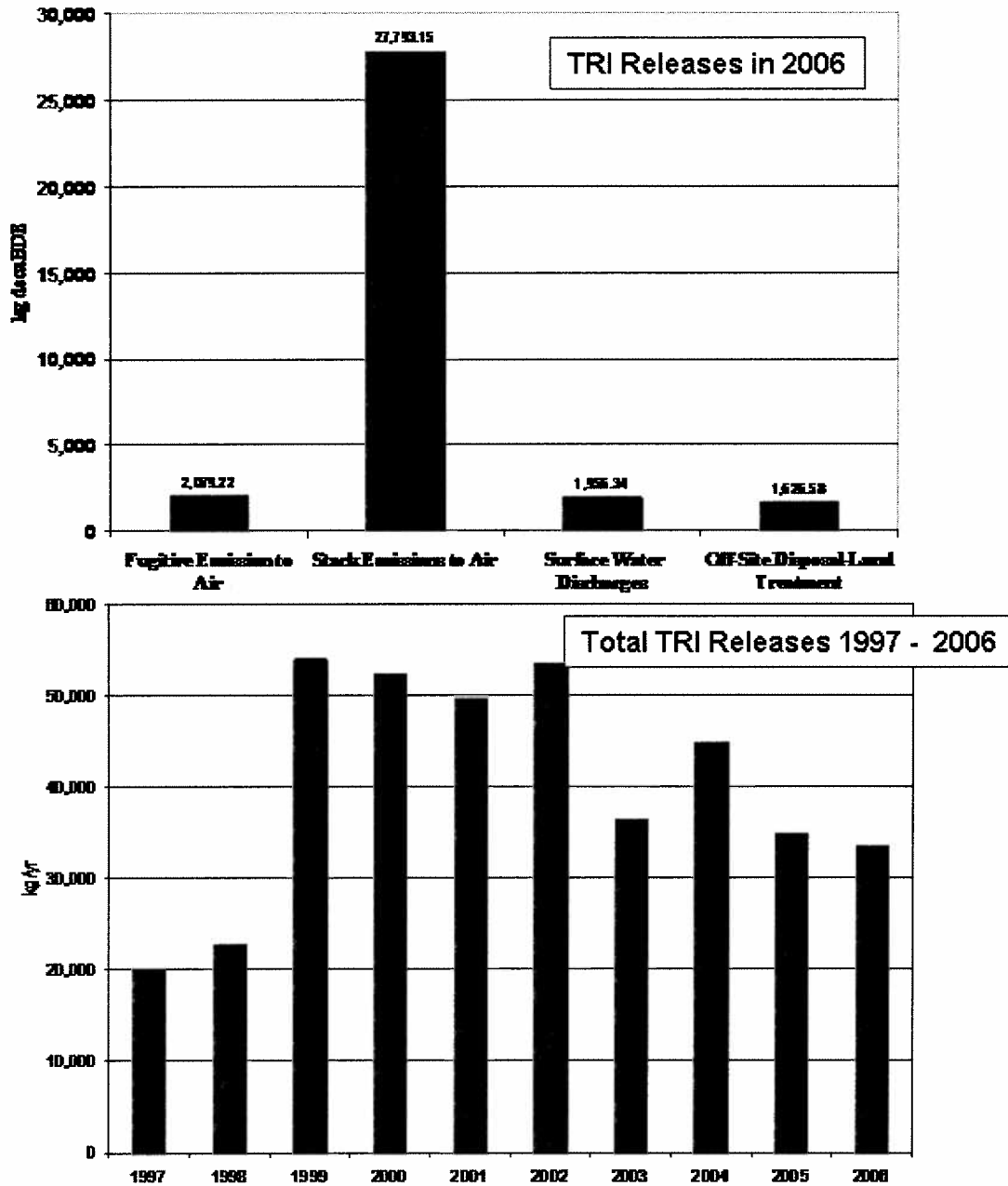
Figure 2-4. Volatilization of BDE 47 from PBDE-treated consumer products in the U.K.
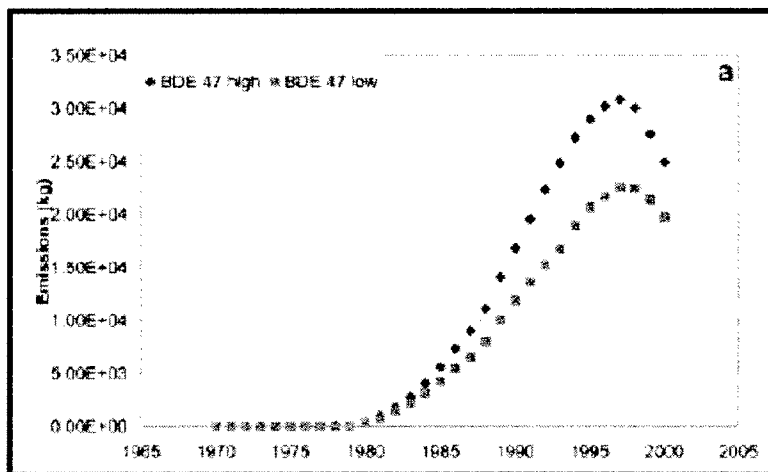
Illustration from Prevedouoro et al. (2004). Assumes an average
Product life of 10 yrs. High and low estimates correspond to range of predicted
consumption rates for the year 2000 (74 and 65 MT, respectively).

3. ENVIRONMENTAL FATE OF POLYBROMINATED DIPHENYL ETHERS

3.1. INTRODUCTION

This chapter reviews the physical and chemical properties important to understanding the environmental fate of PBDEs. Included in this chapter are brief descriptions on how the chemicals move and partition among the environmental media once released into the open environment. The physical and chemical properties control tendencies for PBDEs to move into air, soil, water, and sediments, and to exchange among environmental compartments. They also indicate the physical form and phase of the chemical present in air and water. The physical and chemical properties influence the extent to which biotic and abiotic processes may transform or degrade PBDEs in the environment. Bioconcentration properties indicate the relative propensities of BDE congeners to bioaccumulate and biomagnify in biota and ecological food chains. Overall, the properties most important for understanding the environmental behavior of PBDEs are water solubility (WS), octanol/water partition coefficient (K_{ow}), octanol/air partition coefficient (K_{OA}), the Henry's Law constant (H), and vapor pressure (VP).

3.2 PHYSICAL/CHEMICAL PROPERTIES

The following subsection is a general overview of the physical and chemical properties of the various PBDE congeners. Although there is the possibility of 209 BDE congeners, this section focuses on most of the congeners that comprise the deca-, octa-, and penta-BDE commercial formulations as fire retardants.

3.2.1. Water Solubility (WS)

The chemical parameter of WS describes how readily a chemical compound (referred to as the solute) dissolves in water at a given temperature. This is one of the most important parameters in environmental chemistry. Highly soluble chemicals are easily and quickly distributed within surface water and groundwater. A typical method of estimating the aqueous solubility is by adding an excess amount of the pure chemical to water until equilibrium is achieved and the maximum concentration in water is reached.

Water solubility is typically measured in units of milligram solute per liter of water (mg/L), and at a standard reference temperature (20°–25°C.). The higher the value, the more soluble the chemical is in water. Generally chemicals are considered to have a low, medium, or high aqueous solubility if their WS is ≤ 0.1, 0.1–10, and >10 mg/L (measured at 25°C), respectively (NAS, 2001). Table 3-1 summarizes the WS of commercial PBDE formulations and for some specific PBDE congeners. PBDEs generally have low WS. Organic contaminants with low solubility do not readily dissolve in surface water (relatively hydrophobic) and also typically have high log octanol water partition coefficients, which suggest a high absorption capacity for organic carbon in soils and sediments.

3.2.2. Octanol/Water Partition Coefficient (K_{ow})

Another useful chemical property for indicating how a chemical moves between the aqueous phases and into sediments and biota is the octanol/water partition coefficient, or K_{ow}. The K_{ow} value has become an important parameter in the prediction and understanding of the fate of organic chemicals in the aquatic and terrestrial environments. This property is derived through laboratory experiment and quantitative structure activity relationships to related chemicals, and it is defined as the ratio of the concentration of a contaminant in n-octanol (normal octanol) over the concentration of the same contaminant in water. The n-octanol is intended to generally represent all organic substances. As noted previously, it is related to water solubility in that the higher the chemical water solubility, the lower the propensity for bioaccumulation (the lower the value for K_{ow}). It is expressed as a unitless value. In the experimental derivation of the K_{ow}, octanol is an organic solvent used as a surrogate for organic matter. Although dimensionless, the K_{ow} coefficient is usually expressed as the logarithm, base 10, of the ratio value (i.e., log K_{ow}). In general, organic chemicals with an experimental log K_{ow} coefficient equal to or greater than 5.0 have the property of being very hydrophobic, being tightly absorbed to organic matter, and possessing a high tendency to bioaccumulate. Table 3-2 summarizes log K_{ow} values for brominated diphenyl ethers. All PBDEs tested have high log K_{ow} coefficients, which indicate that they have a high tendency for bioaccumulation.

3.2.3. Henry's Law Constant (H)

Organic contaminants can transfer from water bodies into the air and from air back into water bodies. Henry's Law constant (H) is an air-water partition coefficient, and it is a measure of the chemical's equilibrium distribution between air and water at a specified temperature. In general, H is derived from the ratio of vapor pressure to the chemical's aqueous solubility. Knowledge of H is essential to understanding the direction and mass flux of contaminants transferring from water to air. The rate of volatilization from water to air, and the scavenging of the gaseous phase of the contaminant in air by precipitation (i.e., wet deposition) are governed by H. Usually H is expressed as atm-m^3/mol. Volatilization becomes an important transfer mechanism when the computed H is between 10^{-5} and 10^{-3} atm m^3/mol (Ritter et al., 1995). Chemicals with H values greater than 10^{-3} atm-m^3/mol rapidly volatilize into air. PBDEs are low-volatile organic chemicals with H at 10^{-4} atm-m^3/mol or less, with lower H at higher degrees of bromination. Table 3-3 is a summary of estimated H for PBDEs.

3.2.4. Vapor Pressure (VP)

Volatilization of a chemical and its presence in air is driven by the VP of the chemical. VP also controls the phase distribution of a chemical in air (e.g., the proportion that exists in the vapor and particle-bound phases). The VP is a measure of the force per unit area (i.e., pressure) exerted by a chemical in vapor phase while in equilibrium with its liquid or solid phase at a specified temperature. VP is usually expressed in units of Pascals (Pa) or millimeter of Hg (mmHg). In general, volatile organic compounds (VOC) have a solid phase vapor pressure $\geq$ 10 Pa at an atmospheric temperature of 25° C (Olsen and Neilson, 2001). The semivolatile organic compounds tend to have solid phase vapor pressures < 1 Pa at 25° C. PBDEs are semivolatile organic compounds. Vapor pressures of PBDE decrease with increasing molecular weight and degree of bromination. In a theoretical context, the subcooled liquid VPs of PBDE compounds best represents their tendency to partition between the vapor and particle bound phases in air. Because of this, the scientific literature on chemical and physical

properties has primarily reported values for P_L and not for P_S. However, P_S of the BDE congeners can be calculated from the P_L using equation 3-1 (Paasivirta et al., 1999).

$$\log P_S = \log P_L + (\Delta S_F / R) \times (\ (1 - Tm / T) / 2.3026\ ) \qquad (3-1)$$

Where:

P_S = solid phase vapor pressure, Pa

P_L = subcooled liquid vapor pressure, Pa

ΔS_F = entropy of fusion, ≈ 56 J $k^{-1}mol^{-1}$ (Wania and Dugani, 2003)

R = ideal gas constant, 8.3143 J $K^{-1}mol^{-1}$

T_m = melting point temperature, K

T = reference temperature, K

Table 3-4 summarizes the calculated solid phase (P_S) and subcooled liquid vapor pressures (P_L) of some BDE congeners.

3.2.5. Octanol/Air Partition Coefficient (K_{OA})

The octanol/air partition coefficient, log K_{OA} is the ratio of the concentration of the chemical in air versus the concentration of the chemical in octanol when the octanol-air system is at equilibrium (Harner and Shoeib, 2002). The value of K_{oa} is dimensionless. As with VP, log K_{OA} is temperature dependent. For every 10°C decrease in atmospheric temperature there is a corresponding 2- to 3-fold increase in partitioning of semivolatile organics to the organic phase (Harner and Shoeib, 2002). In broad terms, the log K_{OA} is suggestive of the environmental cycling of semivolatile compounds between the air and organic phases such as soil particles, air particles, and vegetation. The underlying assumption to the log K_{OA} is that n-octanol is a good surrogate for absorption to all organic carbon. The greater the Log K_{OA} (e.g. ≈ 10) the stronger is the propensity of the atmospheric BDE congener to absorb to the organic content of soils and vegetation (Wania, et al, 2002). It is, therefore, a relative indicator of chemical mobility, and the tendency to exchange from the atmosphere to the surface. Log K_{OA} values from 6–11 indicate that atmospheric PBDEs strongly absorb into forest canopies and other

vegetative biomass (Su et al., 2007). Table 3-5 summarizes the octanol/air partition coefficients (log K_{OA}) of PBDE compounds.

3.2.6. Gas-Particle Partitioning In Air

Semivolatile organic compounds present in ambient air partition between the gaseous phase and the particle-bound phase. The physics of this behavior is controlled by the VP of the chemical, the temperature of the surrounding air, and the availability of airborne particulate matter. In terms of environmental fate, the importance of this phenomenon is that the aerosol-bound portion of the contaminant is subject to be transported through the atmosphere over large geographical distances. The wet and dry deposition of the contaminated particles is the most significant mechanism for removal of the particle-bound phase of PBDEs from the air. The gas phase is a good predictor of the air-to-leaf transfer of the semivolatile organic compound and of the possibility for deposition into leafy vegetation, which is integral to the terrestrial food chain. Thus the vapor-phase portion of the contaminant may be more significant in terms of human exposures by way of the dietary pathway.

The fraction of the semivolatile compound that is particle bound (Φ) can be calculated from the subcooled liquid VP using the Junge-Pankow model as indicated in equation 3-2 (Su et al., 2006).

$$\Phi = (c\Theta)/(P_L + (c\Theta)) \qquad\qquad (3-2)$$

Where:

Φ = fraction of the compound adsorbed to aerosol particles

P_L = saturation subcooled liquid phase VP, Pa

Θ = the particle surface area per unit volume of air, cm^2 aerosol/cm^3 of air

c = a constant, 17.2 Pa-cm

Table 3-6 provides the particle bound and vapor phases of the PBDE commercial formulations and BDE congeners calculated from equation 3-2 and assuming the subcooled liquid VP in Table 3-4 and an ambient air temperature of 25°C. The value for Θ in the equation assumes the particle surface area per unit volume air of 1.5E-06

cm^2/cm^3 typical of aerosols in background urban air (Whitby, 1978). The predicted particle-bound phase of the PBDE increases with decreasing VP and increasing number of bromine atoms on the molecule.

By these calculations, >80% of the mono-, di-, tri-, and tetra- and <15% of the penta-, hexa-, hepta-, octa-, and deca-BDE congeners may be present in the vapor phase at an ambient air temperature of 25°C. Greater than 85% of the penta–deca congeners are expected to be associated with airborne particles. As seen in Table 3-6, 72% of BDE 85 is particle-bound, and this appears to be an anomalous calculation due to the fact that Tittlemier et al. (2002) may have overestimated the subcooled liquid vapor pressure at 25°C. About 50–60% of penta-BDE congeners are predicted be present in the vapor phase. These predicted BDE phase distributions in ambient air agree with the observations of Strandberg et al. (2001) for distributions in air over the Great Lakes. Strandberg et al. (2001) found that at 20°C, about 80% of the tetrabromo homologues are in the gas phase and about 70% of the hexabromo homologues are associated with the particle phase. In a study by Chen et al. (2006), the estimated phase distribution of PBDE congeners measured in urban air of southern China was as follows: BDE 28 (97% vapor phase); BDE 47 (80% vapor phase); BDE 66 (77% vapor phase); and BDE 99 through BDE 154 ranged from 50–85% in the particle-bound phase. No ambient air temperature was reported.

3.3. BIOACCUMULATION, BIOCONCENTRATION, AND BIOMAGNIFICATION OF PBDES IN THE AQUATIC ENVIRONMENT

Bioaccumulation describes a process whereby an organism acquires a body burden of a chemical in relation to contact through all possible pathways of exposure (i.e. dietary absorption, transport across the respiratory surface, dermal absorption, and inhalation; Gobas and Morrison, 2000). Bioaccumulation for an aquatic organism occurs from contact of the organism with a chemical contaminant in the water column, the sediments, and through the organism's food chain. The parameter that is often used to model this entire process is the Bioaccumulation Factor (log BAF). This contrasts the Bioconcentration Factor (BCF), which has been used to measure the accumulation from the water column only. In the case of lipophilic and hydrophobic chemicals,

bioaccumulation into fish becomes important in terms of human fish consumption. The log BAF is derived as a logarithm of the ratio of the concentration of a chemical in the tissue of an aquatic organism over the concentration of the chemical in water, in a real aquatic setting. A laboratory-derived BCF entails an experimental setup where the accumulation from the water column only is determined from the concentration of the contaminant in the water tank divided by the concentration in the fish tissue. The log BAF is expressed in units of liters per kilogram of tissue, and can be normalized to apply to wet weight, dry weight, or percent lipid of the organism. The biomagnification factor (BMF) is the ratio of the concentration of the chemical in an organism to the concentration of the chemical in the diet of the organism. The BMF is an indication of increases in concentration of the chemical as it moves up trophic levels in the aquatic ecosystem.

Only a few studies could be located in the scientific literature that has estimated BAFs for PBDE congeners. Table 3-7 summarizes BAF and BCF for various aquatic species.

The United Nations Environmental Program (UNEP) established a screening level for assuming high potential for bioaccumulation of the contaminants into aquatic species (WWF, 2005). The criteria for a high potential for bioaccumulation is that the BCF or BAF in aquatic species for the chemical is greater than 5,000 (log BAF = 3.7). With the exception of BDE 85, it appears that the tri-, tetra-, and penta-BDE congeners exceed these criteria, and, therefore, have a high potential for bioaccumulating in aquatic organisms. BDE 209 has a very low potential for bioaccumulating within the aquatic food web.

3.4. BIOTIC AND ABIOTIC DEBROMINATION AND TRANSFORMATION OF PBDEs

Certain biotic and abiotic processes can transform PBDEs in the environment. The processes most important to the breakdown of PBDEs include biodegradation, biotransformation, and photolysis. Biodegradation involves the breakdown of PBDEs by aerobic and anaerobic microorganisms into smaller compounds. The microbial organisms transform the contaminants through metabolic or enzymatic processes.

Biotransformation is the conversion of the chemical structure of the PBDEs through metabolic pathways. Similar to biodegradation, the reaction is catalyzed by enzymes, but the process occurs in vivo in animals. Photolysis involves the breakdown of PBDEs by the action and the energy of sunlight. All of these environmental fate processes can involve the stripping of bromine atoms from the molecule, a process referred to as 'debromination.' With debromination, the higher brominated BDE congeners can breakdown to form lower brominated species. This section will focus on each of these degradation pathways. It should be stressed that there is only limited scientific information supporting the action of these pathways in degrading, transforming, and debrominating PBDEs.

3.4.1. Microbial Degradation of PBDEs

In theory, microbial communities can degrade and transform organic contaminants present in soils and sediments. This occurs because the microorganisms use the contaminants as a source of carbon (NAS, 1993). Carbon is a building block in the development of new cells during reproduction and growth. In addition, the microbes can extract electrons from the organic contaminant to obtain energy. The microorganism gains energy by breaking chemical bonds and transferring electrons away from the contaminant (NAS, 1993).

One variation of microbial degradation of organic compounds is reductive dehalogenation. In this process, the microbes catalyze reactions that promote the replacement of a halogen atom with a hydrogen atom on the organic compound. This is the primary microbial degradation pathway for higher molecular weight PBDEs. Although the higher brominated congeners are debrominated aerobically, the lower molecular weight congeners that are products of this process may be further debrominated by aerobic bacterial degradation via oxidative dehalogenation (Kim et al 2007).

Several recent studies have provided evidence of microbial mediated reductive debromination of decaBDE and octaBDE under laboratory conditions. Research is currently focused on anaerobic microbial strains that have demonstrated the ability to dehalogenate organic compounds in vitro. In particular, *Dehalococcoides ethenogenes*

195, *Dehalococcoides sp.* strain BAV1, and *Sulfurospirillum multivorans* can dechlorinate a broad range of chlorinated compounds including trichloroethylene (TCE), perchloroethylene (PERC), chlorobenzenes, PCBs, and PCDDs (Fennell et al., 2004; Wu et al., 2002), and, therefore, are being investigated for their potential to debrominate PBDEs (He et al., 2006).

Sulfurospirillum multivorans bacterium incubated with deca-BDE has induced reductive debromination of deca-BDE in vitro to yield octa-BDE and hepta-BDE after a contact time of 2 months (He et al., 2006). The octa- and hepta-BDE did not further debrominate in the presence of the microbe—even after one year. Thus the microorganism was specific to the degradation of deca-BDE and is incapable of debrominating lower brominated PBDE compounds. The organism normally has an affinity for TCE, but when deca-BDE was dissolved in TCE and exposed to *s. multivaornas*, reductive debromination did not occur. However TCE was completely dechlorinated to *cis*-DCE.

Dehalococcoides ethenogenes 195 bacterium was experimentally tried in an attempt to debrominate octa-BDE (He et al., 2006). *D. ethenogenes* of the 195 strain contains reductive dehalogenase (RD) genes and is the only bacterium known to reductively dechlorinate tetrachloroethene and trichloroethane to ethane (Seshadri et al. 2005). *D. ethenogenes* strain 195 was initially isolated from an anaerobic sewage treatment plant (STP) digester containing sewage sludge (Seshadri, et al. 2005). When incubated for 6 months with octaBDE dissolved in TCE, the bacterium showed marked debromination of octaBDE to yield penta, hexa, and heptaBDE congeners (He et al., 2006). However, when incubated with octaBDE without the TCE solvent, no debromination occurred. The authors combined a number of *Dehalococcoides* species in a product they called ANAS195 and incubated it with octa-BDE dissolved in TCE for 12 months. One hundred thirty nMols octaBDE comprised of BDE congeners 153, 183, 196 203, 207, and 208 added to the culture yielded 11.5 nMoles of combined BDE congeners 47, 49, 99, and 154.

When normalized on a cell-count basis, the ANAS195 culture had a rate of debromination of octaBDE that was twice the rate of *D. ethenogenes* 195. An additional strain of *Dehalococcoides species, Dehalococcoides sp* strain BAV1, is the only known

microorganism that dechlorinates the lower chlorinated organic compounds (e.g., dichloroethane, to ethane; Krajmalnik-Brown et al., 2004), and, therefore, He et al. (2006) investigated this species for the potential to further debrominate tetra- and penta-BDE congeners. The experiment showed that bromines in the 2 (*ortho*) and 4 (*para*) positions on the PBDE molecule (e.g., BDE 99 and 47) were the most resistant to microbial reductive debromination.

In another study, Gereck et al. (2005) debrominated BDE 209 *in vitro* to yield BDE s 206, 207, and 208 by contact with anaerobic mesophilic microorganisms indigenous to raw sewage sludge (microbial species not identified). Methane was formed as a product of microbial respiration, and the amount of BDE 209 decreased by 30% within 238 days. This disappearance rate of BDE 209 was statistically significant (p =0.037), and it corresponded to a pseudo first-order degradation rate constant of $1x10^{-3}$ per day, which is equivalent to a half life of approximately 690 days. Debromination of BDE 209 was evident in the subsequent formation of nonaBDE and a number of unresolved octa-BDE congeners. The removal of bromine atoms in the *para* and *meta* positions on the BDE 209 molecule resulted in the formation of BDE 208 and BDE 207, respectively. These products were formed at concentrations that were 5% of the initial concentration of BDE 209. Although BDE 206 was observed in the experiment, it could not unambiguously be concluded that it was a product of microbial degradation of BDE 209.

Rayne et al. (2003a) demonstrated the *in vitro* anaerobic microbial reductive debromination of BDE 15. A non-differentiated anaerobic bacterium from the *in situ* remediation of contaminants in a polluted river system was colonized by passing contaminated water through a bioreactor. Reductive debromination of BDE 15 occurred in vitro at hydraulic retention times of 3.4 and 6.8 hours in a fixed-film, plug-flow biological reactor. The products formed were BDE 3 (4-monoBDE) and non-substituted diphenyl ether.

3.4.2. *In Vivo* Metabolic Debromination in Animals

Once absorbed into an organism, metabolic processes can break down PBDEs. There is a growing body of scientific evidence to suggest that certain freshwater and marine fish species and marine mammals are capable of metabolically debrominating PBDE congeners in vivo. This section is an overview of available information on debromination in wildlife.

3.4.2.1. *Evidence for Debromination in Fish*

Certain fish species have the capacity to debrominate PBDE congeners in vivo. This involves the removal of bromine atoms in the *para* and *meta* positions of a higher molecular weight PBDE congener to form lower brominated PBDE compounds. Debromination appears to mainly transpire in the gastrointestinal tract and fish liver, and catalyzed by one of three possible pathways: the deiodinase (DI) thyroid hormone regulating enzymes (Stapleton et al., 2006a; Tomy et al., 2004), endogenous microbial activity in the gut (Stapleton et al., 2004), and the enzymes of the microsomal monooxygenase system (Stapleton et al., 2004). To date, no one single pathway has been experimentally elucidated. It is not known whether all fish have the metabolic capacity to biotransform PBDEs, nor is it known whether the rate of debromination PBDEs varies among fish species.

Laboratory studies of rainbow trout, lake trout, and carp, involving fish food spiked with pure BDE 209, have clearly shown accumulation of lower brominated BDE congeners not initially present in the feed. This evidence is suggestive of metabolic synthesis of lower brominated congeners through debromination of BDE 209 (Stapleton et al., 2004, 2006a, 2006b; Tomy et al., 2004; Kierkegaard et al., 1999).

The suggestion of metabolic debromination of decaBDE in fish was initially observed in the late 1990's in an *in vivo* rainbow trout study (Kierkegaard et al., 1999). In this study, rainbow trout were fed fish food spiked with 7.5–10 mg of decaBDE/kg of body weight/day for 16, 49, or 120 days. Muscle tissues and livers from exposed fish and controls were analyzed separately for PBDE. Preferential accumulation of PBDE in the liver was observed over the 120-day-study period. Due to a lack of pure analytical

standards for the BDE congeners, it was not possible to identify specific BDE congener present in the fish; only analysis of homologue groups was possible. However, hexaBDE and the first eluting octaBDE congeners were identified as possible products of metabolic debromination of decaBDE, because these congeners were absent in both the spiked fish food and the control fish. This early suggestion of the potential for debromination of PBDEs to occur in fish prompted further in vivo studies.

Tomy et al. (2004) investigated the capacity of juvenile lake trout to debrominate a broad range of BDE congeners. The fish were exposed in aquaria to known amounts of thirteen BDE congeners spiked onto fish food. The BDE congeners included 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, 190, and 209. The duration of dietary exposure to the spiked food was for 56 d, followed by 112 d of depuration. Five fish were sacrificed and tissues were sampled on days 0, 7, 14, 28, and 56 of the period of feeding and on days 7, 14, 28, 56, and 112 of the depuration period. The strongest evidence from this study for the debromination of higher molecular weight BDE congeners in lake trout comes from the comparison of patterns of BDE congeners present in untreated fish tissues to congeners present in the spiked fish food. The presence of one penta (an unidentified congener) and two hexaBDEs (BDE 140 and an unidentified hexaBDE) in the fish tissues served as an indicator of the occurrence of in vivo debrominaization, because these BDE congeners were absent in both the fish food and in the tissues from the control group. It was proposed that BDE 140 was directly formed from the metabolic debromination of BDE 209. Evidence for this is that the bromine substitution pattern of BDE 140 could not have been derived from the substitution pattern of the other two higher brominated congeners tested (e.g., BDE 190 or BDE 183).

Common carp that were fed a diet spiked with either BDE 99 or BDE 183 for 62 days showed significant in vivo debrominazation of both BDE congeners (Stapleton et al., 2004). Six experimental and two control groups (12 fish to a group) were fed spiked or clean fish pellets, respectively. One fish from each tank was sampled on days 0, 5, 10, 20, 30, 44, 62, and during 37 days of depuration following dietary exposure to BDEs. The gastrointestinal tissues and liver was dissected from the fish and analyzed for BDEs as was the remaining homogenized fish sample. BDE 99 was debrominated within carp tissues (gastrointestinal tract and liver) to BDE 47 at a rate of $9.5\% \pm 0.8\%$ of the BDE

99 dietary concentrations. It is interesting to note that BDE 28 was detected in low concentrations in carp tissues from exposure days 20 to 62, suggesting that BDE 47 was further debrominated, although this was not proven. BDE 183 was debrominated to BDE 154 and another unidentified hexaBDE congener. The rate of biotransformation to hexaBDE congeners was estimated to be 14.2% ± 5% of the BDE 183 dietary concentrations. The site of debromination was the intestinal tract. The authors postulated that the metabolic debromination of BDEs 99 and 183 could be mediated by either intestinal microflora, and/or by endogenous enzyme systems such as the hepatic monooxygenase enzymes.

Stapleton et al. (2006a) provided additional confirmational evidence of metabolic debromination of BDE 209 in fish. Sixty juvenile rainbow trout were randomly placed in aquaria holding 15 fish each (with one aquarium containing a control group). The exposed fish were fed fishmeal spiked with 939 ± 14 ng BDE 209/g wet weight for a period of 5 months, which is equivalent to a dose of 9.4 µg BDE 209/kg body weight/day. After 5 months of exposure, the fish were sacrificed and fresh blood was drawn from the dorsal aorta. Livers were removed and analyzed for PBDE congeners and assayed for microsomal activity. Approximately 401 ± 68 ng BDE 209/g (wet weight) PBDEs accumulated in the fish liver during the experiment, indicating that the liver was the primary accumulating tissue. In the pooled serum, collected prior to the experiment, it was determined that BDE 209 averaged <2.4 ng/g serum, whereas during the experiment, the concentration in pooled blood ranged from 26–40 ng/g serum. Steady-state serum concentrations were achieved in the last two months of the experiment. Debromination was observed with the appearance of lower brominated congeners that increased in concentration in the fish tissues throughout the exposure period. Three nonaBDE, six octaBDE, and four heptaBDE congeners were present in the exposed fish tissues that were not present in the spike food mixture. High resolution GC/MS confirmed the identification of the following BDE congeners in the fish tissue: 188, 201, 202, 207, and 208. While BDE 207 and 208 initially increased in tissue concentration, concentrations decreased over the last 2 months of the exposure period. The authors speculated that the BDE 207 and 208 continued to be metabolized, albeit, the study did not identify specific metabolites. A mass balance by Stapleton et al. (2006a)

indicated that debromination of BDE 209 to less brominated congeners occurred in fish liver and the metabolites were subsequently transported throughout the body by means of the circulatory system.

In summary, four major fish feeding studies have clearly demonstrated the potential for *in vivo* debromination of PBDEs. The higher brominated BDE 209 and the decaBDE commercial formulation have been successfully debrominated through a metabolic pathway in fish. The exact mechanism remains unknown. Current theory suggests that enzymes involved in thyroid hormone catabolism, as well as hepatic enzymes, may play a role in the biotransformation of the higher brominated species to lower brominated congeners. It is not clear whether all fish species, both marine and freshwater, have the innate propensity for biotransforming PBDE, but debromination of PBDEs has been observed in juvenile rainbow trout, juvenile lake trout, and common carp (all of which are freshwater species). It seems that bromine atoms in the *meta* positions, positions 3 and 3', on the BDE molecule are most easily removed.

3.4.2.2. Evidence for Debromination in the Rat

In vivo debromination of PBDEs has been observed in a rat feeding study. Huwe and Smith (2007) found evidence of the metabolic debrominazation of BDE 209 in male Sprague-Dawley rats fed a commercial formulation of deca-BDE (98.5% BDE 209). Other congeners detected in the formulation included nonaBDEs, octa-BDEs, and a trace of BDE 183. Deca-BDE was mixed in corn oil to a concentration of $18.9\mu g$ of BDE 209/mL of oil. Eighteen rats were fed an oral, daily dose of 3.8 μg in 200 μL of oil/rat (equivalent to 0.3 $\mu g/g/d$ of the total diet) for a period of 21 days. A control group was fed the standard rat diet over the same period. Experimental rats were sacrificed in groups of three on days 0, 3, 7, 10, 14, and 21 following the cessation of dosing with BDE 209. Prior to sacrifice, daily samples of urine and feces were collected from all rats. After sacrifice, samples of the blood (plasma), the liver, the gastrointestinal (GI) tract, and the remaining carcass were collected from each rat, and homogenized prior to sample analysis by an isotope dilution GC/MS method. BDE 209, nonaBDE, and octaBDE congeners were found to accumulate in the rat liver of the dosed animals at amounts that were 2–3 times higher than in other tissues. Evidence of metabolic debromination of

BDE 209 to lower congeners was observed from an apparent 160% increase in the tissue concentration of BDEs 197, 201, and 207 as compared to levels in the feed. Huwe and Smith (2007) postulated that the possible formation of BDEs 197 and 207 resulted from removal of bromine atoms from the *meta* positions on the BDE 209 molecule. The formation of BDE 201 was postulated to occur from the debromination along *para* and *meta* positions of the BDE 209 molecule.

The rat may serve as an indication of the possibility that metabolic debromination of BDE congeners occurs generally in mammals including humans. However, this remains to be proven.

3.4.2.3. *Evidence for Debromination in Birds*

There is evidence for the metabolic debromination of higher brominated PBDEs in chickens (Pirard and Pauw, 2007) and starlings (Van den Steen et al., 2007). Pirard and Pauw (2007) fed seven Sexaline hens with a diet containing 3.4 mg/kg penta-BDE formulation (De-71, Great Lakes Chemicals) for 14 weeks. Egg samples and daily excreta samples were collected during the experiment. At the end of 14 weeks, the hens were sacrificed and samples of fat and liver were taken for chemical analysis. With regard to tissue and egg distributions, 3,030, 3,711, and 2,826 ng/g lipid adjusted total PBDEs were detected in the liver, adipose tissue, and eggs, respectively. Pirard and Pauw (2007) derived BCFs for select BDE congeners by dividing the congener concentrations detected in abdominal chicken fat to the congener concentrations in the chicken feed. The estimated BCFs were as follows: BDE 47 = 0.7; BDE 100 = 1.8; BDE 99 = 0.6; BDE 154 = 2.2; BDE 153 = 2.0; and BDE 183 = 1.0. The authors investigated absorption/excretion percentages of BDE congeners excreted by the second week of dosing. Higher proportions of lower brominated compounds (i.e., BDEs 47, 100, and 99), were found in chicken excreta compared to hexa- and hepta-BDEs. Pirard and Pauw (2007) postulated that because it was unlikely that the high amount of BDE 47 found in excreta came from the fraction unabsorbed in the intestinal tract, the excess BDE 47 in chicken excreta was evidence of formation from the reductive metabolic debromination of congeners BDE 99 and BDE 100. The authors further speculated that BDE 153 could be debrominated to form BDE 99 and BDE 154 debrominated to form BDEs 99 and 100.

Van den Steen et al. (2007) studied starlings for the bioaccumulation and tissue distribution of BDE 209. Four adult male starlings were housed in a large outdoor aviary and exposed to a solution of BDE 209 in peanut oil through a silastic tube implant. The exposed group received an implantation dose of 46.8 µg BDE 209/day for 76 days, and a control group (n=3) received an implant filled with unfortified peanut oil over the same time period. During the exposure period, 300 µL of blood was taken from each bird every 3–7 days. Following the exposure period, the experimental birds were euthanized and the pectoral muscle and bird liver were excised for analysis of BDE 209. It was found from analyzing the silastic tubes that only 50% of the total dose of BDE 209 diffused from the silastic tube implant into the bird. The half-life of BDE 209 in the blood of the starlings was estimated to be 13 days (95% confidence interval: 11 to 18 days). BDE 209 accumulated in muscle tissue at a rate 2-fold higher than in liver explained by the higher metabolic activity of the liver. In addition to BDE 209, other BDE congeners were detected in bird tissues. The detection of substantial amounts of BDE congeners 208, 207,206, 197, 196, and 183 in liver and muscle was evidence of metabolic debromination of deca-BDE in avian tissues (Van den Steen et al., 2007).

3.4.2.4. Evidence for Debromination in House Cats

Dye et al (2007) reported on the possible metabolic debromination of PBDE in house cats. The purpose of this study was to investigate the relationship between the incidence of feline hyperthyroidism in cats and the dietary ingestion of dry cat food contaminated with PBDE in addition to ingestion of PBDE contaminated house dust. The congeners BDE 47, 99, 207, and 209 were most frequently detected in blood serum of 23 exposed cats, and were related to dietary their intake. The cats consuming only canned-wet cat food (mostly comprised of fish) (n=4), relatively little BDE 207 or 209 was present in serum. BDEs 47 and 99 were present in the highest concentrations in the serum of canned food eaters. Conversely, in cats consuming only dry-food (n=8), BDE 209 dominated and the detected congeners had the following distribution: BDE 209> BDE 207 > BDE 47 > BDE 99. The remaining cats that consumed both food types (n=11) exhibited a mix of BDE congeners in serum with no one congener dominating the others.

The evidence for possible metabolic debromination of BDE 209 in house cats stemmed from a comparison of the BDE congener distributions present in serum to the BDE congeners profiles in the dry and wet cat food. The contamination of PBDEs in dry cat food reflected the congener profile of decaBDE, with BDE 209 representing 83-93% of total PBDE present in the feed. Because of the high content of BDE 209 in dry cat food, BDE 209 dominated serum in cats only consuming the dry food. BDE 209 accounting for 4.2%, 21%, and 30% of serum PBDE levels in house cats consuming canned-, mixed-, and dry-food, respectively. It was noted that BDE 207 was consistently present in serum in significant concentrations of the dry food eaters as compared to the consumers of the other food types. BDE 207 accounted for 4.5%, 9.8%, and 17% of the PBDE levels detected in cats consuming canned-, mixed-, and dry-food-eaters, respectively. BDE 207 was present in the dry food eaters at approximately 50% of the total concentration of BDE 209 which is uncharacteristic of the decaBDE congener profile (BDE 207 is approximately 1% of BDE congeners present in decaBDE), and was not the pattern observed in the wet food eaters. Moreover, the ratio of BDE 207 to BDE 209 in cat serum was relatively constant in all dry cat food eaters. The authors regarded these data as possible evidence for the metabolic debromination of BDE 209 to form BDE 207. These data are only suggestive of the *in vivo* debromination in house cats, but if confirmed through replicate studies, would generally imply the possibility for metabolic debromination of BDEs in humans.

3.4.3. Abiotic Degradation of PBDEs

Abiotic degradation predominantly occurs in the atmosphere and on soil surfaces. The energy of sunlight can degrade PBDEs in air and soils via photolysis, and the presence of the hydroxyl radical in air can deplete some PBDEs present in the atmosphere. The following is a brief review of these processes.

3.4.3.1. *Photogradation of PBDEs*

Several studies have shown that higher brominated BDE congeners can photodegrade to form lower brominated congeners as photochemical byproducts. The photodegradation is defined as the photochemical transformation of a molecule into

lower molecular weight fragments, usually in an oxidation process (IUPAC, 1996). This term is widely used in the destruction (oxidation) of pollutants by UV-based processes; e.g., the absorption of photons present in wavelengths found in sunlight, i.e., ultraviolet (UV) radiation. Photodegradation of PBDE occurs from the removal of a bromine atom on the PBDE molecule, thus transforming the higher molecular weight BDE to lower brominated congeners.

Fang et al (2008) reported on the experimental photodegration of BDE 28, BDE 47, BDE 99, BDE 100, BDE 153 and BDE 183 dissolved in hexane. Known concentrations of the BDE congeners (approximately 150 ng/mL) present in solvent were exposed to >290 nm UV wavelengths in a photo reactor consisting of a 500 W mercury lamp, quartz tubes, Pyrex glass tubes and a water cooling system.. The experiments were performed in triplicate and a control was used that consisted of BDE congener solutions placed in total darkness. Photodegradation occurred for all studied congeners, and generally followed pseudo-first-order kinetics. The photodegradation half lives of the BDE congeners discerned from this study were: BDE 28 = 4.97 h; BDE 47 = 2.53 h; BDE 99 = 0.32 h; BDE 100 = 6.46 h; BDE 153 = 0.29 h; and BDE 183 = 0.26 h. Generally the higher brominated BDE congeners degraded at a faster rate than the lower brominated congeners. It was concluded that the main decomposition mechanism induced by photolysis was reductive debromination to form lower brominated BDE congeners. Fang et al. (2008) observed the following photodegradation products:

- BDE 28 (triBDE) photodegraded to form BDE 8 and BDE 15 (both diBDEs); the diBDEs further degraded to form the monoBDE congener BDE 1 and BDE 3;
- BDE 47 (tetraBDE) photodegraded to form two tri-brominated species (BDE 17 and 28), which in turn further are debrominated to form di-brominated BDE congeners
- BDE 4, 8 and 15, debrominated to form monoBDE congeners BDE 1 and BDE 3;
- BDE 99 (pentaBDE) photodegraded to form the tetraBDE congeners 66, 49 and 47 (tetraBDE), which subsequently undergo further photolysis.

- BDE 100 (pentaBDE) photodegraded to form BDE 75 and BDE 47 (both tetraBDEs).

- BDE 153 (hexaBDE) photodegraded to form BDE 99, 101 and 117 (pentaBDEs).

Kajiwara et al (2007) reported on experimentally photodegrading BDE 209 contained in flame retarded plastic. The purpose of their study was to investigate the potential for the photolytic debromination of the brominated fire retardants (BFR) decaBDE and decabromodiphenyl ethane (DBDPE) in treated plastics. Four different fire retarded plastic samples were evaluated including pure high impact polystyrene (HIPS) not treated with BFR, HIPS treated with decaBDE (0.15% wt/wt), HIPS treated with DBDPE (about 0.10% wt/wt), and used TV casing made from HIPS. It was noted that DBDPE is a chemical substitute for decaBDE, and has a chemical structure similar to BDE 209. The plastic samples were dissolved in toluene and shaken overnight to be completely mixed. The toluene was evaporated by air drying in a dark room. After drying, solid materials including TV casing were pulverized in liquid nitrogen chamber. The pulverized plastic samples were initially passed through a 300 micron (μm) sieve and then a 106 μm sieve. The plastic powder collected between two sieves (106 μm-300 μm) was used in the UV irradiation experiment. Aliquots of the plastic powder (0.3 g) were placed in sealed quartz tubes and subsequently exposed to natural sunlight at time intervals of 0, 7, 14, 28, 56, and 112 days. Controls (tubes of the same powdered plastic samples) were placed in a temperature controlled dark room. Following each test period, samples were extracted and then analyzed using high resolution gas chromatography coupled with high resolution mass spectrometry in the ion monitoring mode. Photolysis of BDE 209 in the decaBDE treated HIPS samples was observed after one week of exposure to sunlight. The BDE 209 concentration in the plastic decreased and hepta-, octa-, and nona-BDE congeners were were formed as products of photolysis. The mechanism of the photodegradation of BDE 209 was postulated to be by the process of eliminating bromine atoms on the molecule. Approximately 80% of the BDE 209 had degraded after 112 days of exposure to sunlight, but only 5% of the BDE 209 mass was converted to lower brominated BDE congeners, suggesting the formation of unknown products of photolysis. Tri though octa polybrominated dibenzofurans (PBDFs) appeared

in the irradiated samples suggesting that the photolysis of BDE 209 formed PBDFs, but not in sufficient quantities to close the mass balance of the products formed. No clear pattern of photodegradation was observed with the other plastic samples. No loss of BDE 209 was observed in the control samples. This study demonstrated the photodegradation of BDE 209 in treated HIPS by natural sunlight.

Stapleton and Dodder (2008) experimentally photodegraded decaBDE present in house dust by exposure to natural sunlight. Two different house dust materials were obtained from the National Institute of Standards and Technology (NIST) in Maryland. The first material was typical indoor house dust known as Standard Reference Material 2585 (SRM 2585). During chemical analysis it was found to be contaminated with a variety of BDE congeners ranging from tri- to decabromo-substituted congeners. The second dust sample was identified as SRM 2583, and was a standard reference material used in the analysis of metals and organics in dust. The SRM 2583 dust sample was precleaned to be free of PBDE contamination and then spiked with 1.298 g of a solution of BDE 209 dissolved in toluene. The sample was air dried to evaporate the toluene, resulting in a concentration of 2,180 ng BDE 209 /g dry weight (dwt) in the SRM 2583 dust sample. The SRM 2585 dust sample was not precleaned nor spiked with BDE 209. Aliquots of both SRM dust samples were placed into two 4.5 mL methylacrylate chambers (referred to as UV cuvettes) that are routinely used for measuring UV absorbance of test materials. The UV cuvettes containing the dust samples were exposed to natural sunlight outdoors in a tray lined with aluminum foil. Except during periods of precipitation, the cuvettes were placed outside daily, Monday through Friday, from approximately 9:00 AM to 4:00 PM until 200-h cumulative exposure to sunlight was achieved. Samples were transferred to a laboratory where they were extracted and analyzed for PBDE using high resolution gas chromatograph coupled with high resolution mass spectrometry operated in the electron capture chemical ionization mode. The average intensity of sunlight over the duration of the experiment was 545 watts per square meter (W/m^2), with a range of 61 to 929 W/m^2. The average of outdoor temperature was 26.9°C and a range from 18.7 to 32°C. Photodegradation of BDE 209 was observed in both SRM dust samples after 100 h exposure to natural sunlight. The first-order BDE 209 photodegradation rates were calculated as 2.3 E-3 and 1.7 E-3 per

hour in the spiked (SRM 2583) and natural (SRM 2585) dust samples, respectively. The octabrominated congeners, BDE 201 and BDE 202, were observed in both reference dust samples to be products formed from the debromination of BDE 209 induced by photodegradation. In the SRM 2583 (spiked) sample, additional congeners were formed: BDE 183, BDE 197, BDE 202, BDE 203, BDE 206, BDE 207 and BDE 208. From these data, the authors calculated a mass balance based on the photodegradation of BDE 209 in the spiked dust samples. After 200 h exposure to natural sunlight, the initial concentration of BDE 209 was observed to have decreased by approximately 38%. The authors concluded that 35% of the decrease in the concentration of BDE 209 as due to debromination and the subsequent formation of lower brominated BDE congeners and 3% of the decrease in concentration was postulated to have been caused by the volatilization of BDE 209.

Rayne et al. (2006) photodegraded BDE 153 in acetonitrile, distilled water, and seawater at ultraviolet radiation wavelengths (UV radiation) of 302 nm (nanometers). BDE 153 dissolved in acetonitrile and irradiated for 5 min formed three primary photodegradation products: (1) penta-BDE isomers 99, 101, and 118 (20% yield); (2) the brominated dibenzofuran congener 1,2,4,7,8-PeBDF (30% yield); and (3) three non-speciated tetrabrominated 2-hydroxybiphenyls (20% yield). Continued irradiation up to 1hr caused the photo degradation of the penta-BDE congeners to form the tetra-BDE isomers BDE 47, 49, 66, and 77. The irradiation of BDE 153 in distilled and/or seawater produced the same tetra-BDE isomer byproducts, albeit, less efficiently than acetonitrile. Rayne et al. (2006) were unable to conclude firmly whether or not the photodegradation of PBDEs in aquatic systems under natural conditions is a viable process.

Bezares-Cruz et al. (2004) decomposed BDE 209 to form lower brominated BDE congeners through natural sunlight at wavelengths of 300, 305.5, 311.4, 317.6, 325.4, 332.4, and 368 nm. Deca-BDE formulation (approximately 97% BDE 209) was dissolved in hexane to create three solutions of BDE 209 ranging in concentrations from 6.92E-06 to 6.14E-06 micro molar (μM). Control samples were prepared in the same manner but kept in darkness. Samples were exposed to sunlight on clear days in the summer and fall of 2003. BDE 209 dissolved in hexane and exposed to sunlight photodegraded within minutes. After 30–45 minutes of exposure to mid-afternoon

sunlight on October 23 and July 2 of 2003, the BDE 209 concentration was reduced to approximately 5% and 1% of the initial concentration, respectively, corresponding to a pseudo-first-order reaction rate of 1.11×10^{-3}/s and 1.86×10^{-3}/s. The higher reaction rate in July was due to the increased intensity of solar flux as compared with October. There was no evidence of degradation of BDE 209 in the control solutions not exposed to sunlight. The solar irradiation of BDE 209 dissolved in hexane catalyzed the reductive debromination of the congener. Forty-three PBDE congeners of various bromine substitutions were formed during different times of exposures to sunlight. After 5 minutes of solar irradiation, the disappearance of BDE 209 was matched by the initially rapid formation of nonaBDE congeners. The other congeners (octaBDEs, heptaBDEs, and hexaBDEs) accumulate successively over the 60-min-exposure period. OctaBDEs are transformed to heptaBDEs that are then transformed to hexaBDEs. With respect to the possibility of the photodegradation of BDE 209 in natural waters, Bezares-Cruz et al. (2004) postulated that the photochemical reaction would expected to be somewhat attenuated by sorption of BDE 209 onto colloidal particles in the water column, and by the light attenuation properties of humic materials in aquatic systems. Furthermore, the presence of hydrogen donors necessary in invoke the reaction would likely be at lower concentrations in natural waters as compared to hexane.

Söderström et al (2004) reported on the experimental photodegradation of BDE 209 in various matrices, including toluene, silica gel, sand, soil and sediment. UV-exposure experiments were conducted both in the laboratory with artificial UV-light (all matrices) and under natural conditions with outdoor sunlight (sand, soil, sediment). To begin the experiment, a 0.5 gram sample of each of the matrices was placed into Pyrex-tubes and fortified with a solution of 10.5 ng/μL decaBDE dissolved in toluene). The toluene was then allowed to evaporate while the samples were kept in the dark. For the exposure to artificial light, the samples were placed in an apparatus consisting of four mercury UV lamps equipped with filters to mimic the sunlight spectra in the UV range of 300-400 nm. The irradiance intensity from the UV lamps was estimated to have been 1.6 mW/cm^2. The exposure of soil and sediment were extended to an additional 121 and 244 hours. For the conditions of natural sunlight, the Pyrex tubes containing fortified samples of each matrix were placed in a tray and put on the roof of the laboratory during the

month of July, 2007. Maximum UV-irradiance from the sun at mid-day was measured to have been 2.3 mW/cm^2. Exposure durations of the samples ranged from 0 to 96 hours. All samples were transferred to a laboratory, extracted and analyzed using HRGC/HRMS. Söderström et al (2004) found that BDE 209 photolytically degraded from exposure to both artificial and natural sunlight. With exposure to artificial light, photodegradation was more rapid and complete when BDE 209 was associated with toluene or silica gel than with sand or sediment. BDE 209 in toluene or silica gel degraded to 1% of the initial BDE 209 concentration over 8 h when exposed to artificial light, but degraded to about 21 and 57% over 96 hrs when associated with sand and sediment. By comparison, natural sunlight degraded BDE 209 in sand and sediment to about 36% and 43% over a 96 h exposure. The half-life of BDE 209 varied by matrix and artificial or natural sunlight. The half-life BDE 209 in toluene or silica exposed to artificial light was less than 0.25 h. By comparison, BDE 209 in sand, sediment and soil had estimated half lives of 12 h, 40 - 60 h, and 150 - 200 h, respectively, when exposed to artificial light. For exposure to natural sunlight, the half lives of BDE 209 in sand and sediments were 37 h and 80 h, respectively. Söderström et al (2004) reported that the photoytic debromination of BDE 209 formed lower brominated BDEs in both artificial and natural sunlight conditions. These included: BDE 47 (silica gel only), BDE 100 (toluene only), BDE 119 (toluene, sand, sediments and soil), BDE 99 (toluene and silica gel), BDE 154 (all matrices), BDE 153 (toluene, sand, sediments and soil), BDE 140 (all matrices except soil), BDE 128 (all matrices), BDE 183 (all matrices), and BDEs 206, 207 and 208 (all matrices).

Sánchez-Prado et al. (2005) investigated the photodegradation of pentaPBDE at two UV irradiation intensities. The pentaBDE technical formulation was dissolved in cyclohexane to a concentration of 10 µg/mL. The penta-BDE mixture was comprised of the BDE congeners 47 (4.1 µg/ml), 85 (0.1 µg/ml), 99 (1.2 µg/ mL), 100 (4.1 µg/mL), 153 (0.23 µg/mL), and 154 (0.34 µg/mL). A 5 ml aliquot of the solution was adsorbed to 100 µm polydimethylsiloxane (PDMS) fibers and placed in a laboratory photo reactor equipped with two low-pressure mercury lamps (8–10 W, 254 nm) for up to 1 hour. Equal control samples were stored in complete darkness. Chemical analyses showed no degradation of the BDE mixture occurred in the controls. Debromination of the BDE

congeners occurred over both intensities of UV irradiation. The reaction rate was observed to be dependent on the degree of bromination (e.g., increasing rate of photodegradation with increasing number of bromine atoms on the molecule). The reaction rate was observed to be independent of the irradiation intensity. Approximately 21 BDE congeners were observed as the degradation products from the irradiation of the mixture of BDEs 47, 85, 99, 100, 153, and 154. Based on sequential formation of these products, Sánchez-Prado et al. (2005) postulated that the principle photodegradation pathway was reductive debromination.

3.4.3.2. *Reaction with the Hydroxyl Radical*

Tropospheric reactions with the hydroxyl radical have been observed to be an important atmospheric degradation pathway for halogenated hydrocarbons. The OH⁻ radical is generally formed from the photolysis of ozone (O_3) to form a single oxygen atom that subsequently combines with hydrogen. No direct measurements of atmospheric degradation rates of PBDEs from interaction with the OH⁻ radical could be located in the literature. However, atmospheric half-lives have been estimated from quantitative structural activity relationships. Assuming an OH⁻ radical concentration of 5 x 10^5 hydroxyl radicals per cm^3, the atmospheric half-lives of penta-, octa-, and deca-bromodiphenyl ether homologues have been estimated to be 29, 140, and 476 days, respectively (ATSDR, 2004). A degradation rate constant for BDE 99 has been estimated to be 1.27 x 10^{-12} cm^3/molecule/s using the same assumption of OH⁻ radical concentration (European Union, 2001). The ATSDR concluded that atmospheric degradation from reaction with the OH radical is likely to be an insignificant atmospheric loss mechanism (ATSDR, 2004).

3.5. THERMAL DECOMPOSITION OF PBDE

PBDEs are added to plastics, textiles, and other materials in order to inhibit combustibility and delay the spread of fire (see Chapter 2 for more detail). Basically the PBDE additives interfere with the combustion process by forming bromine gas which, in turn, displaces the O_2 necessary to sustain the oxidation reactions. In addition, bromine radicals are formed which interfere with the chain reactions of hydrogen and hydroxyl

radicals in the fire. The duality of these processes severely retards and constrains thermal oxidation.

The combustion of PBDE treated materials can lead to the formation and emission of polybrominated dibenzofurans (PBDFs) in the smoke (Weber and Kuch, 2003). It is believed that the molecular structure is suitable for PBDEs to be precursor compounds to PBDFs formation. Debromination of BDE 209 occurs at temperatures of about 500°C, leading to the formation of brominated biphenyls. Further combustion of brominated biphenyls causes PBDFs to be formed within the combustion gases (Webber and Kuch, 2003). Rupp and Metzger (2005) formed both PBDFs and PBDDs as byproducts from the thermolysis of BDE 47 and BDE 153 in quartz ampoules heated to temperatures varying from 250–500°C for 5–10 minutes. OctaBDF (octabrominated dibenzofuran) and heptaBDF congeners can be formed and emitted during the extrusion of HIPS plastic treated with decabromodiphenyl ether/antimony (antimony (III) oxide at 275°C (Luljk et al., 1992). The yields of PBDFs showed a significant increase as a function of the number of extrusion cycles. Lower brominated diphenyl ethers were present in the emissions from the extrusion process confirming that the debromination of decaBDE and subsequent exchange of bromine atoms with hydrogen was a necessary step to the thermolytic formation of PBDFs. In a series of experiments of combusting deca-, penta-, and, octaBDEs in quartz ampoules at 510–630°C, Buser (1987) formed PBDFs and PBDDs from the intramolecular cyclization reactions involving the attack of an oxygen atom on the diphenyl ether molecule. The initial steps to these reactions were the debromination of the parent compound followed by hydrogen substitution. Polybrominated benzenes and polybrominated phenols were also formed as combustion byproducts.

3.6. PATTERNS OF ENVIRONMENTAL FATE OF PBDE

Commercial Octa and PentaBDE consist of mixtures of BDE congeners. PentaBDE predominately contains BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154, and commercial octaBDE mainly contains hepta–, octa–, and hexaBDE congeners. Commercial decaBDE are almost entirely BDE 209 with a small amount of nonaBDE. The BDE congeners are dispersed into the open environment whenever the commercial

formulations are released from their manufacture, use, and disposal. The environmental fate of PBDE congeners is dictated by their physical and chemical properties and their propensity for biotic and abiotic transformation. These characteristics were reviewed in previous sections to this chapter (see section 3.2 of this Chapter). Chapter 2 reviewed the use and life-cycle of PBDE formulations, and presented estimates of environmental releases. The purpose of this section is to describe, in general terms, the fate of PBDEs once they are released into the environment.

PBDEs are mainly discharged into the air from production, use and recycling of PBDE treated plastics, electronics, computers, textiles, and polyurethane foam. (less than 5%) PBDEs are discharged into surface waters from industrial activity and sewage treatment plants (STPs). The land disposal of sewage sludge and industrial sludges also contributes to environmental loadings.

Dispersion of BDE congeners in the environment is governed by their respective physical and chemical properties. Air and water are primary transport media, while soils and sediments are environmental sinks. In general, BDE congeners are highly hydrophobic and lipophilic compounds that have low water solubility and low vapor pressures. The atmosphere and marine currents can transport BDEs over relatively long distances (> 1,000 km). Evidence for this comes from the presence of PBDEs in the tissues of deep ocean-dwelling whales and other marine mammals far from anthropogenic sources activities. With the exception of BDE 209, BDE congeners bioaccumulate into terrestrial and aquatic food webs. The body burdens of BDE congeners in a wide variety of avian species, fish, and aquatic and terrestrial mammals indigenous to geographical areas ranging from the equator to the poles also substantiate BDE's propensity for long-range transport. The following subsections discuss the fate of PBDEs in air, water, and soil.

3.6.1. Fate of PBDEs in Air

The air compartment is the primary medium of dispersing PBDEs over large geographical areas. PBDE behavior in air is that of other semivolatile compounds (i.e., BDE congeners partition between the vapor phase and particle-bound phase in accordance with their respective vapor pressures (see Table 3-6). At standard

temperature and pressure, the mono through tri brominated compounds are primarily in vapor phase; the tetra and penta congeners split between vapor and particle bound phases but predominate in the vapor phase; the hexa and octa brominated BDEs are mostly attached to atmospheric particles; and BDE 209 is exclusively adsorbed to atmospheric particles. Experimental evidence suggests that the lower brominated BDEs, those that predominate in the vapor phase, can be degraded in the atmosphere through reaction with the hydroxyl radical as well as through photolytic chemistry. Wania and Dugani (2003) investigated the propensity for long-range atmospheric transport through the atmosphere with the application of four long-range atmospheric transport models. Wania and Dugani (2003) concluded that the tetra- and penta-BDE congeners have a greater tendency to be transported long distances than lower and higher brominated species. The characteristic travel distances of different PBDEs were calculated from the air models. The characteristic travel distance is defined as the horizontal distance traveled by a parcel of air whereby 63% of the initial air concentration of PBDE is depleted (Wania and Dugani, 2003). The models showed that the characteristic travel distance ranged from 483 - 1,113 km for tetraBDE; 608 - 1,349 km for pentaBDEs; and 480 - 735 km for decaBDE (Wania and Dugani, 2003). In theory,

- Vapor phase mono, di, and tri brominated compounds have the highest tendency for long-range transport in the atmosphere because they are not susceptible to particle surface deposition.

- The tetra and penta brominated congeners more equally partition between vapor and particle-bound phases and have characteristic travel distances that are somewhat less than the vapor-phase compounds.

- This bi-phased distribution in air increases the atmospheric removal of the moderately brominated compounds because of the partial adsorption to atmospheric particles and subsequent wet and dry deposition to the surface.

BDE 209 is almost entirely associated with atmospheric particles and, therefore, its long-range transport potential is theoretically diminished by the deposition processes. However, adsorption of BDE 209 to atmospheric aerosol particles leaves the possibility that a small fraction of the BDE 209 contaminated aerosol can be transported over long distances, which is dependent only on the fate of the aerosol (Wania and Dugani, 2003;

Gouin et al., 2006. There exist significant gaps in the understanding of how aerosols are dispersed regionally and globally. This hinders the possibility of making definitive conclusions on the long-range transport potential of BDE 209. Aerosol particles may mimic the behavior of gases making them less prone to surface deposition; this may explain the measurement of low air concentrations of BDE 209 in the Arctic whenever the prevailing winds emanate from urban areas in Alaska and Canada (Wang et al., 2005). Others have observed the routine presence of the lighter, more volatile PBDE congeners (i.e., BDE 28, 47, and 99) in air over remote oceanic regions, and BDE 209 has only been occasionally detected (Wurl et al, 2006a). It has been noted (see subsection 3.4.3.1 above) that BDE 209 is labile to photodegradation by natural sunlight. Photodegradation results in the loss of bromine atoms on the BDE 209 molecule to form lower brominated BDE congeners as products of photolysis, and this may further explain the low potential for long range transport of BDE 209. Photodegradation is likely a major atmospheric process for degrading and transforming higher brominated PBDEs to less brominated BDE congeners, and experiments have clearly shown that photodegration of PBDEs is induced by the spectra of UV radiation under natural conditions.

Generally BDE 47, 99, and 209 dominate the congener pattern when detected in urban air. The densely settled urban areas are geographically dispersed sources of the BDE air burden, and a strong negative atmospheric concentration gradient from the urban center out to rural and remote areas is often observed in air monitoring studies (Gouin et al, 2005). This implies that urban areas contribute to background air concentrations of PBDE in rural and remote settings, and should be considered as major area-wide sources of PBDEs present in ambient air. Chapter 4, Section 4.5 discusses concentrations of individual congeners and total BDEs in indoor and outdoor air in the United States and abroad.

In addition to the physical processes of wet and dry surface deposition, and photochemical reactions and degradation/debromination, the leafy surfaces of deciduous forest canopies can significantly deplete PBDEs in the atmosphere via air-to-leaf transfer (Su et al., 2007). The uptake into leaves transfers the BDE congeners from the air to the terrestrial ecosystem when the leaves drop to the surface. This process is seen as being both vapor-phase and particle-bound interception by the leaf during the growing

season—with the vapor phase more easily absorbed. The more volatile, low molecular weight PBDE compounds are deposited primarily through gaseous diffusion and achieve a partitioning equilibrium between the air and the leaf. The less volatile, heavier PBDE compounds are scavenged by the leaf via dry particle deposition caused by particle impaction and diffusion (Horstman and McLachlan, 1998). These processes of vegetative atmospheric depletion of semivolatile compounds are also observed with coniferous forests, grasses, and green leafy crops (Horstman and McLachlan, 1998). Therefore dense forests, open grasslands, and productive farmlands functionally reduce the air concentration of BDEs as contaminated air masses move over these areas. The octanol/air partition coefficient (log K_{OA}) of BDE congeners (see Table 3-5) is an important indicator of this propensity for uptake into leafy biomass with all congeners having a log $K_{OA} > 6$. The atmospheric scavenging by forest canopies, grasses and other leafy biomass can partially explain the seasonal variability of atmospheric BDEs. Long-term monitoring studies indicate higher concentrations of BDEs in air during the cooler winter months, and lower concentrations over the spring and summer (Gouin et al., 2005). An early springtime "bud-burst" effect of reducing the air concentrations of BDEs at the onset of spring has been documented to occur (Gouin et al., 2005). In the spring and summer, greater diurnal variability in air concentrations of BDEs is observed during the warmer months, and, conversely, stable air concentrations of BDEs are observed during the cooler winter months. The relative stability of BDEs over rural and remote areas during the winter can be explained by diffusion of BDEs from urban centers induced by a negative concentration gradient, whereas the variability in the spring and summer reflects vegetative influences on the atmospheric concentrations (Gouin et al., 2005).

Large bodies of water also have the capacity to influence the atmospheric concentrations of BDEs by means of the air-water exchange of the contaminant. The rate of exchange of BDEs is dependent on the state of equilibrium between air and water concentrations. If the ratio of the BDE fugacity in water is greater than the BDE fugacity in air (in the vapor phase), then there is a tendency for the BDE to volatilize from the water into the air. Likewise, if the BDE vapor phase fugacity in air is greater than the dissolved phase in water, then there is a tendency for the transfer of BDE from air into

water. Equilibrium is achieved when the water-air fugacity ratio $\approx$ 1 in which case there is no air-water exchange of BDEs. Phytoplankton biomass on the surface of oceans plays a role in influencing the fugacity gradient (Jaward et al., 2004). The BDEs in the dissolved phase in the water column will be drawn to partition to the carbonaceous biomass due to their high K_{ow} (see Table 3-2). The dissolved phase concentration is decreased, which, in turn, may induce diffusion from air to force the air-to-water exchange. In this case, the capacity of an ocean to absorb BDEs from the atmosphere is indirectly controlled by the density of surface biomass. BDEs have been measured in the dissolved phase and in suspended particulates to seawater (Wurl et al., 2006b). In coastal waters off China, concentrations of total PBDE in the dissolved phase ranged between 40.2 and 228.2 pg/L, and between 8.1 and 69.1 pg/L in the suspended particulate matter (Wurl et al., 2006b). In the San Francisco estuary BDE 47, 99, and 209 were found to be the most abundant congeners detected in the dissolved phase (Oros et al., 2005), whereas Wurl et al. (2006b) found BDE 28, 47, and 100 dominated total PBDEs in the open ocean with BDE 209 at detected only at trace levels. In the freshwater of Lake Ontario approximately 60% of the total PBDE was composed of BDEs 47 and 99, with BDE 100, 153, and 154 congeners each contributing approximately 5 to 8% of the total (Environment Canada, 2006). Section 4.2 provides further discussions on sediment concentrations of PBDEs.

3.6.2. Fate of PBDEs in Water

Once encompassed within surface waters as a result of atmospheric deposition and/or the direct discharge from anthropogenic source activities, the PBDEs partition between the water column and the sediments in proportion to their physical-chemical properties. The benthic sediments are a primary sink for PBDEs. PBDEs then can bioaccumulate up the aquatic food web beginning with benthic organisms and ending with predators at the top of the food chain, e.g., piscivorous fish, birds, and terrestrial mammals.

In general, PBDE concentrations are highest in sediment samples collected downstream of the following: industrial/urban areas, outfalls to sewage treatment plants, and urban locations without heavy industries. The lowest PBDE concentrations are

generally found in sediments collected at remote and agricultural areas. BDE 209 appears to dominate the congener profile of aquatic sediments, however most congeners have not been detected. Section 4.2 includes a complete description of sediment studies that bear out these trends.

Only two studies could be located that evaluated the phase distribution of BDE congeners in water. In Lake Michigan, Streets et al. (2006) found dissolved phase BDE congeners 47, 99, 100, and 66 in concentrations ranging from 0.13 to 10 pg/L (for individual congeners). Three congeners were detected in the particle-bound phase: BDEs 47, 99, and 100 were found at concentrations ranging from 0.18 to 1.4 pg/L. BDE 209 was not evaluated in this study. Wurl et al. (2006a) evaluated the phase distribution of BDEs 28, 47, 99, 100, 153, 156, 183, and 209 in the sea-surface microlayer (SML) and subsurface seawater (SSW) from locations off the coast of Hong Kong. The SML of the seawater is defined as a 100-μm thick boundary layer between the atmosphere and the ocean surface that is comprised of naturally occurring organic matter and micrometer-sized suspended particles. The subsurface is the layer below the SML down to a depth of 1 m. Over all samples, total PBDE concentrations ranged from 11.3 to 62.3 pg/L in the dissolved phase and from 26.2 to 32.5 pg/L in the particle-bound phase. BDE 209 was detected at trace levels in the dissolved phase at all sampling locations. Only the BDE congeners 28 and 47 were detected in the particle-bound phase of the SML. Below the SML in the SSW, BDEs 28 and 47 were detected at three sampling locations. BDE 99 was the only other congener detected in the dissolved phase of the subsurface layer.

Once in the sediments or in the suspended organic matter in the water column, the PBDEs bioaccumulate into the ecological food chains beginning with the benthic organisms and continuing up to the top predators. The BDE congeners have a high capacity for bioaccumulation and biomagnifications in biota as indicated by their relatively high K_{OW} factors (see Table 3-2). Contamination of fish tissue with BDE congeners exposes fish-eating fish, piscivorous birds, terrestrial animals, and humans to BDEs via the dietary pathway. There is suggestive evidence that fish are able to transform highly brominated PBDE congeners (*in vivo*) to lower brominated PBDE congeners through the process of metabolic debromination (see section 3.4.2.1.for further discussion). It has been suggested that metabolic debromination may cause the formation

of BDE 47 and BDE 99. Piscivorous raptors bioaccumulate PBDEs through their habitual consumption of contaminated fish. These birds may also have the capacity for metabolic debromination of higher brominated BDE congeners to lower brominated congeners, although the evidence for this is highly suggestive, and stems primarily from the study of chickens and starlings. PBDE congeners have been detected in marine mammals such as whales, seals, and porpoises, indicating their exposures to PBDEs from their diet. Bioaccumulation within a broad range of animals is an indication of the consequences of widespread PBDE contamination in the aquatic and marine trophic networks (see section 3.7.1. Bioaccumulation in the Aquatic Environment, below, for a more detailed discussion).

3.6.3. Fate of PBDEs in Soil

Soil is a major sink and environmental reservoir for PBDEs. Atmospheric PBDEs exchange between the air and the soil compartments by means of gas and particle depositional processes. The atmospheric gas-particle partitioning of BDE congeners and the type of biomass covering the soil surface has an affect on the flux of PBDEs from air to soil. There are apparent differences in the magnitude of soil concentrations of BDE congeners in soils overlaid by coniferous woodlands, deciduous woodlands, and grass. When the soils are covered by vegetation, especially forests, the scavenging of semivolatile organic chemicals from the atmosphere is enhanced, which, in turn, increases their depositional flux to the terrestrial surface relative to deposition to bare soil (MacLeod, 2003). In consideration of gas-particle partitioning and ground cover, Palma et al. (2002) estimated the environmental media partitioning efficiency of air releases of unsubstituted diphenyl ether (DE), and BDEs 47, 99, and 209 from PBDE sources. Fugacity modeling suggested that approximately 68% of DE and 98% of BDEs 47, 99, and 209 of the air emission will partition into the soil compartment at equilibrium. There is suggestive evidence that PBDEs, especially BDE 209, in soils can be degraded by the microbial reductive debromination as well as the photolytic debromination in soils, although the viability of these processes in soils is not presently well understood (see section 3.4.3 for a detailed discussion).

On a geographical and regional scale, the PBDE concentrations in soils usually reflect a gradient (high to low) from the central city out to rural areas consistent with atmospheric measurements (Harrad and Hunter, 2006). Therefore, urban areas are regional sources of PBDEs to soil via the air to soil exchange. Cetin and Odabasi (2007) found a strong relationship between the BDE congener profile in soil and air ($r^2 = 0.13$ to 0.79, $p < 0.01$), thus further supporting the assumption of a close link between air and soil. From this evidence it is concluded that the atmosphere is a major transport media for the PBDEs detected in soils.

Some soil studies of PBDEs show a dominance of congeners 47, 99, 100, 153, and 154 (Hassanin et al., 2004), but other soil data shows that BDE 209 dominants total soil PBDE concentration (Cetin and Odabasi, 2007), especially in urban areas. BDE 209 also has been shown to dominate the sediment profile. Dominance of BDE 209 reflects the change from the general global use of the commercial penta- and octa-BDE formulations to deca-BDE, as well as high sorption and persistence of BDE 209. Further discussions of PBDEs in surface soils are found in Section 4.3.

3.6.4. PBDEs in Sewage Treatment Plant Influent, Effluent, and Sludge: A Cause of BDE Contamination in Surface Waters

Sewage treatment plant (STP) operations are likely a significant source of PBDEs to surface water leading to local contamination of the freshwater and coastal marine environments. The STP receives wastewater from homes, businesses, and, in many cases, industries, which subjects the wastewater to different degrees of treatment before the treated effluent is discharged into surface waters. Much of the STP sewage sludge generated by the treatment of wastewater is disposed of on land, which, in turn, can lead to water pollution through soil erosion into surface waters.

North (2004) sampled influent, effluent, and sewage sludge for the presence of 41 BDE congeners at a STP in Palo Alto, CA. The STP employs tertiary treatment methods and processes approximately 95E+06 L/day of wastewater generated by residents (60%), industries (10%), and commercial businesses and institutions (30%). The Palo Alto STP discharges treated effluent into to the San Francisco Estuary; therefore the STP is likely a source of local PBDE contamination to the water, sediments, and biota. Results showed

that more than 90% of the total PBDE concentrations in the STP effluent discharged to surface waters after tertiary treatment were comprised of BDE congeners 47, 99, 100, 153, and 209. BDE 99 dominated the congener profile. The rank order and mean concentrations of BDE congeners in the STP effluent were BDE 99 (11,200 pg/L) > BDE 47 (10,467 pg/L) > BDE 100 (1,983 pg/L) > BDE 209 (1,730 pg/L) > BDE 153 (983 pg/L). With respect to the sewage sludge from the STP tertiary process, BDEs 47, 99, and 209 represented approximately 85% of the total concentration of BDEs detected. BDE 209 dominated the profile; it represented about 35% of total BDEs. The rank order and mean concentrations of BDE congeners in the STP sewage sludge (µg/kg dwt) were BDE 209 (1,183) > BDE 99 (944) > BDE 47 (757). From a mass balance perspective, North (2004) estimated that 96% of the PBDEs that enter the STP are adsorbed to sludge, and 4% is deposited into surface water with the wastewater effluent. The high percent adsorption of the BDE congeners to the STP sludge can be attributed to their high log K_{ow}, which is > 5.0 for all congeners (see Section 3.2.2). It is not known whether BDE 209 in sewage sludge photolytically or microbially degrades to form lower brominated congeners.

Knoth et al. (2007) investigated the distribution of BDEs 28, 47, 99, 153, 154, 183, and 209 in sewage sludge samples from 11 STPs in Germany. Thirty-nine sewage sludge samples from different stages of the wastewater treatment process (primary sludge, secondary excess sludge, and dewatered digested sludge) were collected from March 2002 to June 2003. BDE 209 dominated the PBDE distribution in all STP sludges. BDE 209 concentrations in sludges ranged from 97.1 to 2,217 ng/g dwt with a mean of 429 ng/g dwt. The sum of BDE congeners 28, 47, 99, 153, 154, 183 ranged from 12.5 to 288 ng/g dwt (mean 126 ng/g dwt). The BDE congener profile remained rather static from one sludge type to another. Knoth et al. (2007) speculated that this may provide evidence for the biotransformation of BDE 209 to lower BDE congeners. With over half the sewage sludge applied to land, Knoth et al. (2007) estimated that 150 kg/acre of pentaBDE plus octaBDE, and 350 kg/acre of decaBDE were applied to land in 2001 from the land farming of contaminated sewage sludge in Germany.

Wang et al. (2007) analyzed sewage sludge samples from 31 STPs in 26 cities in China for the distribution of BDE congeners 17, 28, 47, 66, 71, 85, 99, 100, 138, 153,

154, 183, and 209. The concentrations of the sum of all congeners excluding BDE 209 ranged from 6.2 to 57 ng/g dwt, with a mean and median concentration of 19.6 and 16.0 ng/g dwt, respectively. As with the studies discussed above, BDE 209 was the dominant congener in most of the sludge samples. The percentage of BDE 209 concentration to total PBDE concentration in Chinese sewage sludges averaged 55%—with a median of 69%. BDE 209 concentrations in sewage sludge ranged from non-detect to 1,109 ng/g dwt (mean—70.8 ng/g dwt; median—25.5 ng/g dwt). BDE 209 was not detected in four sludge samples. In more than 80% of the sludge samples, BDE 209 was less than 100 ng/g dwt. Other dominant BDE congeners in the sludges were BDE 47 (mean 24% of total BDE concentration), BDE 99 (mean 22%), and BDE 183 (mean 13%). Wang et al. (2007) regressed the data and determined correlations with BDE congener pairs. A significant and positive correlation ($r = 0.814$, $p < 0.001$) was between BDEs-47 and 99. This association implied significant contamination of the sludge from the pentaBDE commercial formulation.

Song et al. (2006) investigated the fate, partitioning, and mass loading of BDEs throughout the STP process from influent to sewage sludge to effluent. Three sets of samples were taken from various segments of the sewage treatment process at an STP in Windsor, Ontario, Canada over 3 days spaced over a 6-week period between the end of March and early May, 2004. The BDE congeners 28, 47, 71, 99, 100, 138, 153, 154, and 183 were evaluated in the study. BDE 209 was not included in the list of analytes. The congeners of commercial pentaBDE formulation (BDEs 47, 99, 100, 153, and 154) were detected in all samples and at all stages of the STP process. On average, approximately 83% of total BDEs detected in the STP were comprised of BDEs 47 and 99. BDEs 47 and 99 were found to be associated with the colloidal suspension of particles and the dissolved phase of organic matter in the wastewater influent. In the STP process, the wastewater being treated has a relatively short (12 hours) hydraulic residence time. The authors speculated that the short residence time combined with the low Henry's Law constant, and large estimated aqueous degradation half-lives would mean that loss of both BDEs 47 and 99 by volatilization or degradation during treatment would be negligible. Further, the high octanol/water partitioning coefficients of BDE 47 and BDE 99 (log K_{ow} > 5) drives the compounds to partition to the wastewater solids, and BDE 47 and -99 are

removed from the wastewater with the removal of solids during the treatment process. This, in turn, enriches the sewage treatment plant sludges with BDEs, but reduces their loading with the effluent. The mass balance calculations of PBDEs at this 61 million L/d STP were as follows. Of the 10,560 mg/d total PBDEs entering the STP, approximately 9,609.6 mg/d (91%) ends up in the STP sludges (primary settling and activated sludge), and 950.4 mg/d (9%) are discharged into the surface water with the treated final effluent.

Table 3-8 summarizes the BDE congener distributions in influent, sludges, and final effluent from various surveys of sewage treatment plants in various countries. Generally, North America has higher total BDE concentrations in STP sludge than Europe, with relatively high concentrations of BDE 209. However, BDE 209 was not routinely evaluated in European sludge studies.

3.7. BIOACCUMLATION IN BIOTA

3.7.1. Bioaccumulation in the Aquatic Environment

The hydrophobic and lipophilic properties of BDE congeners cause aquatic organisms to bioaccumulate BDEs with exposures within their food web. up the food chain s. Despite the recent prevalence in the use of the decaBDE commercial formulation, which principally contains BDE 209, the lower brominated BDE congeners are most prevalent in the tissues of aquatic organisms. As discussed in previous sections, the BDE congeners enter a waterbody from atmospheric deposition, air-to-water transfer, or direct discharge from industries and sewage treatment plants. Once BDEs enter the aquatic system they partition between the water column and sediments according to their physical-chemical properties. Moreover, within the water column, BDEs partition between the dissolved phase and the particle-bound phase. Benthic invertebrates and mollusks living and feeding directly on contaminated sediments acquire a body burden of BDEs that is then passed up the food chain from bottom-feeding fish to top piscivorous fish, raptors, reptiles, and mammals. Mollusks internally absorb BDEs from the water column through filtering of the water. Fish acquire BDEs through the dietary pathway, but also from the water passing through their gills. In this regard, trophic level bioaccumulation in marine and freshwater biota is similar to the pattern of bioaccumulation of other classes of lipophilic and persistent compounds such as the

PCBs (Evenset et al., 2005; Bragigand et al., 2006; Eljarrat et al., 2004), i.e. the concentration of PBDEs from one trophic level to another seems to biomagnify between organisms. Bragigand et al. (2006) illustrated this point in the study of trophic level transfers of BDE congeners in an aquatic ecosystem in estuaries of France. They found that the bottom-feeder bivalves and worms had the lowest concentrations of BDE 47, while the water column feeder eels and soles had the highest concentrations. High concentrations were also found in flounder and mid-range concentrations in shrimp.

BDEs 47, 66, 99, 100, 153, and 154 have been detected in multiple species of marine and freshwater organisms. In most cases, BDE 209 has not been included in the list of analytes, and, therefore, it is often suggested that BDE 209 cannot be found in aquatic biota. However, BDE 209 has been detected in hardhead catfish, Atlantic stingrays, sharp-nosed sharks, and bull sharks off the Florida Coast (Johnson-Restrepo et al., 2005; in roach fish in a Baltic Sea estuary (Burreau et al., 2004), and in Mysid shrimp off the Dutch coast (Verslyckea et al., 2005).

For the most part, total PBDEs are highest in top predator marine mammals such as 6,500 ng/g lipid wt (lwt) in whitebeaked dolphin off the Dutch coast (Soni et al., 1998). However, water filtering marine mollusks appear to accumulate BDEs at the highest levels observed in any field survey (e.g., an average total PBDE concentration of 13,502 ng/g lwt in clams from the San Francisco Bay estuary [Oros et al., 2005]). Another observation that can be made from existing studies is that marine fish have higher overall body burdens of total PBDEs as compared to freshwater fish. Marine fish species tend to have total mean PBDE body burdens in a range of < 1.0 to 1,600 ng/g lwt (Antarctic Rockcod and Florida bull shark, respectively), whereas freshwater fish typically have body burdens from 1.0 to 300 ng/g lwt (Detroit River bigmouth buffalo fish and Lake Michigan lake trout, respectively) (Corsolini et al., 2006; Johnson-Restretpo et al., 2006; Valters et al., 2005; Streets et al., 2006).

In most fish (ocean and freshwater) and marine mammals, BDE 47 is the major congener contributing >30% to total body burden of PBDEs. The congener distribution in tissues of aquatic biota usually follow the order BDE 47 > BDE 99 > BDE 100 > BDE 154 > BDE 153 > BDE 49 > BDE 28. In the few studies where BDE 209 was measured in fish tissue (ocean fish), BDE 209 has ranged from < 1 % up to 88% of total PBDE

body burden. Because of small sample size, these studies are insufficient to indicate the general contribution BDE 209 makes to PBDE in fish tissue.

This review of PBDEs detected in fish and marine mammals illustrates the fact that PBDEs are transported long distances, far away from their original source. PBDEs are detected in deep ocean water marine fish and mammals and in marine ecosystems ranging from the equator to the poles. This provides additional evidence for the long-range transport of PBDEs. Details of specific studies measuring BDE congeners in freshwater, marine, farmed, and store-bought fish, including a tabular summary of congener-specific concentrations, are provided in Section 4.6.

3.7.2. Bioaccumulation in the Terrestrial Environment

Field studies of the bioaccumulation of BDEs in terrestrial environment have included fish-eating mammals, birds of prey, and carnivorous mammals in the wild. These animals are top predators in the food chain and acquire a body burden of BDEs from trophic level exposures in a similar manner as aquatic organisms. The following provides a summary of bioaccumulation of PBDEs in terrestrial animals from trophic level exposures.

3.7.2.1. Bioaccumulation in Birds

Birds of prey are ideal candidates to study the bioaccumulation of PBDEs because they are carnivores, typically secondary and tertiary consumers in terrestrial and aquatic food chains. Naert et al. (2007) studied the body burden distribution of BDEs in brain and adipose tissue samples of buzzards (*Buteo buteo*), sparrow hawks (*Accipiter nisus*), cormorants (*Phalacrocorax carbo sinensis*), and blackbirds (*Turdus merula*) from field samples collected at different locations in Switzerland between 2003 and 2005. Lower concentrations of BDE congeners were detected in the brain of avian species as compared to adipose tissue. The median total PBDE concentrations (sum of BDEs 28, 47, 99, 100, 153, 154, and 183) in brain ranged from below the detection limit in blackbirds to 14 ng/g wet weight (wwt) in sparrow hawks. The median concentrations of total PBDEs in adipose tissue ranged from below the detection limit, in blackbirds, to 709 ng/g wwt in sparrow hawks. The authors postulated that the difference in median concentrations

between the two tissues (brain and adipose) is attributed to the blood-brain barrier protecting against the accumulation of BDEs in the brain. In this study, the sparrow hawk had the overall highest median body burden of total PBDEs of all the species: 790.2 ng/g wwt verses the common buzzard (34.55 ng/g wwt), blackbird (0.82 ng/g wwt), and cormorant (98.76 ng/g wwt). BDE 99 was the dominant congener detected in the sparrow hawk accounting for approximately 40% of total PBDEs. BDE 47 body burden was about 26% of total PBDEs. The sparrow hawk is a specialist feeder, and consumes finches, sparrows, and wood pigeons. The cormorant (having the next highest mean PBDE body burden) nests in swampy areas or near large bodies of water such as rivers and lakes. Its diet consists mainly of fish. BDE 47 contributed approximately 42% of the total body burden of PCDEs in the cormorant. In the common buzzard, the most prevalent congeners detected were BDEs 153 (29% of total), 99 (23%), and 47 (22%). The common buzzard preys mainly on small mammals (mice, voles, rabbits, squirrels, rats, moles). The blackbird, on the other hand, feeds principally on seeds and insects, and BDE 47 was the only congener detected in the adipose tissue of this species.

Voorspoels et al. (2006a) investigated the occurrence and distribution of PBDEs in sparrow hawks, common buzzards, and owls of Belgium; the highest mean concentration of total PBDEs was measured in liver of the sparrow hawk (i.e., 4,900 ng/g lwt), followed by the liver of the common buzzard (480 ng/g lwt), and owls (250 ng/g lwt). Congeners can be ordered according to their relative contribution to the total PBDE content as follows for buzzards: 153 > 47 > 99 > 183 > 100 > 154; for sparrowhawks: 99 > 47 >153 > 100 >183 > 154; for owls 153 > 99 > 47 > 183 >100 > 154. BDE 209 was present in 6 out of 44 liver samples and 19 out of 25 serum samples of sparrow hawks, for both common buzzards and owls combined (Voorspoels et al., 2006a). In 2/2 samples of sparrow hawk liver, BDE 209 was detected at a mean concentration of 17 ng/g lwt; and 26 ng/g lwt in serum. In buzzards, BDE 209 was detected only at a frequency of 3/29 (10%) in liver (mean = 79 ng/g lwt). However, in the serum of buzzards the frequency of detection was a surprising 16 out of 20 samples (80%). Chen et al. (2007) also confirmed the presence of BDE 209 in liver of 3 buzzards from Beijing, China (mean = 71 ng/g lwt). BDE 209 was the congener present at the highest concentration—a surprising 48% of total PBDEs.

Later, Voorspoels et al. (2007) again evaluated sparrow hawks, common buzzards, and passerines for body burdens of BDE congeners. The authors found similar results: the highest levels of total PBDEs were detected in the liver of sparrow hawks, at 9,506 ng/g lwt. The liver of the common buzzard had a mean concentration of 727 ng/g lwt, which were considerably lower than liver of sparrow hawks. The PBDE concentrations in adipose tissues and eggs of passerines averaged 160 and 220 ng/g lwt, respectively.

Because the passerine is a favorite prey of the sparrow hawk, Voorspoels et al. (2007) was able to observe the biomagnification potential of BDE congeners in a simple trophic system. The biomagnifications potential (BMP) is derived as the ratio of lipid normalized median BDE congener concentrations in the same tissues of both predator and prey (Voorspoels et al., 2007). A ratio >1 would indicate a potential for biomagnifications between species. Voorspoels et al. (2007) calculated the mean BMP of BDE congeners when passed on through the dietary pathway from passerine to sparrow hawk. The rank of BMP from high to low was BDE 183 = 29; BDE 100 = 25; BDE 154 = 24; BDE 153 = 21; BDE 99 = 20; BDE 47 = 10; and BDE 28 = 4. The mean BMP for the sum of these BDE congeners was 17. It is apparent that the BMP increases from lower brominated to higher brominated BDE congeners within this simple terrestrial food chain. This trend generally tracks the log K_{ow} of the BDE congeners (see Table 3-2). For example, the log K_{ow} of BDE 183 is approximately 8.3, which is considerably higher than the log K_{ow} of BDE 47, which is 6.0.

Chen et al. (2007a) reported on the distribution of BDE congeners in birds of prey (i.e., the common kestrel, the sparrow hawk, the Japanese sparrow hawk, the little owl, the scops owl, and the common buzzard) that were collected in the vicinity of Beijing, China. The sum of the mean PBDE congener concentrations was highest in the common kestrel. Chen et al. (2007a) indicated that these concentrations are among the highest levels in birds reported in the open literature: muscle = 12,300 ng/g lwt, liver 12,200 ng/g lwt, and pooled kidney = 5,340 ng/g lwt. Kestrels dwell in the Beijing area year round, establishing nests and foraging in urban fringe or urban centers. This, in combination with their dietary habits (a preference for small mammals), suggests that the dietary pathway may have caused the high concentrations through biomagnification.

Congener profiles of BDE concentrations in bird tissues indicated a dominance of the more highly brominated congeners. Profiles in the muscle and liver of Chinese sparrow hawks, for example, showed that BDE 153 > 99 > 47 > 183 > 154 > 209 > 207. This differed from the BDE profile in the tissues of Japanese sparrow hawks—99 > 153 > 47 > 209 > 207 > 183. In the liver of scops owl, long-eared owl, upland buzzard and common buzzard, the congener profile was dominated by BDE 209 (followed by BDE 99 and 153). Chen et al. (2007a) attributed the dominance of the higher brominated congeners to the heavy use of commercial octa and deca formulations.

She et al (2008) recently reported on the distribution of PBDEs in piscivorous and omnivorous bird eggs obtained from a San Francisco Bay estuary in California, and from Gray's Harbor, Washington. A total of 169 eggs were collected from the following species, combined: Caspian tern (*Sterna caspia*); Forster's tern (*Sterna forsterii*); California Least tern (*Sterna antillarum brownie*); Clapper rail (*Rallus longirostris obsoletus*). All tern species were fish eating birds, and the clapper rail was omnivorous. The pattern of the five major PBDE congeners detected in the bird eggs (i.e., BDE 47>BDE 99>BDE 100>BDE>153> BDE 154) were consistent with the BDE congener patterns detected in fish. BDE 47 was the most abundant congener and represented approximately 60% of total PBDEs detected in bird eggs. BDE 209 was not detected in any egg sample. Total PBDEs in bird eggs ranged from about 1,080 to 63,300 ng/g lwt. Median concentrations of PBDEs in Caspian tern eggs for 2000–2003 were 2,410, 4,730, 3,720 and 2880 ng/g lwt, respectively, in Forster's terns were 1,820, 4,380, 5,460 and 3,600 ng/g lwt, respectively, and in California Least terns for 2001 and 2002 were 5,060 and 5,170 ng/g lwt, respectively. In contrast, median PBDEs concentration in California Clapper rail eggs for 2001 was 379 ng/g lwt. The rails consumed mostly invertebrate species. The authors concluded that the dietary consumption of PBDE contaminated fish was responsible for the accumulation of PBDE congener in the eggs of fish eating terns.

3.7.2.2. Bioaccumulation in Terrestrial Mammals

Only a very limited number of terrestrial mammals have been studied for their body burden of PBDEs. Many mammals are top predators and, therefore, would be expected to bioaccumulate and biomagnify PBDEs from the food web.

Voorspoels et al. (2006b) studied the red fox for PBDE contamination and food chain bioaccumulation. The red fox is a top terrestrial predator that mainly consumes voles, rabbits, squirrels, and mice as prey. To the extent the prey becomes contaminated with PBDEs, the fox may be exposed to PBDEs that have biomagnified after moving through the trophic system. Voorspoels et al. (2006b) sampled tissue for the occurrence and distribution of PBDEs as a body burden in 33 red foxes indigenous to Belgium and then examined biomagnification of BDE congeners from prey to predator. The median sum of PBDE congeners measured in the foxes were considered low, ranging between 2.2 and 3.4 ng/g lipid weight in adipose tissue, liver, and muscle. BDE 209 was detected at a frequency of 40% in liver, 21% in muscle, and 15% in adipose tissue samples. In fox liver, the BDE 209 congener, on average, constituted 70% of total PBDE concentration— a finding inconsistent with other top predator species. BDE 153 was the most frequently detected congener in all tissues (96%–100% of all samples), whereas BDE 47 was detected in 33, 40, and 100% of adipose, liver, and muscle tissues, respectively. Voorspoels et al. (2006b) did not observe any evidence of metabolic debromination in the fox. The authors observed that total PBDE concentrations were lower in fox tissues as compared to voles and mice (the main diet of foxes). Voorspoels et al. (2006b) speculated that this may be due to the high capacity of the fox to metabolize lower brominated BDEs in vivo, similar to what has been observed in the grizzly bear (Christensen, et al., 2005). The authors postulated that this high metabolic activity might be related to the fact that no biomagnification of BDE congeners between prey and predator was observed in the fox (Voorspoels et al., 2007). The study of PBDE distributions in foxes showed that BDE 209 does, however, bioaccumulate in terrestrial top predators such as the red fox (Voorspoels et al. 2006b).

Christensen et al. (2005) reported on the influence of diet on the BDE congener distribution and other persistent organic contaminants in the tissues of 12 grizzly bears (6

each from coastal and interior areas) in British Columbia, Canada. Dietary consumption of meat was estimated to be 0 to 19% and 13 to 61% of total diet for interior and coastal-maritime bears, respectively. Maritime bears mostly consumed salmon as the primary meat source, whereas the meat source of interior bears was more varied. The remaining diet of all bears consisted of vegetation. With the exception of PBDEs, the persistent organic chemicals (POPs) were higher in the tissues of the bears that consumed meat. However, there was no significant difference in total PBDE tissue burden or congener distribution between the two groups of bears. Total PBDEs dominated total POP concentration in interior bears but not in maritime bears. The ranking by higher-to-lower total contaminant concentration of POPs in the tissues of interior and maritime bears showed the following pattern respectively: PBDEs >PCBs > HCB > HCH > CHL > DDT and PCBs > CHL> HCB > DDT > PBDEs > HCH. Both bear groups showed a marked difference in the mean BDE congener profile. Maritime fish-eating bears displayed a BDE congener pattern dominated by BDE 47 followed by 209 > 99 > 100 > 153. In the interior bears the tissues were dominated by BDE 209 followed by 206 > 47 > 207 > 208. The predominance of the lighter congener BDE 47 in the maritime bears suggests that PBDE exposure mainly occurs through the fish consumption pathway. It was postulated that the fish acquire PBDE contamination as a result of the long-range atmospheric transport and deposition into the marine food web. The authors speculated that the dominance of BDE 209 in the interior bears, whose diet was richer in vegetation than the maritime bears, may indicate the possibility of the PBDE exposures via the pathway of air-to-plant-to bear. In this paradigm the vegetation accumulates PBDEs from local source air emissions of decaBDE and passes BDE 209 onto the bear through its diet. This notion was supported by the observations that BDE 209 is not detectable in fish, and the higher BDE congeners had negative bioaccumulation slopes in the tissue of bears indicating a preferential exposure to local sources through their consumption of terrestrial vegetation (Christensen et al., 2005).

The polar bear diet is chiefly comprised of ringed seal. Wolkers et al. (2004) studied the cod-seal-polar bear bioaccumulation pattern of PBDEs in an Arctic food web and found a food chain pattern consistent with what might be expected for the BDE 47 congener: BDE 47 concentrations in polar bear were great than those in seal, which in

turn were greater than those in polar cod. From the 22 brominated compounds measured, only BDEs 47, 85, 99, 100, and 154 were detected in polar cod. In addition to these congeners, the tissues of ringed seals contained BDE 66. Concentrations of PBDEs in ringed seals were overall higher than in cod. In particular, BDE 47 and 99 were approximately one order of magnitude higher in seals than in cod. PBDE 47 comprised more than 90% of the total PBDEs present in ringed seals. Only BDE 47 was detected in polar bears. Male and female polar bears were found to have a body burden of BDE 47 that was 1.5 and 3 times the body burden of seals, respectively.

3.7.3. Bioaccumulation in Insects

Insects may acquire a body burden of PBDEs through contact with contaminated environmental media or through the dietary pathway. Only a very limited number of species of aquatic insects, caddisflies (trichoptera), and midges (diptera), have been investigated for the occurrence and distribution of PBDE in insect tissues (Bartrons et. al 2007). Most insect species undergo a number of developmental transformations from larva and pupa to adult, and they begin their life cycle in aquatic sediments. Insects are at the beginning of the food chain in most aquatic ecosystems. It has been noted that pupae emerge during periods of high fish activity and are an attractive source of food to many fish species (Bartrons et. al 2007). Therefore, in terms of movement through trophic systems and biomagnifying from lower to higher predators, insects can be viewed as an important beginning to contamination of PBDEs and other persistent organic pollutants in aquatic organisms.

Bartrons et al. (2007) obtained 22 samples of larvae and pupae of four variants of caddisflies and midges from two, high-altitude mountain lakes in the Pyrenees Mountains of Spain. Samples of the larvae and pupae of trichoptera belonged to the polycentropodidae and limnephilidae families, and samples of diptera belonged to the chironomidae and ceratopogonidae families, thus producing samples from a total of four distinct insect types. Total concentrations of PBDEs in insect larva ranged from 0.65–1.68 ng/g dwt and 0–13.07 ng/g dwt for caddisflies and midges, respectively. Total PBDEs in insect pupa ranged from 5.17–9.32 ng/g dwt in caddisflies and 3.91–27.38 ng/g dwt in midges. In general, pupae contained significantly higher concentrations of total

PBDE than larvae of the same taxonomic group. BDE 209 was detected in the larva and pupa of the limnephilidae and polycentropodidae families of caddisflies but not in the ceratopogonidae and chironomidae families of midges flies. Caddisfly pupa of the polycentropodidae family had the highest overall level of BDE 209 (4.93 ng/g dwt). BDE 47 was the most consistently detected congener in larva and pupa of midges and caddisflies, with the exception of midge larva of the chironomidae family where it was not detected.

3.8. ENVIRONMENTAL TIME TRENDS

Sediment cores, archived vegetation and biological tissue samples have been used to infer time trends of the levels of PBDEs in the environment. The dated sediment cores and archived vegetation provide a clear record of the varying PBDE concentration over the decades. Whale blubber, bird eggs, and bird fat have been studied at different time intervals and can be used to evaluate the time trends of PBDE levels in biological tissues. The following is a limited review of these studies; further detail can be found in the studies themselves. These studies all show a similar trend: that PBDEs were absent in the environment until their introduction as flame retardant products in the 1970s. Their presence in the environment increased throughout the remainder the 20th century into the 21st century.

3.8.1. Time Trends from Sediment Core Studies

Sediment core studies have been used to study 20th century temporal trends in environmental levels of such contaminants as PCBs, dioxins, and PBDEs. The approach is to take several cores usually within a lake (which is quiescent unlike moving water bodies such as rivers), vertically , and date sectionssectioning the cores, and dating the slices using radiotracers. Once a ctionsection is dated, then a measurement of the contaminant in that segment provides an indication of relative environmental levels during that time period. Further, knowing the deposition rate of sediments in that particular water body, the total loading into that water body can be estimated. This approach is useful for persistent contaminants that predominantly partition to benthic sediments. These characteristics are true for persistent and bioaccumulative toxics

(PBTs) such as PBDEs. This section provides an overview of sediment core studies undertaken for PBDEs around the world and what has been learned from them.

Li et al. (2006) reported on the chronology of deposition flux of PBDEs into the sediments of all five Great Lakes (Lake Michigan, Lake Huron, Lake Superior, Lake Ontario, and Lake Erie) and three inland seepage lakes. Their work was also chronicled in three literature articles (Song et al., 2005a, b; 2004). Twenty-two sediment cores were collected in 2001 and 2002 and horizontally sectioned into a total of 247 samples (175 from the Great Lakes and 48 from the inland lakes). Analytical results are separately reported as the sum of nine PBDE (Σ_9BDEs) congeners (BDE 28, 47, 66, 85, 99,100, 153, 154, and 183) and BDE 209. The range of mean surface sediment concentrations in all the Great Lakes was 1.4–5.6 ng/g dwt and 10.5–226.6 ng/g dwt for the Σ_9BDEs and BDE 209, respectively. BDE 209 dominated the total PBDE concentrations in all the lake sediments. Figure 3-1 graphically displays the temporal trends of deposition fluxes of total PBDE and PCBsto the sediments in each of the Great Lakes as determined by Li et al. (2006). In general, PBDE levels began to rise between 1920 and 1950. There is a striking increase in the deposition of total PBDE to the lake sediments from 1970's to 2002. The temporal trends in the dated sediment cores from this study suggest that the increase in PBDE input to the Great Lakes tend to be first order. From these data Li et al. (2006) calculated concentration doubling times (t_2) ranging from 9 year to 43 years for Σ_9BDEs and from 7 to > 70 years for BDE 209. There is no evidence of any recent decline in PBDE loadings to the Great Lake sediments. Li et al. (2006) also estimated the annual PBDE loading rates to the surface sediments of the Great Lakes by multiplying the mean surface flux times the total surface area of the lakes. According to their calculations, the Great Lakes received approximately 0.17 tons of Σ_9BDEs and 4.4 tons of BDE 209 in 2002, primarily from atmospheric deposition. Total deposition of BDE 209 to the Great Lakes was estimated to be over 25 times the deposition of the sum of BDE 28, 47, 66, 85, 99,100, 153, 154, and 183 combined.

Qiu et al. (2007) evaluated the distribution of flame retardants Dechlorane Plus (DP), 1,2-bis-(2,4,6-tribromophenoxy)ethane (TBE), and PBDEs in dated sediment layers in a sediment core study of Lake Ontario. They measured 20 BDE congeners, including BDE 209. In the sediment surface corresponding to 2004, Qiu et al. (2007) detected Σ_3.

7PBDE congeners (i.e., tri thru hepta BDE congeners 28, 47, 49, 99, 100, 116, 153, 154, 181, and 183) at a concentration of 2.8 ng/g dwt. The concentration of BDE 209 was approximately 15 ng/g dwt in the surficial layer, which was approximately 5 times higher than the sum of the other BDE congeners. The authors postulated that the sedimentary record of PBDEs in Lake Ontario reflected atmospheric deposition as the primary transport mechanism to the lake. BDE 209 was initially detected in Lake Ontario sediments around 1980 and underwent a dramatic increase from 1990 to 2000. Σ_{3-7}PBDE congeners were initially detected around 1955, gradually increased in concentration between 1960 and 1990, and sharply increased in concentration from 1990 through 2000.

In 2004, Zhu and Hites (2005) conducted core studies for 18 BDE congeners from two study sites: Lakes Michigan and Erie. Like the other Great Lakes studies, BDE 209 predominated, making up 95-99% of the concentration. The concentration of BDE 209 is 315 and 39 ng/g dwt in Lakes Michigan and Erie, respectively, while all other congeners measured 2.6 and 1.1 ng/g dwt in Lakes Michigan and Erie, respectively. This article plots the rise in concentration, showing how BDE 209 was about 10 ng/g dwt in 1960 in Lake Michigan and rose to above 300 ng/g dwt by the early 2000s. The sum of the others similarly started well below 1.0 ng/g in 1960 to end up at 2.6 by the 2000s. Similar trends were seen in Lake Erie, with 209 not showing up until 1980 at all at 1 ng/g dwt to rise to 38 by the 2000s.

Marvin et al. (2007) studied the temporal trends of PBDEs in archived freeze-dried samples of suspended sediment taken from the water column of the Niagara River feeding into Lake Ontario. The archive contained samples that had been collected from 1980 to 2002. The Niagara River flows into Lake Ontario, and there has been a history of heavy industry along its river banks. A total of 16 BDE congeners were evaluated in the samples. PBDE concentrations in the suspended sediments significantly increased between the time periods 1980 and 1988. However, the most current samples indicate a decline in total PBDE concentration in suspended sediments since 2001. This is different than the sediment core studies above, which show increases—at least until the last dating time at 2004. This difference could be attributed to the differences in the type of samples taken (suspended particles verses dated sediment core layers) and the fact that BDE 209 was not specifically analyzed in the samples. Another factor that may contribute to this

difference is that Marvin et al. (2007) indicated that the primary inputs to the Niagara River were industrial point sources in the watershed, whereas inputs of PBDEs to the Great Lake sediments were primarily from atmospheric deposition.

Chen et al. (2007b; with earlier discussions of the partial data set also in Mai et al., 2005) examined the temporal trends of 3 dated sediment cores obtained from the Pearl River delta in China. A total of 17 congeners were measured in these cores. Generally, the total concentrations increased from 1975 to the late 1980's and early 1990's. BDE 209, in particular, displayed an exponential increase in concentration in the sediments from the time period 1990 to 1995 and the year 2005. Chen et al. (2007b) surmised that the sediments reflect the dominant use of the pentaBDE formulation prior to 1990, and a subsequent dominance of the use of decaBDE from 1990 onward. Deposition flux of BDE congeners not including BDE 209 ranged from 5.8–106.2 ng/cm^2, with an average of 56.0 ng/cm^2, while the deposition flux of BDE 209 was higher ranging from 172.8 to 563 ng/cm^2. Chen et al. (2007b) estimated the total deposition flux of total congeners, not including BDE 209 and BDE 209, to the Peal River delta in 2005, to be 2.1 and 29.7 MT, respectively.

Minh et al. (2007) analyzed the occurrence and distribution of 11 BDE congeners, including BDE 209, in three dated sediment cores of Tokyo Bay. The investigators reported results in terms of the sum of BDE 3 through BDE 207 (ΣPBDEs) and BDE 209, separately. There were differences in the chronology of mass concentration of PBDEs from one sediment core to another. In addition there was a clear concentration gradient of ΣPBDEs and BDE 209 in the surface sediments, with the sediments at the mouth of Tokyo Bay having the highest concentrations. The authors indicated that the apparent concentration gradient clearly demonstrated that populated areas such as the cities of Tokyo and Yokohama are major emission sources of PBDEs to the bay. Core dating data suggested that the ΣPBDEs concentrations consistently increased beginning in 1945 and reached a maximum in 1988, with concentrations at about 3 ng/g dwt or less at that maximum. There appeared to a slight decrease from 1988 to the surface sediments representative of the year 2000. BDE 209 appeared in the cores in the 1960s, reaching maximums between 20 and 80 ng/g dwt in 2000, the last year of dating. The authors explained that the apparent decease in the ΣPBDEs concentration in the mid 1990s may

reflect Japan's phase-out and reduction in the general use of penta- and octaBDE commercial formulations in the early 1990s, and similarly, that the continual rise of BDE 209 reflects ongoing and increasing use of decaBDE.

Stern et al. (2005) reported the results of a sediment core study of a remote northern Arctic lake on Devon Island in Canada. A number of persistent organic pollutants, including PBDEs, were investigated in order to observe the temporal trends of contaminant loadings. Four sediment cores were collected in 1999 and archived in a freezer. Core #2 was analyzed for the concentration of PBDEs. Maximum deposition of total PBDEs occurred in the most recent surface sediments with a depositional flux estimated at 28.5 $ng/m^2/yr$. BDE 47 was the most abundant congener followed by BDE 99 and BDE 100. These three congeners represented about 80% of total PBDEs determined in the sediment core slices. The authors speculated that this congener pattern may be indicative of anaerobic microbial decomposition of more highly brominated PBDEs in the sediments, although there was no direct proof this had occurred (Stern et al., 2005). The authors concluded that the contamination of the remote Arctic lake with PBDEs was the result of the long-range transport and deposition into the lake, because there were no local sources of these contaminants.

Evenset et al. (2007) undertook a sediment core study to examine the historical deposition of PBDEs and other persistent organic pollutants into the sediments of Lake Ellasjoen, a remote lake on an island in the central Barents Sea of the Norwegian Arctic. Four replicate sediment cores were collected in April, 2001 from a depth of 34 m and measured for 10 BDE congeners (not including BDE 209). PBDEs could only be detected in the upper 4 cm of the sediment core, which corresponds to a time range encompassing the 1940's through 2001. Of the ten BDEs, only BDEs 28, 47, 99, 100, and 153 were detected. They were first detected in core segments corresponding to 1953, at levels of 0.1 ng/g dwt and less, and they continuously rise in concentration to 1994, with the maximum individual congener concentration of 0.45 ng/g dwt. This dominant congener was BDE 47, followed by BDE 99, 28, and 100. BDE 153 was only found in the surface sediments to the lake. The primary route of entry of BDE congeners was assumed to be from atmospheric deposition, thus indicating the long-range transport of PBDEs (Evens et al., 2007).

Zegers et al. (2003) summarized various studies on the chronology of PBDE concentrations in dated sediment cores collected in Europe. The sediment core samples were obtained from the Oslofjord river in Norway; from the marine sediments of the Wadden Sea off the coast of The Netherlands; from Lake Woserin in the state of Mecklenburg-Vorpommern, Germany; and from the Kimmeridge clay formation in the United Kingdom, a marine formation from the Jurassic period. Pb-210 and Cs-137 (one or both) isotopes were used to mark distinct years in the age of the sediment layers. The findings of all of these studies are similar to all of the previously described core studies: Zeger at al. (2003) showed rising concentrations from about the 1970s to the present, with dominance of BDE 209, particularly in the later years. From the Drammenfjord River in Norway, BDE 47 initially appeared in the sediments in 1975 at 1.2 ng/g dwt and steadily increased to a level of 5.8 ng/g dwt in 1999. Similarly, BDE 209 also first appeared in the sediments in 1975, but it increased at a sharper rate than BDE 47. The surface sediment concentration of BDE 209 is 80-fold higher than in 1975, rising from 1.3 to 105 ng/g dwt. The concentration pattern from high to low suggests that BDE 209>BDE 99>BDE 47>BDE 100. In Lake Woserin in Germany, the oldest sediment layer was dated to 1628, and the most current was 1997. BDE congeners initially appeared in 1973, continued to gradually increase, and reached a peak in 1994, with slight decreases seen for all congeners from 1994 to 1997. BDE 99 was the most abundant congener in the sediments, and increased 3-fold in concentration from 1973 to 1994 to a high of 11.3 ng/g dwt. Unlike other study results, BDE 209 did not dominate the profile. Rather, it had concentrations similar to BDEs 99 and 47, reaching a high of 10.7 ng/g dwt in 1994. Clay layers from the Kimmeridge Clay Formation (Blackstone-Band) in southern England, dated from the Jurassic period 100,000 to 150,000 year ago, showed no detectable PBDE congeners. BDE congeners initially appeared in 1965 in cores from the western Wadden Sea off the coast of the Netherlands, although the dating resolution of decades was not sufficient to determine if PBDEs were present from 1945 to 1965. All detectable BDE congeners increased in concentration from their initial appearance until 1989. From 1989 to 1995, all congeners slightly decreased in sediment concentration. BDE 209 was the most abundant congener in the sediments from 1978 through 1995, reaching a high concentration of 380 ng/g dwt. In 1995 BDEs 47 and 99

reached high concentrations—about 20 ng/g dwt. Concentrations of 12 other congeners were mostly non-detects (at detection limits of about 1-3 ng/g dwt).

3.8.2. Time Trends from Aquatic Wildlife Samples

The body burden of PBDEs and other persistent organic pollutants in whales, seals, and different fish species have been measured over discreet time intervals. This provides a basis for assessing the changes in relative body burdens in wildlife with the passage of time. Like sediment core studies, these studies show a rise of BDE congeners through the 1990s into the 2000s. However, very different than sediment core studies, these show a predominance of BDE 47 and a virtual absence of BDE 209. The absence of BDE 209 is also true with human blood and tissue sampling (see Chapter 5).

Lebeuf et al (2004) studied the levels and temporal trends of PBDEs in the blubber of 54 stranded adult beluga whales from the St. Lawrence Estuary in Quebec, Canada. The beaching of whales on the shores of the St. Lawrence Estuary had occurred in the time period 1988 through 1999, and samples were collected during these years and frozen for future analysis. The total PBDE concentration (including BDEs 28, 47, 71, 77, 99, 100, 153, 154, 155, and 183) ranged from about 20 to almost 1000 ng/g wwt in samples from 54 whales. The data suggests an exponential increase of PBDE congeners from 1988 to 1999. The authors estimate the doubling times of BDE congeners to range between 2 and 9 years for all congeners (Lebeuf et al., 2004).

Kajiwara et al. (2004) used archived fur seal adipose tissue samples to investigate the relative time-trends of PBDEs in coastal waters of Japan. Ten fat samples had been collected over a period between 1972 and 1998 and stored in the Environmental Specimen Bank for Global Monitoring at Ehime University. Kajiwara et al. (2004) analyzed tissue samples for the presence of PCBs, DDT, and BDEs (3, 15, 28, 47, 99, 153, 154, 183, and 209). The sum of the PBDE congeners ranged in concentration from a low of 0.33 ng/g lwt in the year1972 up to 100 ng/g lwt in the year1994. BDE 47 was the most abundant congener of the total PBDEs in all samples analyzed. No BDE 209 was detected in the fur seal fat samples despite the fact that the decaBDE formulation constituted 67% and 100% of total commercial PBDE usage in Japan in 1985 and 2000, respectively.

Batterman et al. (2007) reported on the time-trends of PBDEs in archived frozen tissues samples from rainbow smelt, walleye, and lake trout obtained from the Great Lakes over the time period 1979-2005. All fish species had been collected from sites in Lakes Erie, Huron, Michigan, Ontario and Superior every other year as part of a monitoring program conducted by the U.S. Environmental Protection Agency (U.S. EPA). Only BDE congeners 47, 99, 100, and 153 were investigated in this study due to their usual pattern of dominance in fish tissue. The total PBDE concentrations in trout at each of the lakes increased exponentially and rapidly over the period from 1979–1980 to the mid-1990s. Concentrations were always highest for BDE 47, rising to as high as 50 - > 100 ng/g wwt in the latest samples in all lakes, while concentrations of the other three congeners were between 5 and 10 times lower than BDE 47, rising to no more than 15 ng/g wwt. Doubling times were calculated for all species and lakes. They ranged from about 2 to over 20 years. The relatively stable congener pattern in all lakes across all fish species (i.e., BDE 47 > PBE 100 > PBE 99 > PBE 153) suggests that atmospheric deposition was the primary reason for the presence of PBDEs in the Great Lakes. The authors claim that there is no evidence to suggest any declines in PBDE concentrations in the Great Lakes region.

Rayne et al. (2003b) reported on the time trends of PBDEs in tissues of 41 mountain whitefish and 6 sucker fish in the Columbia River system in southeastern British Columbia, Canada. Eleven congeners were measured, but not BDEs 183 and 209. From 1992 to 2000 the ΣPBDE congeners in whitefish increased 11.8-fold and 6.5-fold in fish caught near the towns of Genelle and Beaver Creek along the Columbia River, respectively. The authors calculated a short doubling time of total PBDEs of 1.6 years between the years 1995 and 2000 in whitefish caught near Genelle. At the confluence of Beaver Creek and the Columbia River (25 miles further downstream from Genelle and 9 miles downstream of a secondary metal smelting operation), the PBDE concentrations increased over 6-fold from 1992 to 2000—from 4.5 ng/g wwt in 1992 to 29.2 ng/g wwt in 2000. In whitefish caught on the Slocan River in an unpopulated pristine area that was not directly impacted by urban or industrial activities, PBDE concentrations were 0.9 ng/g wwt in 1996, which is about 20-50 times lower than those at other testing locations

near towns and possible sources of PBDEs. Based on concentration differences in species, the authors speculated that PBDEs bioaccumulated less in sucker than whitefish.

3.9. CONCLUSIONS

This chapter focused on the environmental fate of PBDEs. Sections have discussed fate properties, movement and transformations in the environment, temporal trends in the environment, and also evidence for metabolic transformations, specifically debromination, in animals and humans. The following conclusions are made:

1. PBDE congeners are lipophilic and persistent organic compounds having a strong propensity for bioaccumulation and biomagnifications in the aquatic and terrestrial food webs.

2. PBDE congeners are ubiquitous environmental contaminants and are detected globally in air, soils, sediments, oceans, and wildlife.

3. The atmospheric transport and surface deposition of PBDEs is the primary means of distributing PBDEs over long geographical distances.

4. The detection of low molecular weight BDE congeners in Arctic air suggests long-range atmospheric transport of these contaminants from industrialized countries.

5. Once released into the air, PBDEs partition between the vapor and particle bound phases in the atmosphere in accordance with their respective VPs. The BDE congeners with 1–4 bromines atoms primarily exist in the vapor phase; BDE congeners with 5–6 bromine atoms more equally partition between the vapor phase and the particle bound phase, and BDE congeners having >6 bromine atoms are primarily adsorbed to atmospheric particles.

6. Photochemical reaction with the hydroxyl radical appears to be an insignificant atmospheric degradation pathway for vapor phase BDE congeners.

7. High molecular weight BDE congeners exhibit a propensity for the breakdown and decomposition both in soils and air by UV light (i.e., photolysis). Photo-degradation in air and soils can debrominate the higher BDE congeners to form

lower brominated BDE congeners, and may be a significant degradation pathway in the environment.

8. Ocean currents also play a significant role in globally distributing PBDE contamination in aquatic food webs and in terrestrial food webs connected to the aquatic environment.

9. Higher brominated PBDE congeners can undergo metabolic debromination in fish, mammals, and birds to form lower brominated congeners.

10. PBDEs can undergo reductive debromination by anaerobic microorganisms.

11. Soils and sediments are environmental sinks for PBDEs.

12. There exists good evidence that microbial anaerobic degradation naturally occurs in sediments and soils. However, the degradation rate has yet to be determined. Also, there is evidence of photolysis of PBDE in soils and suspended sediments, although again degradation rates are unavailable.

13. Although BDE 209 dominates in soils and sediments in North America, BDE 47 usually dominates in fish tissues. BDE 209 has a low potential for bioaccumulation, and once absorbed, may be easily excreted. In the human and other animals (see Chapter 5), BDE 209 has a half-life on the order of weeks whereas other BDE congeners, including BDE 47, have half-lives on the order of years.

14. Since the 1970s, sediment core samples show predominance and a starker rise of BDE 209, while the animal tissue samples show predominance and rise in BDE 47.

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Table 3-1. Estimated water solubility values for PBDEs.

Brominated diphenyl ether	Aqueous Solubility (mg/L @ 25° C)	Reference	Solubility score
Deca-BDE (Commercial)	< .001	European Union, 2002	Low
Octa-BDE (Commercial)	0.005 0.002	European Union (2003) ATSDR (2004)	Low
Penta-BDE (Commercial)	0.013	European Union (2001)	Low
BDE 11	0.088	Palma et al. (2002)	Low
BDE 15	0.13	ATSDR (2004)	Low/Medium Borderline
BDE 17	0.026	Palma et al. (2002)	Low
BDE 18	0.026	Palma et al. (2002)	Low
BDE 28	0.07	ATSDR (2004)	Low
BDE 32	0.026	Palma et al. (2002)	Low
BDE 35	0.005	Palma et al. (2002)	Low
BDE 37	0.005	Palma et al. (2002)	Low
BDE 39	0.005	Palma et al. (2002)	Low
BDE 47	0.001–0.002	Palma et al. (2002) ATSDR (2004)	Low
BDE 66	0.018	ATSDR (2004)	Low
BDE 77	0.006	ATSDR (2004)	Low
BDE 85	9×10^{-7}–8×10^{-5} 0.006	Palma et al. (2002) ATSDR (2004)	Low
BDE 99	9×10^{-7}–2.4×10^{-3} 0.009	Palma et al. (2002) ATSDR (2004)	Low
BDE 100	0.04	ATSDR (2004)	Low
BDE 116	9×10^{-7}–8×10^{-5}	Palma et al. (2002)	Low
BDE 119	9×10^{-7}–8×10^{-5}	Palma et al. (2002)	Low
BDE 128	4.15×10^{-6}	Palma et al. (2002)	Low
BDE 138	0.001	ATSDR (2004)	Low
BDE 153	0.001 0.0009	ATSDR (2004) Tittlemier et al. (2002)	Low
BDE 154	0.001 0.0009	ATSDR (2004) Tittlemier et al. (2002)	Low
BDE 172	2.16×10^{-7}	Palma et al. (2002)	Low

BDE 176	2.16×10^{-7}	Palma et al. (2002)	Low
BDE 181	2.16×10^{-7}	Palma et al. (2002)	Low
BDE 183	0.002 0.0015	ATSDR (2004) Tittlemier et al. (2002)	Low
BDE 185	2.16×10^{-7}	Palma et al. (2002)	Low
BDE 190	2.16×10^{-7}	Palma et al. (2002)	Low

Solubility score is as follows: low = relatively insoluble; medium = somewhat soluble; high = very soluble

Table 3-2. Estimated octanol water partition coefficients (log K_{ow}) values for PBDEs.

Brominated diphenyl ether	Log K_{ow} coefficient	Reference
Deca-BDE (Commercial)	6.27 6.27	ATSDR, 2004 European Union (2003)
Octa-BDE (Commercial)	6.29 6.29	ATSDR, 2004 European Union (2003)
Penta-BDE (Commercial)	6.64–6.97 6.57	ATSDR, 2004 European Union (2001)
BDE 11	5.83	Palma et al. ,2002
BDE 15	5.86 5.74 5.55	Kuramochi et al., 2004 ATSDR, 2004 Tittlemier et al.(2002)
BDE 17	5.52–5.88	Palma et al. (2002)
BDE 18	5.52–5.88 5.74	Palma et al. (2002) ATSDR, 2004
BDE 28	5.94 5.98	ATSDR (2004) Tittlemier et al.(2002)
BDE 32	5.52–5.88	Palma et al. (2002)
BDE 35	5.52–6.72	Palma et al. (2002)
BDE 37	5.52–6.72	Palma et al. (2002)
BDE 39	5.52–6.72	Palma et al. (2002)
BDE 47	6.01–6.77 6.81 6.48 6.55	Palma et al. (2002) ATSDR (2004) Kuramochi et al. (2004) Tittlemier et al. (2002)
BDE 66	6.73	Tittlemier et al. (2002)
BDE 77	6.73	Tittlemier et al. (2002)
BDE 85	6.57–7.66 7.03	Palma et al. (2002) Tittlemier et al. (2002)
BDE 99	6.53–7.66 7.32 7.21 7.13	Palma et al. (2002) ATSDR (2004) Kuramochi et al. (2004) Tittlemier et al. (2002)
BDE 100	7.24 6.86	ATSDR (2004) Tittlemier et al. (2002)
BDE 116	6.71–7.66	Palma et al. (2002)
BDE 119	6.71–7.66	Palma et al. (2002)
BDE 128	7.39– 8.55	Palma et al. (2002)
BDE 138	7.91	Tittlemier et al. (2002)

BDE 153	7.9 7.83 7.62	ATSDR (2004) Kuramochi et al. (2004) Tittlemier et al. (2002)
BDE 154	7.82 7.39	ATSDR (2004) Tittlemier et al. (2002)
BDE 172	9.44	Palma et al. (2002)
BDE 172	9.44	Palma et al. (2002)
BDE 176	9.44	Palma et al. (2002)
BDE 181	9.44	Palma et al. (2002)
BDE 183	8.27	ATSDR (2004)
BDE 185	9.44	Palma et al. (2002)
BDE 190	9.44 8.36	Palma et al. (2002) Tittlemier et al. (2002)

Table 3-3. Estimated Henry's Law constants (H) for PBDEs

Brominated diphenyl ether	(H) (atm-m³/mol @ 25°C)	Reference
DecaBDE (Commercial)	1.20E-08 3.95E-07	ATSDR, 2004 Cetin et al. (2005)
OctaBDE (Commercial)	7.5E-08–2.6E-07	ATSDR, 2004
PentaBDE (Commercial)	3.5E-06–1.2E-05	ATSDR, 2004
BDE 15	2.07E-04 2.10E-04	ATSDR, 2004 Tittlemier et al. (2002)
BDE 28	5.03E-05 7.80E-05 5.10E-05	ATSDR (2004) Cetin et al. (2005) Tittlemier et al. (2002)
BDE 47	1.48E-05 8.39E-06	ATSDR (2004) Cetin et al. (2005)
BDE 66	4.93E-06	ATSDR (2004)
BDE 77	1.18E-05	ATSDR (2004)
BDE 85	1.09E-06	ATSDR (2004)
BDE 99	2.27E-06 5.92E-06	ATSDR (2004) Cetin et al. (2005)
BDE 100	6.81E-07 2.37E-06	ATSDR (2004) Cetin et al. (2005)
BDE 153	6.61E-07 4.34E-06	ATSDR (2004) Cetin et al. (2005)
BDE 154	2.37E-06 7.90E-07	ATSDR (2004) Cetin et al. (2005)
BDE 183	7.3E-08	ATSDR (2004)
BDE 209	3.95E-07	Cetin et al. (2005)

Table 3-4. Estimated solid phase vapor pressures (P_S) and subcooled liquid vapor pressures (P_L) of some PBDEs (Pascals at 25°C)

Brominated diphenyl ether	T_m (K)	T_m Reference	P_S	P_L	P_L Reference
Deca-BDE (Commercial)	573.15	European Union	9.28E-09	4.63E-06	European union (2002)
Octa-BDE (Commercial)	473.15	European Union	1.26E-07	6.59E-06	European union (2003)
Penta-BDE (Commercial)	475.15	European Union	8.60E-07	4.69E-05	European union (2001)
BDE 1	NA			0.163	Wong et al. (2001)
BDE 2	NA			0.128	Wong et al. (2001)
BDE 3	NA			2.59E-01	Tittlemier et al. (2002)
BDE 7	NA			1.68E-02	Wong et al. (2001)
BDE 8	NA			1.37E-02	Wong et al. (2001)
BDE 10	NA			2.77E-02	Wong et al. (2001)
BDE 12	NA			1.19E-02	Wong et al. (2001)
BDE 15	329.15	Marsh et al. (1999)	8.59E-03 4.88E-03	1.73E-02 9.84E-03	Tittlemier et al. (2002) Wong et al. (2001)
BDE 28	337.15	Marsh et al. (1999)	9.07E-04 6.51E-04	2.19E-03 1.57E-03	Tittlemier et al. (2002) Wania et al. (2003)
BDE 30	358.15	Marsh et al. (1999)	1.18E-03	4.56E-03	Wong et al. (2001)
BDE 32	350.40	Palm et al. (2002)	6.91E-04	2.25E-03	Wong et al. (2001)
BDE 33	NA		1.49E-03	1.49E-03	Wong et al. (2001)
BDE 35	413.01	Palm et al. (2002)	1.04E-04	1.39E-03	Wong et al. (2001)
BDE 37	321.65	Palm et al. (2002)	6.00E-04	1.02E-03	Wong et al. (2001)
BDE 47	353.65	Palm et al. (2002)	7.42E-05 5.52E-05	2.50E-04 1.86E-04	Wong et al. (2001) Tittlemier et al. (2002)
BDE 66	NA			2.38E-04 1.22E-04	Wong et al. (2001) Tittlemier et al. (2002)
BDE 69	NA			4.00E-04	Wong et al. (2001)
BDE 75	408.15	Marsh et al. (1999)	4.10E-05	4.92E-04	Wong et al. (2001)
BDE 77	368.15	Marsh et al. (1999)	3.21E-05 1.40E-05	1.56E-04 6.79E-05	Wong et al. (2001) Tittlemier et al. (2002)
BDE 82	NA		4.80E-05	6.47E-05	Wong et al. (2001)

BDE 85	396.45	Palm et al. (2002)	1.07E-06	9.86E-06	Tittlemier et al. (2002)
BDE 99	365.45	Palm et al. (2002)	1.49E-05 3.85E-06 7.94E-06	6.82E-05 1.76E-05 3.63E-05	Wong et al. (2001) Tittlemier et al. (2002) Wania et al. (2003)
BDE 100	371.15	Marsh et al. (1999)	5.50E-06 7.07E-06	2.86E-05 3.68E-05	Tittlemier et al. (2002) Wania et al. (2003)
BDE 115	NA			3.02E-05	Wong et al. (2001)
BDE 138	NA			1.58E-06	Tittlemier et al. (2002)
BDE 153	456.15	Palm et al. (2002)	1.63E-07 5.80E-06	5.80E-06 2.09E-06	Wong et al. (2001) Tittlemier et al. (2002)
BDE 154	416.15	Marsh et al. (1999)	2.64E-07	3.80E-06	Tittlemier et al. (2002)
BDE 183	NA			4.68E-07	Tittlemier et al. (2002)
BDE 190	470.4	Palm et al. (2002)	1.85E-08 5.76E-09	9.05E-07 2.82E-07	Wong et al. (2001) Tittlemier et al. (2002)

Note: P_S was calculated from P_L using equation 3-1 and assuming the melting point temperature (T_m) in the table. NA = not available in the literature.

Table 3-5. Estimated octanol/air partition coefficients (log K_{oa}) of PBDEs

Brominated Diphenyl Ether	Log K_{oa} (@ 25° C)	Reference
BDE 1	7.24	Wania et al. (2002)
BDE 2	7.36	Wania et al. (2002)
BDE 7	8.37	Wania et al. (2002)
BDE 8	8.47	Wania et al. (2002)
BDE 10	8.12	Wania et al. (2002)
BDE 12	8.55	Wania et al. (2002)
BDE 13	8.57	Wania et al. (2002)
BDE 15	8.64	Wania et al. (2002)
BDE 17	9.30	Harner and Shoeib (2002)
BDE 21	9.49	Wania et al (2002)
BDE 28	9.50	Harner and Shoeib (2002)
BDE 30	9.02	Wania et al. (2002)
BDE 32	9.28	Wania et al. (2002)
BDE 35	9.48	Wania et al. (2002)
BDE 37	9.68	Wania et al. (2002)
BDE 47	10.53 10.34	Harner and Shoeib (2002) Wania et al. (2002)
BDE 66	10.82 10.49	Harner and Shoeib (2002) Wania et al. (2002)
BDE 69	10.23	Wania et al. (2002)
BDE 75	10.13	Wania et al. (2002)
BDE 77	10.87 10.70	Harner and Shoeib (2002) Wania et al. (2002)
BDE 82	11.14	Wania et al. (2002)
BDE 84	11.52	Wania et al. (2002)
BDE 85	11.66	Harner and Shoeib (2002)
BDE 99	11.31 11.28	Harner and Shoeib (2002) Wania et al. (2002)
BDE 100	11.13	Harner and Shoeib (2002)
BDE 126	11.97	Harner and Shoeib (2002)
BDE 153	11.82 12.15	Harner and Shoeib (2002) Wania et al. (2002)
BDE 154	11.92	Harner and Shoeib (2002)
BDE 156	11.97	Harner and Shoeib (2002)
BDE 183	11.96	Harner and Shoeib (2002)

Table 3-6. Calculated theoretical vapor-particle partitioning of PBDE congeners in ambient air at 25°C (calculated using equation 3-2).

Brominated Diphenyl Ether	Vapor phase, %	Particle phase, %
BDE 1	100	0
BDE 2	100	0
BDE 3	100	0
BDE 7	100	0
BDE 8	100	0
BDE 10	100	0
BDE 12	100	0
BDE 15	100	0
BDE 28	99	1
BDE 30	99	1
BDE 32	99	1
BDE 33	98	2
BDE 35	98	2
BDE 37	98	2
BDE 47	90	10
BDE 66	87	13
BDE 69	94	6
BDE 75	95	5
BDE 77	81	19
BDE 82	71	29
BDE 85	28	72
BDE 99	61	39
BDE 100	56	44
BDE 115	54	46
BDE 138	6	94
BDE 153	7	93
BDE 154	13	87
BDE 183	2	98
BDE 190	1	99

Table 3-7. Estimated BAF and BMF values for various aquatic species.

Brominated Diphenyl Ether	Species	Factor	Reference
BIOACCUMULATION FACTORS (BAF)			
BDE 28	Lake Trout	7.6	Tomy et al. (2004)
BDE 47	Lake Trout	7.3	Streets et al. (2006)
	Blue Mussels	6.1	Gustafsson et al (1999)
BDE 66	Lake Trout	7.3	Streets et al. (2006)
BDE 85	Lake Trout	2.3	Tomy et al. (2004)
BDE 99	Lake Trout	6.7	Streets et al. (2006)
	Blue Mussels	6.1	Gustafsson et al (1999)
BDE 10	Lake Trout	7.5	Streets et al. (2006)
BIOMAGNIFICATION FACTORS (BMF)			
BDE 15	Porpoise	1.6–2.4	Ramu et al. (2006)
BDE 28	Lake Trout	7.6	Tomy et al. (2004)
	Porpoise	1.6–2.4	Ramu et al. (2006)
BDE 47	Lake Trout	2.1	Tomy et al. (2004)
	Coho Salmon	3.2	Stapleton and Baker. (2003)
	Porpoise	1.5	Ramu et al. (2006)
BDE 66	Lake Trout	7.8	Tomy et al. (2004)
BDE 99	Lake Trout	6.6	Tomy et al. (2004)
	Porpoise	1.8–2.4	Ramu et al. (2006)
BDE 100	Lake Trout	6.5	Tomy et al. (2004)
	Porpoise	1.7–2.4	Ramu et al. (2006)
BDE 138	Lake Trout	3.2–8.7	Tomy et al. (2004)
BDE 153	Lake Trout	9.4	Tomy et al. (2004)
	Coho Salmon	4.0	Stapleton et al. (2003)
	Porpoise	1.6–2.2	Ramu et al. (2006)
BDE 154	Lake Trout	13.3	Tomy et al. (2004)
	Porpoise	1.4–2.2	Ramu et al. (2006)
BDE 183	Lake Trout	3.9	Tomy et al. (2004)
	Porpoise	0.8–2.2	Ramu et al. (2006)
BDE 190	Lake Trout	1.6–5.1	Tomy et al. (2004)
BDE 209	Lake Trout	0.3	Tomy et al. (2004)

Table 3-8. The BDE congener distributions in influent, sludges and final effluent from various surveys of sewage treatment plants in various countries.

PBDE Congener (Br substitution)	STP Influent (ng/L)	STP Sludge (µg/kg dwt)	STP Final Effluent (ng/L)	Location	Reference
BDE 1	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 2	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 3	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 7	NA	ND	0.016	Palo Alto, CA	North (2004)
BDE 8	NA	ND	0.0042	Palo Alto, CA	North (2004)
BDE 10	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 12	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 13	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 15	NA	0.62	0.008	Palo Alto, CA	North (2004)
BDE 17	NA	5.7	0.19	Palo Alto, CA	North (2004)
BDE 25	NA	0.62	0.0099	Palo Alto, CA	North (2004)
BDE 28	NA	13	0.266	Palo Alto, CA	North (2004)
	1.3	22	ND	Ontario, CAN	Song et al. (2006)
BDE 30	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 32	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 35	NA	ND	0.005	Palo Alto, CA	North (2004)
BDE 37	NA	0.24	0.0038	Palo Alto, CA	North (2004)
BDE 47	NA	757	10.5	Palo Alto, CA	North (2004)
	102	1,819	14	Ontario, CAN	Song et al (2006
	NA	2.77	NA	Germany (11 STPs)	Knoth et al (2007)
	NA	7.0*	NA	Sweden (22 STPs)	Oberg et al.(2002)
BDE 49	NA	18	0.266	Palo Alto, CA	North (2004)
BDE 66	NA	21	0.217	Palo Alto, CA	North (2004)
BDE 71	NA	2.8	0.043	Palo Alto, CA	North (2004)
BDE 75	NA	1.0	0.018	Palo Alto, CA	North (2004)
BDE 77	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 85	NA	34	0.352	Palo Alto, CA	North (2004)
	NA	0.42*	NA	Sweden (22 STPs)	Oberg et al. (2002)
BDE 99	NA	944	11.2	Palo Alto, CA	North (2004)
	121	2,004	16.0	Ontario, CAN	Song et al (2006
	NA	137.19	NA	Germany (11 STPs)	Knoth et al (2007)
	NA	10*	NA	Sweden (22 STPs)	Oberg et al. (2002)
BDE 106	NA	165	2.0	Palo Alto, CA	North (2004)
	19	289	2.8	Ontario, CAN	Song et al (2006)
	NA	154.45	NA	Germany (11 STPs)	Knoth et al (2007)
	NA	1.7*	NA	Sweden (22 STPs)	Oberg et al. (2002)
BDE 105	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 116	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 119	NA	ND	0.014	Palo Alto, CA	North (2004)
BDE 126	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 138	NA	7.7	0.096	Palo Alto, CA	North (2004)
	1.0	26.1	ND	Ontario, CAN	Song et al (2006)
	NA	ND	NA	Sweden (22 STPs)	Oberg et al. (2002)
BDE 140	NA	2.7	0.031	Palo Alto, CA	North (2004)
BDE 153	NA	88	0.98	Palo Alto, CA	North (2004)
	11	193	1.60	Ontario, CAN	Song et al. (2006)
	NA	27.31	NA	Germany (11 STPs)	Knoth et al. (2007)

PBDE Congener (Br substitution)	STP Influent (ng/L)	STP Sludge (µg/kg dwt)	STP Final Effluent (ng/L)	Location	Reference
	NA	0.86*	NA	Sweden (22 STPs)	Oberg et al. (2002)
BDE 154	NA	68	0.776	Palo Alto, CA	North (2004)
	7.6	120	ND	Ontario, CAN	Song et al. (2006)
	NA	18.51	NA	Germany (11 STPs)	Knoth et al. (2007)
	NA	0.72*	NA	Sweden (22 STPs)	Oberg et al.(2002)
BDE 155	NA	7.1	0.073	Palo Alto, CA	North (2004)
BDE 181	NA	ND	ND	Palo Alto, CA	North (2004)
BDE 183	NA	10	0.080	Palo Alto, CA	North (2004)
	1.7	34	ND	Ontario, CAN	Song et al. (2006)
	NA	13.66	NA	Germany (11 STPs)	Knoth et al. (2007)
BDE 190	NA	1.0	0.0039	Palo Alto, CA	North (2004)
BDE 206	NA	16	0.041	Palo Alto, CA	North (2004)
BDE 207	NA	22	0.095	Palo Alto, CA	North (2004)
BDE 208	NA	11	0.051	Palo Alto, CA	North (2004)
BDE 209	NA	1,183	1.73	Palo Alto, CA	North (2004)
	NA	363.46	NA	Germany (11 STPs)	Knoth et al. (2007)
	NA	11	NA	Sweden (22 STPs)	Knoth et al. (2007)
Total PBDEs	NA	3,381	29	Palo Alto, CA	North (2004)
	265	4,324	36	Ontario, CAN	Song et al. (2006)
	NA	987.21	NA	Germany (11 STPs)	Knoth et al. (2007)

NA = Not analyzed for; ND = not detected; *median concentration as opposed to mean.

Figure 3-1. Time-trends of deposition flux of PBDEs (blue solid diamonds) and PCBs (red open diamonds) to the sediments in each of the Great Lakes. (left x axis shows PBDE levels and right x axis shows PCB levels)
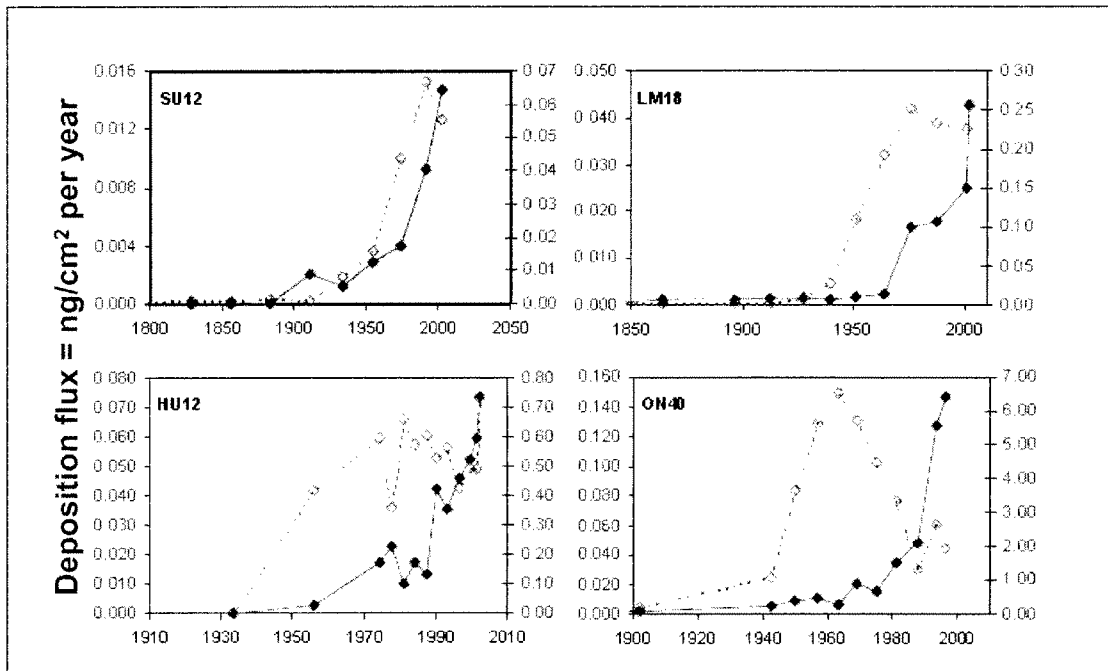
Chapter 4 ENVIRONMENTAL AND EXPOSURE MEDIA CONCENTRATIONS

4.1. INTRODUCTION

This chapter summarizes the concentrations of individual congeners and total PBDEs in environmental and exposure media. The emphasis is on data from the United States, although key data sets from other countries will be described as a contrast and a supplement. In addition, emphasis will be placed on the measurements for BDE 209, as this deca congener is essentially the sole component of the deca formulation that is still currently being produced and marketed in the United States. Much of the literature has focused on the profile of congeners ("profile" is the term used in this chapter to describe the suite of congeners) associated with the penta formulation (BDEs 47, 99, 100, 153, and 154), and, subsequently, there is a paucity of data for the primary markers of the octa formulation, BDE 183 (about 40% of the octa formulation), and the deca formulation, BDE 209 (about 98%). When possible, information on the possible debromination of BDE 209 will be provided. The sections on air, indoor dust, fish, and food include a comprehensive table of reported measurements of individual congeners in the United States; sections on water, sediment, and surface soil do not include such tables because there is little or no United States data on these media in the literature. The chapter concludes with development of a tabular assignment of BDE congener concentrations for each exposure media. These assignments will be used in the next chapter, which determines exposure dose of BDEs based on exposure media concentrations and contact rates.

4.2. WATER AND SEDIMENT

Water has been rarely sampled for PBDEs in America, and it is questionable whether available monitoring can be considered representative of drinking water. The San Francisco (SF) Estuary Regional Monitoring Program for Trace Substances sampled water, surface sediments, and bivalves (oysters, mussels, clams) in the SF Estuary for 22 BDE congeners, including BDE 209 (Oros et al., 2005). A total of 33 water samples were taken, with total PBDEs ranging from 3–513 pg/L, and a mean concentration of 146

pg/L. The region of the bay which had the highest concentrations, the Lower South Bay, receives 26% of the Estuary's total publicly owned treatment works (POTW) wastewater effluents and only 10% of the Estuary's freshwater inflow. The most abundant congeners were 47, 99, and 209. When 209 was not reported as "Q" (outside QA limits, detected but not reported), it was positive and of a comparable magnitude as 47, though mostly at a slightly higher concentration. BDE 47 concentrations ranged from about 17 to greater than 60 pg/L, with one high sample at 123 pg/L; BDE 99 was next highest with quantified concentrations (about 1/3 of the samples were outside quality limits) ranging from about 11 to 35 pg/L, with one high level at 90 pg/L . BDEs 17, 100, and 28/33 (congeners notated as "28/33" translate to congeners which have co-eluted and are analyzed as though they were one congener) were quantified at levels less than 5 pg/L, and others not below detection or very infrequently detected at levels less than 5 pg/L. It was found that the PBDEs were predominantly associated with the sediment fraction of the water column.

A second study, by Johnson et al. (2006), sampled for PBDEs in water and fish of Washington state rivers and lakes. Results from 15 samples taken in 7 rivers and 3 lakes in 2005 and 2006 were available. BDEs measured included 47, 49, 66, 71, 99, 100, 138, 153, 154, 183, 184, 190, and 209, although BDE 209 was never detected. Total concentrations ranged from 1 to 926 pg/L, although only two samples were above 100 pg/L, with one of them at 146 pg/L. The average was 91 pg/L, but there was a much lower median of 16 pg/L. The most abundant congeners found, and at the highest concentrations, were 47, 99, and 100.

Finally, Streets et al. (2006) measured PBDEs in the dissolved and particle-bound phases in Lake Michigan and found BDE congeners 47, 99, 100, and 66 were in the dissolved phase in concentrations ranging from 0.13 to 10 pg/L (for individual congeners). The quantified particle-phase concentrations for BDEs 47, 99, and 100 ranged from 0.18 to 1.4 pg/L. BDE 209 was not evaluated in this study.

In summary, only three studies measuring surface water concentrations in the United States could be found. In one study, total concentrations in the SF estuary ranged from 3 to 500+ pg/L, dominated by BDE congeners 47, 99, and 209. In the second study, BDE 209 could not be quantified, and total concentrations of the lower brominated

congeners ranged widely from 1 to 916 pg/L, although only 2 samples were above 100 pg/L, and one of them was at 146 pg/L. The mean and median in that study were 91 and 16 pg/L, respectively. The third study did not measure BDE 209, and, in that study, the authors separated out the dissolved and particle phase concentrations. In total, only the sum of BDEs 47, 66, 99, and 100 ranged up to 10 pg/L.

Much of the data on PBDEs in sediments of water bodies were taken in the context of sediment core studies, whose purpose is to elaborate on temporal trends of PBDEs in the environment (see Section 3.8.1). This section provides an overview of studies just focusing on surface sediments and on the surficial sediment concentrations in the sediment core studies.

Briefly, the sediment core studies showed BDE 209 to dominate surficial sediment profiles. The average concentrations of BDE congeners, not including BDE 209, in these studies were generally less than 10 ng/g, while BDE 209 concentrations ranged anywhere from 10 ng/g to 300+ ng/g. The maximum total BDE concentrations found exceeded 1,000 ng/g in some cases and they were dominated by BDE 209, which comprised over 90% of the total mass measured.

The study of the SF Estuary included both water and sediment sampling (Oros et al., 2005). A total of 48 sediment samples were taken and positive detections were only noted for BDEs 47, 99, 183, 204, and 205. It is noteworthy that BDE 209 was not found with a detection limit of 1.5 ng/g. This contrasts all other sediment studies where BDE 209 dominated the profile. It is also the only study found which provided concentrations of BDEs 204 and 205. Total concentrations ranged from ND (not detected) to 212 ng/g dry weight (dwt) basis. BDE 47 was detected 42% of the time, with a range of detections of 1.1 to 100 ng/g dwt, with an average of 12 ng/g dwt; BDE 99 was detected 77% of the time with a range of 0.3 to 71 and a mean of 5 ng/g dwt; BDEs 183, 204, and 205 were detected once, twice, and once respectively.

Raff and Hites (2004) collected suspended sediment samples from 26 sites along the Mississippi River and five of its major tributaries during July/August 2002 and March 2003. A total of 15 congeners were measured; individual congeners were not identified nor were individual congener concentrations provided. Total concentrations ranged from 31–1,548 ng/g dwt, with an average of 327 ng/g dwt. Consistent with the Great Lakes

sediment cores described in Chapter 3, BDE 209 was the overwhelmingly dominant congener. On average, it comprised 96.8% of the total concentration. Congeners 47 and 99 were the only other congeners accouting for concentrations in excess of 1% of the total PBDE concentrations—1.16 and 1.26%, respectively. Based on concentrations near the mouth of the river, combined with records on suspended sediment concentrations and outflows, the authors estimated that 8 tons/yr of PBDEs are discharged into the Gulf of Mexico. Other interesting trends include higher concentrations in the spring, attributed to high runoff, and evidence of debromination, as two sites contained higher concentrations of the nona congener BDE 206, as compared to BDE 209.

Ashley et al. (2006) analyzed four sediment samples, along with samples from eels taken from the Delaware River (eel data summarized below). The sediment samples were collected in 2002 as part of an earlier polychlorinated biphenyl (PCB) study, and they were reanalyzed in 2006 for PBDEs. While the total concentrations in these samples were lower than some of the studies above—between 0.7 and 21.7 ng/g dry weight—the proportions were similar. BDE 209 explained about 50% of the concentration, while BDEs 99 and 47 were next, accounting for 15 and 14%, respectively.

Four surficial sediment samples were taken in Lake Hadley in Indiana (Dodder et al., 2002). Total BDEs, including 47, 99, 100, 153, 154, and 209, ranged from 24 to 71 ng/g dwt, dominated by BDE 209 at 19 to 33 ng/g dwt. Other than a measurement of 22 ng/g dwt for BDE 99, all other measurements of the other congeners were near or less than 5 ng/g dwt.

Hale et al. (2002) studied the environmental impacts of a polyurethane foam manufacturing facility in the United States mid-Atlantic Region which had ceased production in 1997; sampling occurred in 2001. The interior of the facility was tested, as was soil adjacent to the facility, sediment in a stream leaving the facility, and sediment and bluefish in a pond about 250 meters from the facility. Total concentrations of PBDEs (including BDEs 47, 99, and 100; 153 and 154 were measured but not detected) in surface sediments in the stream leaving the facility were 17.2 ng/g dwt and 132 ng/g dwt, with one sample having all non-detects. In the pond, total concentrations in surface sediments were 0.5 and ND ng/g in two pond sediment samples These concentrations are

similar to measurements described at other settings, suggesting that the foam production facility did not result in noteworthy impacts to nearby aquatic settings.

Hale et al (2001) also measured PBDE (along with PCB) concentrations in fish and sediment samples from two large Virginia watersheds. The study sites included the larges bodies of freshwater in Virginia; they were not selected based on concerns from a particular source of contamination, although the general predominance of furniture manufacturing facilities was noted. Total PBDE concentrations, including BDEs 47, 99, 100, 153, and 154, ranged from non-detect to 52.3 ng/g dwt in 17 samples. BDE 49 was also measured, but not detected in sediment samples. The concentrations were dominated by BDE 47, explaining about 53% of total concentrations, with BDE 99 second at about 35% of total, and the remaining concentration due to BDE 100 (7%), 153 (<5%), and 154 (<5%).

Toms et al. (2006) undertook a comprehensive assessment of the fate and distribution of 26 BDE congeners in the aquatic environment of Australia in 2003-2004. An aim of this study was to determine the background concentrations and congener compositions of BDEs in estuarine, freshwater, and marine sediments. Ninety sediment samples were analyzed from locations representing various land uses ranging from remote to industrial. A total of 25 BDE congeners were detected in samples from 35 of 46 sites (76%), and total PBDE concentrations ranged from non-detect to 60.9 ng/g dw with an overall mean ($\pm$ SD) and median of 4.7 $\pm$ 12.6 and 0.3 ng/g dw, respectively. BDE-209 dominated the congener distribution in 86% of the sediment samples.

Christensen and Platz (2001) sampled sediment from Danish marine coastal areas, freshwater lakes, and a river in 2000. The congeners measured included BDEs 47, 99, 100, 153, and 209, and the total concentrations ranged from 0.06–24.7 and 0.07–10.6 ng/g dw in marine and freshwater sediment, respectively. BDE-209 dominated the congener profile in marine and freshwater sediments with a median concentration of 3.35 ng/g dw and 2.05 ng/g dw, respectively. The rank order of BDE congeners by concentration in sediment was 209 > 99 > 47 > 100 > 153. The highest concentrations of BDEs were detected in sediments in harbors and lakes located in urban areas. Because PBDEs were never produced in Denmark, the authors postulated that PBDEs entered the

Danish environment primarily by long-range transport. The authors suggested that additional local sources could be evaporation and leaching from PBDE-treated products.

In a study by Eljarrat et al. (2005), 13 marine sediment samples were collected from three coastal areas of Spain in 2002. The sediments were analyzed for 40 BDE congeners, but only 12 were detected in the sediments: BDEs 28, 33, 47, 66, 77, 100, 99, 118, 154, 153, 183, and 209. Total PBDE levels ranged from 2.7 to 134 ng/g dwt in coastal marine sediments, with highest levels in sediments off the coast of Barcelona. All the sediment samples were dominated by BDE-209, which constituted between 50 and 99% of the total PBDE contamination. The usual congener profile found in the coastal marine sediments was 209 > 47 > 99 > 100 > 153.

Rayne et al. (2003) measured surficial sediment in the Columbia River System in southeastern British Columbia and found total BDEs (for mono- through hexabrominated BDEs) ranged from 3.8 to 90.9 ng/g oc (organic carbon). Given that organic carbon is around 1% total dwt in water body sediments, this suggests concentrations less than 1 ng/g dwt. They did not measure for BDE 209.

Water body sediment studies in North America included one in the San Francisco Estuary, one in Virginia watersheds, one in a pond near a closed polyurethane foam manufacturing factiliy, one in British Columbia, and several core studies describing trends in the Great Lakes (the trend studies are reviewed in Chapter 3). Overall, total concentrations were mostly less than 20 ng/g dwt but ranged as high as 300 ng/g dwt in Lakes Michigan and Erie. With one exception, the study in the SF Bay Estuary (Oros et al., 2005), over 90% of the total concentration was BDE 209 when it was measured. In the SF Bay, BDE 209 was not detected in sediments at all (detection limit sufficiently low at 1.5 ng/g dw), and BDE 47 dominated the profile.

4.3. SURFACE SOIL

Only one systematic study could be found that looked at PBDEs in surface soils in the United States (Offenberg et al., 2006) in predominantly suburban, background, settings. A total of 33 surface soil samples were taken in 15 states and measured for 30 BDE congeners. Concentrations of total BDEs averaged 103 ng/g and had a geometric mean concentration of 5.3 ng/g dry weight dwt, and a range of 0.09 to 1200 ng/g dwt.

BDE 47 was detected in 31 of 33 samples averaging 1.9 ng/g dwt over the entire data set (ND = 0). BDE 99 was observed in 30 samples and averaged 3.6 ng/g dwt. BDE 209 was found in 24 samples and averaged 15.3 ng/g dwt. The highest concentrations were found for BDE 183, but it was only found in 3 samples at concentrations ranging from 121 to 562 ng/g dwt, so that the survey-wide average was 37.4 ng/g dwt.

Hale et al (2002) measured surface soil near a polyurethane foam production facility which had been closed a few years earlier than sampling. They quantified concentrations of BDEs 47, 99, and 100 (BDEs 153 and 154 measured for but not detected) in 3 samples with total concentrations of ND (no congeners detected), 13.6, and 76.0 ng/g dwt. In these two samples, BDEs 47 and 99 dominated explaining near half the total concentrations each.

One European study looked at background soils, and one study was found evaluating soil concentrations near an electronics recycling facility in China. Hassanin et al. (2004) reported on sampling of 66 surface samples (0–5 cm) and for 38 of these, a paired subsurface sample (5–10 cm), taken from grassland and woodland areas in the United Kingdom and Norway. These samples were collected in 1998, and 20 BDEs were measured for, although BDE 209 was not measured. The congeners most routinely detected (85–100% of the time) in the samples were 47, 99, 100, 153, 154, and 183. Major trends include the following: United Kingdom concentrations were higher than Norway concentrations, woodland concentrations were higher than grassland concentrations, and subsurface concentrations were significantly lower than surface soil concentrations. The total concentration of all BDEs ranged from 0.065 to 12.0 ng/g dwt. The results for grassland/United Kingdom, woodland/United Kingdom, and woodland/Norway were presented in terms of medians, min/max, and percent detected. The selected range of median concentrations for these three groupings of surface soils, were (pg/g dwt, with percent detected in parenthesis): BDE 17 (52-75%) – 27, 28, 35; BDE 28 (14-76%) – 17, 21, 29; BDE 47 (100%) – 61, 490, 250; BDE 99 (92-95%) – 280, 900, 360; BDE 100 (90-95%) – 36, 110, 58; BDE 153 (54-95%) – 72, 210, 51; BDE 154 (90-100%) – 22, 100, 42; BDE 183 (54-100%) – 26, 70, 25. The authors state that the percentage found in soil mirrored the technical penta product, Bromkal 70-5DE, with BDE 47 averaging 21% of total PBDE, 99 averaging 40%, 100 averaging 6%, 153

averaging 8.7%, and 154 averaging 4.4%. A major shortcoming of this surface soil study, however, was that BDE 209 was not measured. Given high measurements of BDE 209 in sediments, its lack of measurement in this broad ranging surface soil sampling study is unfortunate.

Cai and Zang (2005) characterized soil concentrations near an electronics recycling facility in China. For the six soil samples, the congeners measured and their average concentrations were as follows: BDE 3 presented as ND (3 is a mono-BDE); BDE 15 presented as 0.76 ng/g dwt (15 is a di-BDE); BDE 28—5.01 (tri-BDE); BDE 47—217; BDE 99—552; BDE 139—35; BDE 153—74; BDE 154—49; and BDE 183— 12.3. The total concentration is 945 ng/g dwt. The authors state that the isomer pattern of 47, 99, 139, 153, and 154 is consistent with the penta-BDE formulation. Again, no measurements of BDE 209 were made in this study.

In summary, the sparse literature suggests background soil concentrations of total BDEs might average above 100 ng/g dwt, although a central tendency estimate (geometric mean or median) might be lower at near 5 ng/g dwt. Total concentrations in soils at industrial sites, which might suggest a more direct source, could range as high as 1,000 ng/g dwt. The congener pattern noted in the studies on background soils is similar to the penta-BDE formulation. However, BDE 209 has mostly not been sampled frequently in soil studies; in the one study where it was measured, it was found in 24 of 33 samples, averaging 15.3 ng/g dwt.

4.4. INDOOR DUST

House dust was the focus of several United States studies because of the concern for indoor exposures in residences. Data was also available in one study on concentrations in a computer laboratory. As will be seen, indoor dust from both homes and places of work are dominated by BDEs 47, 99, and 209. Table 4.1 provides congener-specific concentrations for indoor dust from the literature.

House dust and dryer lint samples were collected from 16 homes in the Washington, DC area and 1 from Charleston, SC. The samples were analyzed for 22 individual PBDE congeners (Stapleton, et al., 2005). Total concentration ranged from 780 ng/g dwt to 30,000 ng/g dwt, with a mean total of 5,900 ng/g dwt. The dominant

congeners were the ones associated with the penta and decaBDE commercial mixtures, BDEs 47, 99, and 209. The mean concentration of these three congeners was 1,220; 1,700; and 2,090 ng/g dwt, respectively. No correlations were found in the study with year of construction, type of flooring (hardwood vs. carpet), or the number of televisons and PCs (and hours of computer use per week). However, an inverse relationship was found with the area of the home and the contribution of BDE 209 to the total PBDE concentration in dust. Clothes dryer lint, examined in five of the homes, showed concentrations ranging from 480 to 3,080 ng/g dwt. The two house dust samples with the highest concentration had the commercial pentaBDE commercial ration of 0.6 for BDE 47/BDE 99.

Ten women from a prior Environmental Working Group (EWG) study on breast milk (of a total of 20 women in the earlier study) collected samples of dust from their home (Sharp and Lunder, 2004). The 10 samples were taken in CA (2 samples), TX, CO, DC, MI, WA, OR, FL, and MT. A total of 13 congeners were sampled; detection levels ranging from 50 ng/g dwt for the lower brominated congeners to 90 ng/g dwt for the mid-brominated congeners and finally 400 ng/g dwt for BDE 209. One of the 10 individuals had very high measurements, at a total of 41,203 ng/g dwt for all congeners; the next highest was 16,366 ng/g dwt. This individual had used her vacuum to clean up polyurethane foam residues when she removed carpet padding, two mattress pads, and an uncovered foam cushion from her home. This study did not find the archtypal penta BDE commercial blend of 0.6 for BDE 47/BDE 99 in all cases. In half of the samples, a significantly higher amount of BDE 47 was found. However, the overall averages of the three highest congeners, BDEs 47, 99, and 209, were similar to the findings for homes in the Washington, DC area: BDE 47 was found at an average of 1,847 ng/g dwt, BDE 99 at 2,352 ng/g dwt, and BDE 209 at 2,394 ng/g dwt. Concentrations in the dust were not correlated to the number of electronic appliances or computers, foam furniture, or recent remodeling.

BDE concentrations in dust were determined from 10 vacuum bag samples from Atlanta, GA and from 10 vacuum bag samples from Germany, Australia, and the United Kingdom (Sjodin, et al., 2004) for a total of 40 samples. Only the United States and German samples, and only BDEs 47, 99, 100, 153, 154, 183, and 209 were available in

Sjodin et al. (2004). The United States data were substantially higher than the German data: the range of total PBDEs in Germany ranged from 17–550 ng/g dwt (median = 74) while the United States data ranged from 530–29,000 ng/g dwt (4,200 median). The authors claimed that the pentaBDE pattern found in these samples was similar to that in the products; specifically, that BDE 99 is similar in concentration to that of BDE 47. This contention is different than made by others, who note that the ratio of BDE 47 to BDE 99 in commercial penta formulations is about 0.60. Median concentrations (mean concentrations were not available) of the top three congeners, 47, 99, and 209, were 430, 880, and 2,000 ng/g dwt. Wenning et al. (2006) collected vacuum bag dust and air conditioner filter dust from homes in Northern California and Wellington, New Zealand, but the average congener-specific concentrations they presented were for all samples, not distinguishing by geography or whether it was vacuum or air conditioner generated dust. The average total BDE concentration over 13 samples, including 10 vacuum and 3 air conditioner samples, was 13,570 ng/g dwt, with BDE 209 comprising 9,052 ng/g dwt, and BDEs 99 and 47 comprising 2,140 and 1,120 ng/g dwt respectively. The BDE 209 results were skewed by a business air conditioner filter showing 84,500 ng/g dwt, from a total of 96,300 ng/g dwt, so the general representativeness of these samples must be questioned.

Indoor air and dust were sampled in 120 homes in Cape Cod, MA, in two rounds of 60 samples each starting in 99 and ending in 2001 (Rudel et al., 2003). A total of 89 homes were sampled for PBDEs in dust. Only 3 PBDEs (47, 99, and 100) were analyzed, and the reporting limits were high—400 ng/g dwt for BDEs 47 and 99 and 300 ng/g dwt for BDE 100; the data were not tremendously informative. BDE 47 was found in about 50%of the samples with a maximum of 9,860 ng/g dwt; BDE 99 was found also about 50% of the time with a maximum of 22,500 ng/g dwt; and BDE 100 was found 20% of the time at a maximum of 3,400 ng/g dwt.

Four computer wipe and 9 vacuum bag bulk dust samples were taken in Dallas Texas in 2004 (Schecter et al., 2005; date of sampling not given but presumed to be in 2004 as paper was submitted in July, 2004). The computer wipe samples were in units of ng/100 cm^2, and because no other data are available in these units to compare with, results from this study are not provided in Table 4.1. Briefly, concentrations ranged from

77 to 1,536 ng/100 cm^2, with BDE 209 explaining over 90% in the two samples of PC monitor screens, and about 53% in both PC casing samples. In these PC casing samples, BDEs 99 and 47 were next in predominance, accounting for 25 and 11%, respectively. In 9 bulk dust samples, the total ranged from 705 to 65,777 ng/g dwt, with a median of 2,507 and a mean of 12,136 ng/g dwt. BDE 209 was the prominent congener in 7 of 9 samples (BDE 99 was the dominant in the other 2 samples), explaining 95% of the concentration in the highest sample (65,777 ng/g dwt of 69,283 ng/g dwt total) and 66-87% in the other samples. In one sample, however, BDE 209 explained less than 1% of the 30,368 ng/g dwt total, with BDE 99 explaining 13,841 (46%) and BDE 47 explaining 10,538 ng/g dwt (35%).

Allen et al. (2008) reports on BDEs in house dust samples that were part of a study looking at the correlations between indoor air, dust wipe samples, and bulk dust samples in 20 homes in the urban setting of Boston, MA. They took three bulk samples out of each home and developed geometric mean congener-specific concentrations for the three common locations: the main living room, the bedroom, and a sample from a home vacuum (location undetermined). Like other studies, BDE 209 dominated all locations, with the geometric means over these three locations being 4702, 1866, and 1811 ng/g dwt, respectively. BDE 99 was second highest at 2460, 1170, and 536, respectively, and BDE 47 was the third most prevalent at 1865, 837, and 338, respectively. The geometric mean of the total concentrations (including 17, 28/33, 47, 49, 66, 75, 85/155, 99, 100, 138, 153, 154, 183, 196, 197, 203, 206, 207, 208, and 209) of the three locations were 13732, 6255, and 4,269 ng/g dwt, respectively. They found a high correlation between dust wipes and bulk dust samples. They did find a correlation between air and dust concentrations of the penta formulation congeners (BDEs 17 up to BDE 154), but did not find a correlation between BDE 209 air and dust concentrations. They found the highest concentration of BDE 209 – 527,000 ng/g dwt – and the highest concentration of total PBDEs – 544,000 ng/g dwt – that has been reported in the literature.

In another study of the Boston, MA, area, Wu et al. (2007) sampled 11 houses and measured for 17, 28, 47, 66, 85, 99, 100, 138, 153, 154, 183, and 209. This was a study in which womens' breast milk in these homes were also sampled (results reported in Chapter 5). Results were log normal with a geometric mean of total BDE concentration

of 1,910 ng/g dwt. BDE 99 and 47 were quantified nearly all the time, with median concentrations of 1010 and 670 ng/g dwt, respectively. BDE 209 was quantified in 5 of 11 samples, with quantified concentrations ranging between 1360 and 9020 ng/g dwt. The median total concentration was 1910 ng/g dwt. There was a "high correlation" between dust levels and breast milk levels, as well as with reported dietary habits, particularly the consumption of dairy products.

Harrad et al (2008) sampled between 10 and 28 homes in each of these locations: Amarillo/Austin in Texas; Birmingham, UK; Toronto, Canada; and Wellington, New Zealand. They measured for BDEs 28, 47, 99, 100, 153, 154, 183, and 209. The most interesting finding was that the profile of BDE congeners found in the different locations. The UK site had the highest BDE 209 concentrations, with an average of 45,000, a median of 2,800, and a geometric mean of 3,800 ng/g dwt (n=16), compared to the results from Texas which had an average of 1,600, a median of 1,300, and a geometric mean of 1,300 ng/g dwt (n=17). The two highest concentrations of BDE 209 in the UK, at 520,000 and 100,000 ng/g dwt, are the highest for this congener ever reported in dust (Harrad et al 2008). The concentrations of the tri-hexa congeners (BDEs 28 thru 154) were the highest for the Texas dust. For example, the geometric mean for the sum of these six congeners from the Texas sites was 1,800 compared to 52 from the UK sites. Due to the high BDE 209 findings, the overall total concentations were highest for the UK. The geometric mean total concentrations for the UK, US, and Canadian sites were: 4,500; 3,600; and 1,200 ng/g dwt. Totals were not provided for New Zealand because the BDE 183 and 209 congeners were not measured. Concentrations of the tri-hexa congeners totaled 92 ng/g dwt, which was comparable to the tri-hexa totals for the UK.

The California Air Resources Board (CARB) has conducted a substantial amount of sampling for brominated flame retardants in air and dust, both outside near industrial sites and in urban and background locations, and inside at workplaces and industrial sites (see http://www.arb.ca.gov/toxics/pbde.htm). Two samples of carpet dust in an indoor computer training facility totaled 10,200 and 4,800 ng/g dwt (CARB, 2005). Congeners in the dust that were not in the air that was sampled in this facility included BDEs 203, 183, 155, 138, and 77 (203 and 183 are congeners in the octa formulation). The

dominant congener was BDE 209, at 7,560 and 2,800 ng/g dwt, followed by BDE 99 at 856 and 695 ng/g dwt and BDE 47 at 502 and 411 ng/g dwt.

Sampling of indoor dust has also occurred in foreign countries, including Europe and Kuwait. Fabrellas et al. (2005) report sampling done by "Euroconsumers Organization" and "CIEMAT-POPs Group," in which dust was collected from 100 vacuum cleaner bags from Spain (34 bags), Belgium (32 bags), Portugal (22 bags), and Italy (12 bags) and measured for BDEs. Homologue group concentrations from mono- to hepta-BDE were determined; Levels of detections ranged from 0.006–0.220 ng/g dwt (BDE 3) to 0.55–20.9 ng/g dwt (for BDE 209). Results suggest Italy has the highest total concentrations, at 581 ng/g dwt, followed by Portugal (354 ng/g dwt), Spain (238 ng/g dwt), and Belgium (190 ng/g dwt), however, it is not known if these mean values are significantly different The highest value from Spain was sucked out from the inside of a computer. Urban samples were found to be higher than rural, and BDE 209 was found to comprise greater than 60% of total PBDE in all cases. The ratio of BDE 47 to BDE 99, which is 0.5—0.7 in 24% of the samples, is the same as the commercial penta formulation, DE-71, which has ratio of 0.6, suggesting these samples were dominated by this commercial mixture. Harrad et al. (2006) measured BDEs in indoor dust at 8 homes sampled in 2005 in the UK. They reported an average of 215 ng/g total (maximum of 625 ng/g total), which included BDEs 28, 47, 49, 66, 85, 99, 100, 153, and 154 (209 not measured). The ratio of 47:99 was about 0.5. The study that Harrad et al. (2008) later published on different locations around the world (United Kingdom, United States, New Zealand, and Canada) included this earlier data. Karlsson et al. (2007) report on measurements of PBDEs in air, housedust, and blood from five households. The average total BDE (including 13 BDEs) was about 690 ng/g dwt, dominated by BDE 209 that was 490 ng/g dwt, followed by BDEs 99 and 47, at 99 and 51 ng/g dwt, respectively. Gevao et al. (2005) report on simultaneous air and dust from homes in Kuwait in 2005. PUF samplers were used to measure air samples and bulk dust samples were obtained from vacuum bags in 17 homes in Kuwait between Feb 29 and April 11, 2004. Individual congeners measured included BDEs 28, 47, 100, 99, 85, 154, 153, 183 (209 not measured), although individual concentrations were not provided. Total PBDE dust concentrations ranged from 0.2–24 ng/g dwt (geometric mean 9 ng/g dwt) and were log

normally distributed. BDEs 47 and 99 seemed to track well with what the authors claimed was a commercial penta mixture; however, the 47/99 ratio of this mixture was not 0.6, but rather close to 1.0. House dust correlated with indoor air —when one was high, the other was also.

Recent data from Europe show very high levels of BDE 209. Household dust and lint samples were collected in Scotland, Northern England, and Germany (Pless-Mulloli, et al., 2006) and compared with other published data from the United States, although the comparison did not include more recent data such as that from Allen et al (2008). The United Kingdom had the highest concentrations, though they were dominated by BDE 209, while Germany's concentrations were much lower. The United Kingdom dust samples had a mean of 11,325 ng/g, and a median of 3,933 ng/g and a maximum of 54,858 ng/g. The UK samples were dominated by BDE 209, which had an average concentration of 11,233 ng/g (of the 11,325 ng/g total). The median of United States dust measurements were similar to the median of the United Kingdom's measurements at 4,200 ng/g, but BDE 209 was not nearly as dominant, having a median of only 2,000 ng/g. As discussed earlier, the data in Allen et al (2008) from homes in the Boston, MA, area had higher BDE 209 concentrations than these measurements summarized in Pless-Mulloli et al. (2006).

In summary, the most common indoor congeners include those associated with the penta formulations, BDEs 47 and 99, and the single congener most associated with the deca formulation, BDE 209. Concentrations of total BDEs ranged from the low 100s to well over 10,000 ng/g dwt. Most authors reported that the results suggested the presence of the penta formulation, based on the ratio of the two congeners, BDEs 47 and 99. However, there was not uniform agreement on the ratio: some claimed that comparable amounts of the two congeners suggest the presence of the commercial product, while others claim that the penta formulation translates to a consistent 47/99 ratio of about 0.6. When quantified, BDE-209 dominated the profile above 47 and 99, with concentrations generally in the 2,000–10,000 ng/g dwt range, although with some outlier concentrations above 50,000 ng/g dwt. Dust concentrations of BDEs appear much higher in United States's dust, well into the range of 1,000s of ng/g dwt for total concentrations, compared with limited measurements in Europe, which appear to be

limited to the 100s of ng/g dwt (except for data from the United Kingdom showing substantial concentrations of BDE 209), and, in one study from Kuwait, with the highest measurement just above 100 ng/g dwt.

4.5. AIR CONCENTRATIONS

Efforts to monitor PBDEs in outdoor air have been more extensive than efforts to monitor indoor air in the United States, despite evidence that shows indoor air to have generally higher concentrations of BDEs than outdoor air. Outdoor measurements have been taken in California, in the Great Lakes Region, and in several other states including New York, Louisiana, Indiana, Maryland, and other states. Only one study focusing on indoor air in residential settings, in the Boston MA area, could be found. Sites in Europe and Asia suggest lower concentrations as compared to the United States, although one study from China reports much higher concentrations. One study reported on air concentrations within cars in Greece. Section 4.5.1 describes efforts to monitor outdoor air while Section 4.5.2 describes indoor-air studies. Table 4.2 provides congener-specific concentrations for both outdoor and indoor air in the United States.

4.5.1. Outdoor Air Concentrations

CARB established the California Ambient Dioxin Air Monitoring Network. This Network, comprised of 11 sites, began sampling in 2002, and the data for the years 2002-2004 are posted at http://www.arb.ca.gov/pub/dioxin/cadamp.php. PBDE data was taken from 7 of these 11 sites, 4 from the Bay Area and 3 from the South Coast, beginning in 2003. There were 6 monthly samples in 2003, and 12 monthly samples in 2004. Individual site data and site/statewide averages are available from the Web site. Twelve congeners were measured, including 17, 28, 47, 65, 88, 85, 99, 100, 153, 154, 183, and 209. BDEs 47 and 99 had similar 12-month averages in 2004, of 53 and 51 pg/m^3, respectively, with BDE 209 coming in third at 25 pg/m^3. BDE 100 averaged 13 pg/m^3, and others ranged from 0.04 to 4.0 pg/m^3. The total average total concentration for 2004 was 160 pg/m^3.

CARB (2005) also sponsored a research monitoring effort that included indoor and outdoor sampling at industrial sites, office sites, and outdoors at the University of

California at Davis (UC Davis). The UC Davis sampling was conducted to evaluate the effectiveness of two different active outdoor samplers: one included a filter (for capture of particle-bound PBDEs), followed by a PUF (for vapor-phase PBDEs), and the second sampler included a filter followed by an XAD-2 resin. Four sampling units, two of each, took two samples per day on two consecutive days, 3/17/2004 and 3/18/2004, providing a total of 8 samples. Statistical analysis suggested the two measured comparably, except that the filter/PUF setup could not measure BDE 209 due to analytical difficulties. A total of 33 congeners were measured, making it the most substantial air study found in the literature. Total BDE concentration outdoors in this test averaged 38 pg/m^3 for the filter/PUF setup and 93 pg/m^3 for the filter/XAD setup where BDE 209 made up 10 pg/m^3 of that total. BDEs 47 and 99 dominated the profiles, averaging 34.6 and 12.5 pg/m^3, respectively, between the two sampling units.

Hoh and Hites (2005) took air samples every 12 days at 5 locations from Lake Michigan to the Gulf of Mexico, between 8/2002 and 12/2003, covering most (but not all) months of the year. One site was urban, Chicago, two sites were in remote locations in Michigan and Louisiana, one site was in an agricultural region, and one was in the university city of Bloomington, Indiana. Although numerous congener data were evaluated, individual data was only available for 47, 99, 100, and 209 (these 4 contributed about 80% of the total PBDE). The highest congener individually was 47, averaging between 7 and 17 pg/m^3 over all sites, while the other congeners mostly averaged under 7 pg/m^3. The key exception was BDE 209, which was important in Chicago, averaging 60 pg/m^3 while it was 9 pg/m^3 or less in other sites. Overall, the total PBDE concentration at the Chicago site was the highest, averaging 100 pg/m^3; the concentrations at the other four sites were comparable, averaging under 30 pg/m^3.

Strandberg et al. (2001) monitored BDEs at four sampling sites include downtown Chicago (urban site), Sleeping Bear Dunes, MI (rural site), Sturgeon Point, NY (rural), and Eagle Harbor, MI (remote). Four samples per year (taken May through October) for each of the four sites and for three years (1997, 1998, and 1999) resulted in a total of 48 samples. BDEs included 47, 99, 100, 153, 154, 190, and 209. The average total BDE for Chicago was about 50 pg/m^3 over 3 years, while it was about 5–15 pg/m^3 for the other three sites. BDE 209 was not detected at the 3 rural/remote sites, and detected at levels

only at 0.3 pg/m³ in Chicago. This is in stark contrast to the monitoring of Hoh and Hites (2005), which showed an average of 60 pg/m³ in 2002/2003. This could likely be due to the fact that Strandberg et al. (2001) did their measurements between 1997 and 1999, prior to the time when the penta and octa formulations were taken off the United States market, and the market became dominated by the deca formulation (which contains 97% BDE 209). Also, Strandberg et al. (2001) note analytical difficulties with BDE 209 (degradation during analysis, low recovery) and discusses other possibilities for low findings in air (low mobility from source, etc); Hoh and Hites (2005) notes much higher BDE 209 in their data and attributes it to greater usage of decaBDE, not analytical problems. Gas/particle partitioning suggests 80% of BDE 47 was in the vapor phase, 55–65% in the vapor phase for BDEs 99 and 100, and only about 30% in vapor phase for BDEs 153 and 154.

This sampling network reported on by Strandberg et al. (2001) and Hoh and Hites (2005) continued to be monitored during 2005 and 2006, and PBDE results from that effort are reported on by Venier and Hites (2008). The total PBDE concentrations were highest at the "urban" sites of Chicago and Cleveland, with mean concentrations of 65 and 87 pg/m³, respectively, with the higher concentrations at Cleveland due to the presence of higher concentrations of BDE 209 in several samples. The mean concentrations at the "rural" sites of Sturgeon Point and Sleeping Bear Dunes were 9.2 and 8.1 pg/m³, respectively, and the mean concentration at the "remote" site of Eagle Harbor was 5.8 pg/m³. The authors also studied the time trends of BDE 47, 99, and 209 from data starting in 2003, and observed that BDEs 47 and 99 were declining rapidly, but that BDE 209 was not declining at any of the 5 sites.

Three sites within the Cheasapeake Bay area were sampled for BDEs as part of a larger effort at air quality monitoring in the region (Goel et al., 2006). A total of 240 samples were taken between 2001 and 2003, using high volume samplers to measure both particle and gas phases for BDE 47, 99, 100, and 154. BDEs were detected in 75% of the samples, but detection frequency was highest at Lewes, 98%, compared to Dover, 77%, and Horn Point, 52%. Gaseous phase concentrations were presented and were highest at Lewes, with geometric mean concentrations over the 3-year period of 174 pg/m³, followed by Dover and Horn Point at 19 and 10 pg/m³, respectively. It is not clear why

particle phase concentrations were not presented in Goel et al. (2006), except that they were much less frequently found (<5% of samples from Horn Point and Dover), but at 40–50% of samples at Lewes. BDE 47 was the highest found in all three sites, from about twice as high as 99 and 100, in Horn Point and Dover (10-20 pg/m^3 for BDE 47 compared to 5-8 pg/m^3 for BDEs 99 and 100 as geometric means), to about 7-10 times as high at Lewes (175 pg/m^3 for BDE 47 compared to 17-26 pg/m^3 for BDEs 99 and 100). BDE 154 was essentially not detected in either gaseous or particle phases. The finding at Lewes was attributed to use of spray irrigation of municipal wastewater near the sampling site.

One of the CARB sites involved outdoor sampling near a possible source of PBDE release—near an autoshredder. On this site, there were 4 samplers, including 2 upwind and 2 downwind, sampling for 3 days, resulting in 12 samples. There were no field activities during the first day of sampling, and the results were lower that day when compared to days afterward—when the facility was operating. Another key observation was that samples were much higher downwind compared to upwind. However, even the upwind sites were higher than the UC Davis site discussed earlier, suggesting that there was offgasing from previously deposited BDEs from the nearby source. The highest measurement was for BDE 209, averaging over 2,400 pg/m^3 for 4 sampling dates. Only one other sample had at least one reading above 100 pg/m^3, and that was for BDE 99, which had the second highest readings overall, with an average of 165 pg/m^3 over the 9 days. BDE 47 had the next highest, averaging 64 pg/m^3.

A second CARB site was near another source: an electronics recycling plant. Three days of outdoor sampling with 4 samplers (2 in front and 2 in back) for a total of 12 samples, measured the impact of the release of PBDEs from electronics within this recycling facility. Samplers located in front, near the loading dock, which was open most of the time, had higher concentrations as compared to the back two samplers. BDE 209 was the highest congener measured, averaging 2,764 pg/m^3, with the next highest BDE 183 averaging 116, and then BDE 47 averaging 70 pg/m^3. The interesting thing about BDE 209, which exists predominantly in the particle form, is that the concentrations were 40-fold higher in the front samplers, near the open doors to the indoor recycling facility. This suggests that near a source (such as near the front doors in a recycling facility where

BDE 209 could be released), BDE 209 concentrations can be extremely elevated, but far from a source (in the back of the building), concentrations are much lower.

Another study evaluating outdoor air impacts near a source occurred in the United Kingdom. Air concentrations before and after a major bonfire were analyzed for both PAHs and PBDEs in November of 2000 (Farrar, et al., 2004). "Bonfire Festival" in the United Kingdom occurs every year on November 5. Three samplers were set up in the garden of a home in a residential area. They were not in the immediate vicinity of any public bonfires, but there were bonfires known to be occurring in the general area. Daily samples were taken from November 1 to November 13, and the evidence clearly showed a rise in concentration on the 5th, with background levels on the 4th and before and from the 6th onward. A total of 21 BDE congeners were measured, although BDE 209 was not measured. Background concentrations were only about 4 pg/m^3, with quantifiable measurements of BDEs 47 (2 pg/m^3), BDE 99 (1.5 pg/m^3), and BDE 100 (0.5 pg/m^3). This clean air was attributed to air originating over the Atlantic Ocean. Concentrations reached 95 pg/m^3 on November 5, with the highest concentrations of BDEs 99 (14 pg/m^3), BDE 153 (13 pg/m^3), BDE 154 (10 pg/m^3), BDE 166 (10 pg/m^3), BDE 47 (8 pg/m^3), BDE 49 (8 pg/m^3), and similar concentrations of 4 pg/m^3 for BDEs 66, 85, 100, 181, and 190. Likely sources of the PBDEs were the burning of discarded clothing or furniture, with temperatures not hot enough to destroy the PBDEs. No explanations were provided for the predominance of BDE 153 and 154, which is different from the more typical predominance of BDEs 47 and 99 (as seen in background air).

Wilford et al. (2008) sampled air at a semi-rural site in northwest England in April to May of 2004. Their analysis focused almost exclusively on characterizing the particulate phase of BDEs, with a particular emphasis on BDE 209 and other higher brominated congeners. They did measure for the vapor phase using a polyurethane foam (PUF) sampler for a 7-day period, and found very low levels of BDE 209 in a limited number of samples. More importantly, they stated that the vapor phase was dominated by tri- to hexa-BDE congeners, and that these congeners were found at vapor-phase concentrations at least two orders of magnitude higher than in the particle-phase. However, they did not provide any results for these vapor-phase concentrations, or for total (vapor+particle) phase concentrations. Their mean and median concentration of

total BDEs in the particle phase over 28 samples was 41 and 18 pg/m^3, respectively. The mean and median BDE 209 concentrations were 20 and 13 pg/m^3, respectively, showing the dominance of this congener in the particle-phase profile. In fact, the sum of hepta thru deca-congeners, or more specifically, the sum of BDEs 183, 196, 197, 206, 207, 208, and 209, explained over 90% of the total profile over all samples. Concentrations of the lower brominated congeners in the particle phase were mostly less than 1 pg/m^3. BDE 183 was found in 10 of the 28 samples, at a mean of 4.6 pg/m^3, at a high of 92 pg/m^3.

Jaward et al. (2004, 2005) have provided a comprehensive sampling of outdoor air in both Asia (Jaward et al., 2005) and Europe (Jaward et al., 2004). They used polyurethane foam (PUF) disk samplers in a passive mode, meaning they leave the PUF exposed to outdoor air to capture PBDEs for a period of 6 weeks, then analyze the PUFs. Two drawbacks from this approach are that it purports to capture only the vapor phase BDEs (it was stated that the PUF captures some particles, but the amount on particles was hard to ascertain), and, secondly, that BDE 209 was not analyzed in their studies. In their Asian study, Jaward et al. (2005) employed PUF samplers simultaneously at 77 sites and measured the air between Sep. 21 and Nov 16, 2004. Rural and urban sites were sampled in China (32 samples), Japan (20), South Korea (15), and Singapore (10). BDEs measured include 17, 28, 32, 47, 49, 75, 99, and 100. Total BDEs ranged from 0.1 to tens of pg/m^3, which the authors note is consistent with measurements for the remote coast of Ireland and rural/semirural England. However, some samples in China measured up to 340 pg/m^3 in an industrial city known to manufacture electronics, which is similar to values reported in urban United Kingodm where PBDE usage is known to be high. Mean or median concentrations were not provided. Only ranges were given and, as such, trends between congeners cannot be described. With a DL of 0.13 pg/m^3, maximums found include (in units of pg/m^3) the following: BDE 17: 35 in China, <1.7 otherwise; BDE 28: 130 in China, 6, 52, and 2.6 otherwise; BDE 32: 13 in China, <1.2 otherwise; BDE 47: 78 in China, <10 otherwise; BDE 49: 48 in China, <3 otherwise, BDE 75: 13, 12, 19, 1.2 in four locations; BDE 99: 50 in China, <10 otherwise; BDE 100: 5.5 in China, <2.3 otherwise. It was stated that 47 and 99 dominated the profile, contributing around 75% to the overall BDE burden. A similar approach was taken in Europe: 71 PUF samplers were deployed in 22 European countries, including 25 in urban locations

and 46 in rural/remote locations. BDEs measured and the results, percent detected in parentheses, and ranges, pg/m^3, are as follows: BDE 28 (82%): <0.5-30; BDE 47 (55%): <8-80; BDE 49 (30): <0.5-12; BDE 75 (54%): <0.5-3; BDE 99 (45%): <10-120; BDE 100 (41%), <2-20; BDE 153 (55%): <0.7-15; BDE 154 (44%): <0.8-10. Generally there was over a 700 fold ratio between high and low total measurements, with the highest being in urban locations in the United Kingdom and the lowest in remote areas of Iceland, Ireland, Norway, and Sweden. Generally low levels were found in Eastern Europe. As in Asia, BDEs 47 and 99 constituted about 75% of the total PBDEs.

A study measuring BDEs in China suggested much higher concentrations compared to the measurements of Jaward et al. (2005) discussed above. Chen et al. (2006) reported on results from 32 pairs of samples collected from four sites—2 industrial, 1 urban, 1 background, in China during June 15-30, 2004. High volume PUF/GFF PS-1 samplers were used and detection limits were 0.14-0.58 pg/m^3 for BDEs 28, 47, 66, 85, 99, 100, 138, 153, 154, and 183, and was 14.3 pg/m^3 for 209. BDE 209 dominated the profile, averaging 4,200, 750, 264, and 478 pg/m^3 for the two industrial, urban, and background setting, while the sum of the other PBDEs correspondingly averaged 3,673, 230, 89, and 105 pg/m3. The results appeared to be correlated to wind patterns: when blowing in from the west where there were several big electronic markets (where computers are assembled and dismantled), BDE 209 used in circuitry was very dominant; whereas when wind blew in from the southeast, directly from industrial areas, the sum of the other BDEs dominated. Congener patterns in one of the industrial sites, the urban sites, and the background sites suggests nearby use of the commercial formulation, with the high readings of BDE 209, and the nearby use of the commercial penta formulation, with BDEs 47 and 99 comprising greater than 50% of the total.

In summary, the major trends seen from the air monitoring studies include: 1) outdoor concentrations measured in the United States tended to be in the range of 20–200 pg/m^3 for total BDEs; 2) the profile was dominated by BDEs 47 and 99, suggesting a penta formulation influence; however, when BDE 209 was measured, it was seen to have concentrations equal or greater than 47 and 99; one study in Chicago had BDE 209 at concentrations 6 times higher than 47 or 99, and a study in China also showed a dominance of BDE 209 in air profiles of industrial, urban, and background settings; 3)

concentrations were found to be higher in industrial/urban settings as compared to rural/background settings. This is consistent with the expectation that concentrations should be higher nearer to where BDEs are released from their use in commercial products – such as near a source burning such products (like the UK bonfire described above) or shredding/recycling such products (like the autoshredding and recycling sites studied by CARB and described above); and 4) comparisons with measurements in Europe and Asia are hard to make—one study from China suggests airborne concentrations well into the thousands, while other measurements in Europe and Asia appear more in line with those in the United States with total BDEs mostly under 100 pg/m^3.

4.5.2. Indoor Air and Simultaneous Indoor/Outdoor Monitoring

The primary sources of PBDEs to which the general population is exposed are the products in which they are used. This would suggest that indoor exposures would be of primary importance for this class of PBTs, and, specifically, indoor air might contain higher concentrations of PBDEs as compared to outdoor air. Because the primary sources tend to be indoors, several researchers have focused their efforts in the indoor environment, and their efforts include simultaneous indoor/outdoor air measurements. In all these studies, the indoor measurements are higher than nearby outdoor measurements, often by factors of 10 or more. There is some evidence that combustion of products containing PBDEs release the PBDEs, but the evidence for this was primarily an open-air bonfire, not a controlled waste combustion process (see discussion above on the United Kingdom bonfire study by Farrar et al., 2004). There is also evidence that combustion of products containing PBDEs can result in formation of polybrominated dibenzo-p-dioxins and dibenzofurans (PBDD/Fs), and monobromo-polychlorinated dibenzo-p-dioxins and dibenzofurans (MoBPXDD/Fs). Hayakawa et al (2004) found that the levels of MoBPXDD/Fs correlated positively with that of polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs) in Japanese ambient air testing, and cited other laboratory testing showing concurrent formation of PCDD/Fs and MoBPXDD/Fs (Sakai et al, 2001). They also showed a correlation between PBDD/Fs and some key PBDEs, citing other work showing formation for PBDFs from combustion of materials containing

decaBDE (Lenoir et al., 1994). So, while the combustion of PBDEs appears to produce other brominated compounds, unlike dioxins, PBDEs are not formed themselves from combustion.

Unfortunately, United States researchers have not measured indoor air as much as indoor dust, or as much as researchers from Canada and abroad. Only two studies have been located which measured indoor air: The first was an indoor air study in which dust were sampled in 20 urban residences in the Boston, MA, with the air data reported in Allen et al. (2007). At each home, one personal and two area samples were taken over the course of a week, presumably during 2005 (the date of sampling was not provided but the data was reported in 2006). BDEs include 17, 28/33, 47, 49, 66, 85/155, 99, 100, 153, 154, and 209. The area samples were taken in the bedroom and living room, and the personal air sample was taken near the breathing zone in the bedroom. Participants turned on the samplers when returning in the evening, and turned them off when leaving to work. The sum of the geometric mean concentrations of reported congeners over the 20 residences were 605, 392, and 366 pg/m^3 for the personal, bedroom, and living room air. BDE 47 had the highest concentration, with geometric means of 227, 158, and 145 pg/m^3 for the same three locations, with BDE 209 second highest at 174, 95, and 94 pg/m^3, and BDE 99 third highest at 111, 67, and 60 pg/m^3.

The second United States study found for indoor air measurement of PBDEs was conducted by CARB (2005). Air monitoring was conducted indoors within a computer training facility in a public office building, within and around an electronics recycling facility, and outside of an automotive shredding/metal recycling facility (the recycling and shredding sites were also described in the previous section) on the UC Davis campus. This was the most comprehensive single data set that could be found in the literature, with these four sites and the measurement of 33 congeners. The octa- and nona-BDEs were not measured, however, for most of the samples because there was analytical intereference. The sampling for the indoor training facility included sampling when the computers were *on* versus *off* and also included carpet dust samples. The dominant congener found in the air was BDE 47, followed by BDEs 99, 100, and 28. In all cases, BDEs were higher in air when the computers were *on* versus *off*, but the most meaningful difference was for BDE 209, when considered in terms of the relative difference between

off and *on*, rather than on the magnitude of the concentration alone. Specifically, BDE 209 increased from 2 pg/m³ when the computers were *off* to concentrations from 56-74 pg/m³ when they were *on* in one set of tests and from 18, when *off*, to 47-56 pg/m³, when *on*, in the second test. The largest absolute concentration difference was for BDE 47, which was 213 and 112 pg/m³ in the two tests (starting from concentrations in the 800 pg/m³ range in the *off* condition and then increasing by 213 and 112 pg/m³ when the computers were turned *on*), followed by BDEs 99, 100, and then 209. The average concentrations of total BDEs, *on* (3 measurements) averaged with *off* (1 measurement) on the 2 days of measurement was 1,550 and 2,010 pg/m³, with BDE 209 averaging 50 and 65 pg/m³ for the two days.

A second site studied by CARB was an electronics recycling facility. Outdoor measurements from 2 samplers in front and 2 in back suggested concentrations in the range of 2,000–3,000 pg/m³, dominated by BDE 209. Generally, sampling indoors showed substantially higher BDE concentrations compared to outdoors at this facility. Two samplers sampled 3 days consecutively in 2004 for a total of 6 samples. Concentrations ranged from 316,000 to 833,000 pg/m³. The overwhelmingly dominant congener was BDE 209, ranging from 79,000 to 833,000 pg/m³, with an average over 423,000 pg/m³. The next highest congeners were BDE 183, averaging 23,813 pg/m³, 153 averaging over 5,600 pg/m³, 154 averaging over 3,400 pg/m³, and then 99, 47, and 49, all about at 1,770 pg/m³. Measurements of the filter and XAD-2 separately showed comparable amounts in vapor (XAD-2) as compared to particle (filter) for the tri-BDEs, about 10 times more particle than vapor for the tetra-BDEs, and then essentially none in the vapor phase for penta-, hexa-, hepta-, and marginal amounts for deca BDE congeners.

Only the sampling within the computer training facility showing total concentrations ranging from 1,500–2,000 pg/m³ might represent non-occupational concentrations that could apply to the general public. However, these concentrations are high when compared to other indoor monitoring studies conducted in Canada and abroad.

Shoeib et al. (2004) reported on measurements from 10 indoor and 3 outdoor samplers in Toronto, Canada, using a traditional high-volume, two-phase air PS-1 air sampler. Samples were collected in Nov/Dec of 2001 and then again in March of 2003. BDEs congeners included 17, 28/33, 47, 85, 99, 100, 153, and 154. Total concentrations

outdoors were 39 and 48 pg/m^3 (2 locations, 3 samples) and indoors were 410, 358, 490, 2,088, 381, and 76 pg/m^3 (6 locations, 7 samples), leading to an average ratio difference of 15 between indoor and outdoor samples. BDE 47 represented about 46% of the sample, followed by 99 at 25%. Both the Junge-Pankow model (Pankow, 1987) and the model based on the octanol air partition coefficient, the K_{oa} model (Shoeib and Harner, 2002) very well predicted the gas/particle partitioning that was observed in the high-volume sampler. Observed particle phase percentages for the congeners, derived from the indoor sampler, from high to low are as follows: BDE 183–82%, BDE 154–82%, BDE 153–81%, BDE 85–75%, BDE 99–62%, BDE 66–24%, 47–20%, BDEs 28/33–4%, and BDE 17–3%.

Wilford et al. (2004) sampled air using a PUF disk as a passive sampler in 74 homes and at 7 outdoor sites during the winter of 2002/3 in Ottawa, Canada. Indoor air concentrations of PBDEs were log-normally distributed with a geometric mean of 120 pg/m^3 (high of 3,600 pg/m^3), which is approximately 50 times higher than outdoor air concentrations, <0.1–4.4 pg/m^3. Congeners measured include 17, 28, 47, 66, 71, 85, 99, 100, 153, and 154. The highest mean concentration was 47 at 160 pg/m^3, followed by 99 at 42 pg/m^3, 28 at 24 pg/m^3, and so on. Wilford et al. (2004) stated that the indoor passive samplers are sampling mainly the gas phase with only a small contribution from particulates. It was found that the technical formulations tend to be enriched with heavier congeners, 99 and 100, while the indoor air was dominated by the lighter more volatile congeners, 47 and 28.

Limited studies abroad (United Kingdom and Kuwait) showed indoor concentrations of total BDEs similar to these Canadian studies suggesting a range of 20–200 pg/m^3 total. However, like the Canadian studies, measurements were not made for BDE 209. Hazrati and Harrad (2005) passively sampled 12 homes, 10 offices, and 1 private car in the United Kingdom for a period of one year, for sampling events which took between 4 and 6 weeks. BDE congeners sampled include 28, 47, 49, 66, 85, 99, 100, 153, and 154. Concentrations ranged between 5 and 1,418 pg/m^3, with a mean of 148.2 and median of 38.4 pg/m^3. In comparison with outdoor air from other studies, the authors suggested a 20-fold difference between indoor and outdoor air. PUF samplers were used to measure air samples and bulk dust samples were obtained from vacuum

bags in 17 homes in Kuwait between February 29, 2004 and April 11, 2004 (Gevao et al., 2005). Individual congener concentrations included 28, 47, 100, 99, 85, 154, 153, and 183. Air concentrations ranged from 2.5-385 pg/m^3, with a geometric mean of 10 pg/m^3. BDE 47 was the most abundant congener representing, on average, 51% of the total PBDE concentration measured. The next most abundant congener, BDE 99, represented about 28% of the total.

Harrad et al. (2004) presented BDE congener data on 47, 99, 100, 153, and 154 for indoor and outdoor air, and meat and vegan diets. The purpose of these measurements was to estimate daily exposure to BDEs via inhalation and diet. Indoor air concentrations were much higher than outdoor air concentrations; BDEs 153 and 154 were comparable in both environments, but 47, 99, and 100 were each over 100 times higher indoors than outdoors. The mean total concentrations of BDEs in air in various environments include the following: 21 pg/m^3 in outdoor air, 525 pg/m^3 in domestic indoor air, and 2,788 pg/m^3 in workplace environments.

Harrad et al. (2006) collected indoor air samples at 92 locations in the United Kingdom, including 31 homes, 33 offices, 25 cars, and 3 public locations (post office, coffee shop, and supermarket) using PUFs. They measured BDEs 28, 47, 49, 66, 85, 99, 100, 153, and 154. The overall average of all measurements was 273 pg/m^3 total, with a median of 47 pg/m^3 and a maximum of 8,180 pg/m^3 found in cars. Cars had the highest average concentration, although that was skewed by three measurements greater than 2,000 pg/m^3; the highest concentration in the non-car environments was 1,416 pg/m^3 and the average excluding the cars was 110 pg/m^3. Like other studies, the major contributors to these concentrations were BDEs 47 and 99.

Karlsson et al. (2007) report on measurements of PBDEs in indoor air, housedust, and blood from five households, although air was not sampled in one of the households. Unlike some of these other studies, BDE 209 was measured. This congener was found in 1 of 4 samples, at 257 pg/m^3 (DL = 173 pg/m^3). BDE 47 was found in 3 of 4 samples, ranging from 126 to 171 pg/m^3. The only other two congeners quantified in air were BDEs 28 and 66, and they were found in 7 of 8 measurements ranging from 6 to 28 pg/m^3.

Mandalakis et al. (2008) measured the levels of PBDEs inside automobile cabins in Greece. They measured the indoor air of 31 cars, taking 41 samples from low-volume samplers which were on for 48 hours, both when the car was in use and when not in use. The median concentration of total PBDEs was 201 pg/m^3, dominated by BDE 209, explaining about half the total concentration. Seventeen other congeners were measured, with the next two predominant congeners being BDEs 47 and 99. The maximum found was over an order of magnitude higher than this median at 2644 pg/m^3.

In summary, only one study could be located which measured indoor air concentrations of BDEs in the United States in residences rather than in occupational settings. This was the study of urban residences in the Boston, MA, area, where the average total concentration was in the range of 200–500 pg/m^3 total. The only other indoor measurements came from within a computer lab, and total concentrations were in the range of 1,500-2,000 pg/m^3, with BDE 209 in the range of 50-70 pg/m^3. This was higher than indoor concentrations measured in Canada and abroad, which were mostly less than 200 pg/m^3 total, although BDE 209 was most often not measured in studies in Canada or abroad. Indoor industrial/occupational concentrations were substantially higher, with measurements in the hundreds of thousands of pg/m^3. In United States, Canadian, and studies abroad, indoor concentrations were found to be higher than outdoor concentrations in simultaneous measurements by factors of 10 to 100. BDE 47 appears to dominate indoor air concentrations, encompassing about one-third the total congener concentration when BDE 209 was measured in one study (Allen, et al., 2007), and about half the total concentration in other studies when it was not measured. In the one study where BDE 209 was measured in the indoor environment, it was the second highest concentration, followed by BDE 99. In one study of PBDE concentrations inside cars, BDE 209 did dominate the profile explaining about half the total concentration.

4.6. FISH CONCENTRATIONS

This section reviews the data on fish, including fish caught in the wild, farmed fish, and fish samples from market basket surveys. While emphasis is on the United States studies, noteworthy studies from Canada and abroad are included as well. Like much of the data on other environmental levels, these studies often did not include BDE

183 and 209, the primary markers for the octa and deca formulations, respectively. Another issue for evaluation and comparison of fish studies is that some results are reported on a wet weight of whole tissue and others on a lipid-basis. Like other PBTs, PBDEs bioaccumulate in lipids of animals, so it would be useful to have all data reported on both a wet weight and a lipid-basis. While the author's reported results are provided below, lipid- or wet-based concentrations are concurrently provided in parenthesis when possible. Table 4.3 provides congener-specific fish concentrations for fish caught in the United States.

4.6.1. Farmed Fish Concentrations

A total of 70 farmed and wild salmon were collected from wholesale and retail outlets in Maine in August 2003 and May 2004 (Shaw et al., 2005). They represented salmon from three regions: two farms in eastern Maine, three in eastern Canada, and one in Norway. Samples were composited so that the Maine sample results were displayed for the two farms. Results were provided for 9 congeners including 28, 47, 66, 85, 99, 100, 138, 153, and 154. Samples were analyzed with skin on and skin off to see if that made a difference, and results suggested essentially no difference. There was also not a correlation between lipid content and fresh weight concentrations, which is counter-intuitive. It was found that levels, in the neighborhood of 1 ng/g total wet weight (wwt) basis, were 4-5 times lower than PBDE concentrations reported in farmed salmon from British Columbia and northwestern Europe, but comparable to levels found in farmed salmon from Chile.

Jacobs et al. (2002) measured PCBs, DDT, and PBDEs in farmed and wild European Atlantic salmon, aquaculture feeds, and fish oils used to supplement the feed. Seven British salmon samples, 5 additional salmon samples (two from Ireland, and 3 purchased from a Belgian market), 8 salmon feeds (from 4 different Scottish sources), 5 fish oils, and 1 vegetable oil were analyzed. BDE congeners analyzed include 28, 71, 47, 75, 66, 100, 99, 153, and 154. Total BDEs ranged from 1.1–85.2 ng/g lipid weight (lwt) in 13 salmon samples (average = 33.8), with the highest found in a wild salmon sample. BDE 47 predominated, averaging about 53% of total. The levels of BDE in feed ranged

from 8.1 to 23.9 ng/g lwt for 8 feed samples, and the range in fish oil was ND to 12.7 ng/g lwt. BDE 47 similarly dominated the feed and fish oil samples.

The most comprehensive study on BDEs in farmed fish was conducted by Hites et al. (2004). PBDEs were measured in about 700 farmed and wild salmon collected from around the world, including from Maine and Washington, at the AXYS Analytical Lab in Sidney, BC. PBDE congeners 1, 2, 3, 7, 8, 10, 11, 12, 13, 15, 17, 25, 28, 30, 32, 33, 35, 37, 47, 49, 66, 71, 75, 77, 85, 99, 100, 105, 116, 119, 126, 138, 140, 153, 154, 155, 166, 191, 183, 190, 206, 207, and 208 were found. BDE 209 was looked for but not detected with a detection limit of 0.1 ng/g wwt (other congeners had DLs at 0.001 to 0.01 ng/g wwt). The concentrations of total PBDEs in these samples ranged up to 10 ng/g wwt. In order of magnitude, the samples suggested the following: Europe farmed salmon > North America farmed salmon > Chile farmed salmon > wild salmon. Farmed salmon ranged from around 0.3 up to nearly 10 ng/g wwt, while the bulk of wild salmon were less than 0.3 ng/g wwt. In terms of wild salmon species, Chinook was the highest, with an average of 2.26 ng/g wwt for the 9 samples as compared to 0.13 ng/g wwt for the 36 others. In terms of composition, BDE 47 dominates (around 47%), followed by BDE 99 (18%), BDE 100 (10%), BDEs 28/33 and 49 (5%), with other congeners under 5%. Although not found in the farmed salmon, BDE 209 was found in the salmon feed and at about 15% of total concentration. Total PBDE concentrations in 13 feed samples ranged from 0.5 to 10.9 ng/g wwt.

Hayward et al. (2006) collected 18 samples of farmed and wild caught fish from supermarkets in Maryland or Washington, DC in 2004, and an additional 4 samples were collected in 2001, including one from North Carolina. There were 6 farm-raised salmon, 6 wild caught salmon, 5 bluefish, and 5 rockfish among the 22 samples. They were analyzed for 22 BDEs, including 209, but only BDEs 28, 47, 49, 99, 100, 153, and 154 could be found routinely. Individual congener results were graphed and not provided in a table, so they were not added to Table 4.3. However, they appear consistent with other measurements described here and in Table 4.3. Wild bluefish had the highest total PBDE concentration averaging 15.1 ng/g wwt (n = 5), followed by wild rockfish at 5.4 ng/g wwt (n = 5), farmed salmon at 1.0 ng/g wwt (n = 6), and Alaskan salmon (King, Coho, Sockeye) at 0.4 ng/g wwt (n = 6). BDE 47 had the highest concentrations, explaining

55–70% of the total, followed by 99 and 100, both of which took turns being second behind 47 in different samples. BDE 153 was not found in wild salmon and only found at 1–3% of total in other species, and BDE 183 was found in only one sample.

4.6.2. Fresh Water and Marine Fish

Oros et al. (2005) measured 22 BDE congeners but detected only BDEs 47, 99, and 100 in bivalves (clams, mussels, and oysters) in composited samples originating from 16 locations, at wet weight concentrations with an average of 5.7 ng/g wwt, ranging from 2–13 ng/g wwt (855–13,502 ng/g lwt) in the San Francisco Estuary. The BDE congener found most abundantly was 47; it is found in all samples above its MDL and comprising about 50-70% of the total concentration, while 99 and 100 were found in about 50% of the samples.

Manchester-Neesvig et al. (2001) sampled 21 coho and Chinook salmon from Lake Michigan tributaries in 1996 and analyzed them for 6 PBDEs: 47, 66, 99, 100, 153, and 154. These samples have among the highest fish (and terrestrial animal) concentrations in the literature, ranging from 45 to 148 ng/g wwt (773 to 8,120 ng/g lwt), with an average of 80.1 ng/g wwt (2,440 ng/g lwt). BDE 47 dominated the concentration, comprising 56% of the total, followed by BDE 99 at 19%, BDE 100 at 12%, BDE 154 at 6.6%, and BDE 153 at 3.6%. The concentrations of PCBs tracked very well with PBDEs, suggesting that PBDEs have been part of Lake Michigan for many years, like PCBs.

A more comprehensive evaluation of Great Lakes fish was conducted by Zhu and Hites (2004). Lake trout collected between 1980 and 2000 from Lakes Superior, Michigan, Huron, and Ontario, and walleye from Lake Erie, were analyzed for 15 PBDEs, including 17, 28, 47, 49, 66, 71, 85, 99, 100, 138, 153, 154, 183, 190, and 209. The levels of BDE 209 found in all fish samples was in the undetectable range of 3.6 ng/g lwt, thus it was deemed to be ND in all samples and not reported. Total PBDEs (sum of BDEs 47, 99, 100, 153, and 154 only) rose from under 15 ng/g lwt in 1980 to the range of 58-180 ng/g lwt in 1990, and finally in the range of 400-1,400 ng/g lwt in 2000. The authors concluded that the trends suggest a doubling time of 3-4 years. The authors discussed some spatial trends, but mostly the concentrations in the lakes over time were

comparable. The summary information is the average congener concentration over all lakes for the most recent year, 2000. There was a systematic change of congener distribution over time. More of the octa-BDE product, which was high in 153 and 154, was used in the 1980s, and there was a higher proportion of these two in the 1984 fish as compared to the 1996 fish. The ratio of penta-BDE use divided by octa-BDE in the mid-1980s was 0.7 (more octa used), but by the mid-1990s, there was a shift and the ratio was now 2 (twice as much penta used). Subsequently, fish in the latter 1990s were dominated by BDE 47.

Batterman et al. (2007) also conducted a comprehensive temporal study of archived and fresh fish from the Great Lakes. Chapter 3 summarized this study as it measured numerous archived and freshly caught fish from all of the Great Lakes from 1979 to the present. Batterman et al. (2007) focused only on BDEs 47, 99, 100, and 153, and found consistent increases with doubling times in the range of 2-4 years. The total concentrations (sum of the four congeners) in trout ranged from about 20 to over 100 ng/g fw in the last samples taken, between 2000 and 2005. Concentrations were a bit lower in the last samples of smelt taken, between 3 and 81 ng/g fw. BDE 47 dominated the total, contributing about 70% of total concentration, with BDEs 99 and 100 contributing about 13% each.

Although the focus was on analog compounds (which are defined as compounds structurally analogous to PBDEs, such as hydroxylated PBDEs, or OH-PBDEs), 6 BDEs including 47, 99, 100, 153, 154, and 183 were measured in the plasma of fish from the Detroit River (Valters et al., 2005). Composited plasma samples (blood was centrifuged to separate red blood cells from plasma) were obtained from 13 fish species. Total PBDEs ranged from 0.16 to > 21.0 ng/g wwt (in channel catfish; all other samples less than 10.0 ng/g wwt). Given that plasma was 0.6-2.7% lipids, lipid concentrations would be much higher, at over 200 ng/g lwt. BDE 47 was the dominant congener (over 60% in most samples), followed by 99 and 100, which were of similar magnitude; then 153 and 154 at lower and sometimes non-detect values. BDE 183 was only detected in channel catfish at 0.805 ng/g wwt; BDE 47 was 11.5 ng/g wwt in this catfish.

A total of 63 samples of fish were collected from Washington State rivers and lakes by the Washington State Department of Ecology in 2005 and 2006 (Johnson et al.,

2006). These samples were measured for 13 congeners including 47, 49, 66, 71, 99, 100, 138, 153, 154, 183, 184, 191, and 209. Results for total ranged from ND to 1,059 ng/g wwt, with a median of 2.8 ng/g wwt and a mean of 35 ng/g wwt (derived assuming ND = 0). The most frequently detected congener was BDE 47; it was detected 84% of the time with a median concentration of 1.5 ng/g wwt. It accounted for 68% of the total concentration found, with BDE 99 accounting for 16% as the second highest concentration. Other congeners detected frequently equal BDEs 100, 154, 153, and 99, detected 51, 49, 40, and 38% of the time. BDE 209 was not detected, but it had a high detection limit ranging from 1-6 ng/g wwt (all other detection limits were less than 0.5 ng/g wwt). The Spokane River was unambiguously the most impacted river, with 3 samples taken measuring 76, 417, and the survey maximum of 1,059 ng/g wwt. Not ironically, this river also had the highest water concentrations in this same survey, with two measurements above 100 pg/L at 146 and 926 pg/L, with all other water measurements well under 100 pg/L. Johnson et al. (2006) report on an earlier study by the Washington State Department of Ecology on fish in the Spokane River, and they similarly found high concentrations, ranging from 30 to 1,222 ng/g wwt in 47 samples (some of which were composites).

Fish from three locations in the Savannah River were sampled in 2005 and analyzed for 12 BDEs (Sajwan et al., 2006). Individual congener results were not provided, and results suggested generally higher levels of BDEs, similar to other fresh water bodies, with totals at 10 to >300 ng/g lwt (which would equal about 1/10 as much on a fresh weight basis). The interesting finding from this study was that the predominant congener was identified as BDE 30 in all three locations, a congener which not looked for in any other fish study. The second most predominant congeners were the familiar BDEs 47 and 99.

Ashley et al. (2006) analyzed samples of eel and sediment from the Delaware River (sediment samples described above). The eel samples were collected in 1998 as part of an earlier effort focusing on PCBs. Eel was selected as a good bioindicator of the quality of the water body since eels have a small range of habitat throughout the water body where they live. Like other aquatic biota samples from freshwater systems, the PBDE concentrations in these eels were high, with totals ranging from 1 to 408 ng/g wwt,

with an average of 86 ng/g wwt over 17 samples. The predominant congener was BDE 47, explaining 56% of the concentrations. BDE 100 was the next most predominant congeners at 30%, and other congeners contributed 3% or less. BDE 209 was not detected in the samples, although the detection limit was not provided.

Hale et al (2001) measured BDEs 47, 49, 99, 100, 153, and 154 in 70 samples of fish from the Roanoake River, the Dan River, and the Hyco River, which are within two large watersheds in Virginia. Overvall statistics of findings were not provided. It was noted that BDE 47 was quantified (at over 5 ng/g lwt) in 88% of samples, with over half the fish samples having concentrations greater than 100 ng/g lwt, and with 16 samples containing BDE 47 at over 1000 ng/g lwt. The highest total concentration was 47,900 ng/g lwt. Overall, BDE 47 dominated the profiles, explaining between 40 and 75% for the five different species tested, with BDEs 99 and 100 explaining in the range of 5 – 20% of concentrations. BDE 49 was also measured and identified, generally explaining less than 5% of total PBDE concentrations in the sampled fish.

Marine fish sampled off the coast of Florida were analyzed for 12 PBDEs by Johnson-Restrepo et al. (2005). Specifically, a total of 88 specimens from nine species of marine fishes and two species of dolphins were analyzed for BDEs 28, 47, 66, 85, 99, 100, 138, 153, 154, 183, 203, and 209. The overall mean concentration of total PBDEs were 10-fold greater than the levels reported for Arctic marine fish such as polar cod from Storfjorden, and for several fish species from southern Greenland. On the other hand, the concentrations were one order of magnitude lower than what was reported for lake trout in the Great Lakes. Total concentrations in teleost fishes ranged from 8 ng/g lwt to 88 ng/g lwt, with a mean of 43 ng/g lwt. Mean concentrations of total BDEs in sharks and dolphins were higher, ranging from 37.8 ng/g lwt to 1,630 ng/g lwt. BDE 47 was overwhelmingly found at the highest concentrations in teloest fish and dolphins (10s of ng/g lwt in teleost, but 100s of ng/g lwt in dolphins), but BDE 209 was the highest found in sharks (16-778 ng/g lwt). Other than for sharks, BDE 209 was found at an average of 0.5 ng/g lwt with several non-detects. Johnson-Restrepo et al. (2005) also did a temporal study, evaluating 9 samples of dolphin blubber collected between 1991 and 1996, with 6 analogous samples collected between 2001 and 2004. The authors similarly also compared bull shark samples collected in 1993 and 1994 with those collected in

2002 and 2004. A clear elevation was seen in both PCB and PBDE concentrations; total PBDEs in the dolphins rose from 363 to 1,190 ng/g lwt, and total PBDEs in sharks rose from 78 to 1,630 ng/g lwt.

Rayne et al. (2003) studied the temporal trend of increasing BDE concentrations in fish from the Columbia River System in southeastern British Columbia. The concentrations of 33 individual mono- through hexabrominated BDE congeners (which includes congeners up to BDE 156) were measured in mountain whitefish, largescale suckers, and surficial sediments from several locations on the Columbia and Kootenay River systems in southeastern British Columbia, Canada. A total of 41 Whitefish samples were obtained from the period of 1992 to 2000, specifically 1992, 1994 and 1995, 1998, and 2000. Eleven sediment and 6 sucker samples were taken in 2001 and 2000, respectively. Total PBDE concentrations in whitefish, obtained at two locations, increased by factors of 11.8 and 6.5, respectively, over the period from 1992 and 2000; they went from 6.1 ng/g wwt (avg) in 1992 to 19.1 ng/g wwt in 1994 and 1995 to 71.8 ng/g wwt in 2000. The 6.5 factor increase at the other location was seen from the starting concentration of 4.5 ng/g wwt to 29.2 ng/g wwt. At a remote site, total PBDE was 0.9 ng/g wwt in the whitefish, which the authors attribute to domestic wastewater draining directly into the river at the other two sites. Suckers sampled in 2000 had lower concentrations than whitefish at 5.0 ng/g wwt. The sediment congener pattern was somewhat different than the fish. The primary congeners in the sediment and fish were 47, 99, and 100, but BDE 47 was the major congener in sediments (46-63% total), followed by 99 (23-39%), and 100 (6-8%), while for whitefish, 99 was the dominant congener, followed by BDEs 47 and 100. This is contrary to other literature, which showed similar dominance of 47 in both sediment and fish.

Vives et al. (2004) collected fish from eleven high mountain lakes in Europe and one in Greenland. The importance of these lakes being high is that the only way BDEs could have reached these aquatic ecosystems is by long range transport. Liver and muscle tissue of trout (brown trout, brook trout, and arctic char) were sampled. The authors were unclear as to which BDEs were measured: they stated that all congeners were identified using external standards comprised of 39 individual congeners up to BDE 190 (not including 209), but they only present results for BDEs 28, 33, 47, 99, 100, 153,

and 154, while not providing any information on other congeners including that they were looked for but not detected. Results from 55 trout specimens were as follows: 0.1-1.3 and 0.07-0.77 ng/g wwt in liver and muscle, respectively (2.4-40.0 and 2.9-41.0 ng/g lwt). BDE 47 was the highest congener found, followed by 99, 100, 153, 154, and 28. This disparity was much more apparent in liver as compared to muscle. There was an age relationship found in the data: older fish had significantly higher BDE concentrations. Concentration increases of between 4 and 12 times were found between 1- and 21-year-old individual fish samples.

Peng et al. (2005) collected 60 tissue samples from 6 rivers and 3 estuaries in 2003 in China. BDE congeners 28, 47, 99, 100, 153, 154, and 183 were measured. Results showed river-average results ranged from 25 to 152 ng/g lwt, and 31 to 281 ng/g lwt from the estuaries. The fish species were not identified. In all rivers, BDE 47 is the predominant congener found, exceeding other congeners by factors of 3 or more and comprising over 50% of the total concentration. In most cases, BDE 154 was the next highest, followed by BDEs 99 and 100. The one exception was an estuary, where BDE 154 accounted for 101 ng/g lwt of a total of 281 ng/g lwt, followed by 47 at 92 ng/g lwt. In all other locations, BDE 154 was 10 or less ng/g lwt.

4.6.3. Fish From the Retail Marketplace

The fish data that might be considered the most relevant for estimating exposure to the general population is the retail market basket sampling described in this section. Also, this was essentially the only data on fish in the literature that reported measurements of BDEs 183 and 209. Fish were either the primary target of the sampling or included among a variety of food products.

Schecter et al. (2006a; derived from data in Schecter et al., 2004, with additional data and analyses) reports the results of a comprehensive market basket survey entailing 62 samples that encompassed meat products, dairy products, and fish. BDEs 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209 were reported, although the average results reported, which were based on the assumption of ½ DL, could be misleading due to high detection limits, coupled with a high frequency of "nd" (not detected) or "na" (not analyzed). For example, BDE 17 was only detected in 4 of 18 samples, at detection

limits ranging from 0.0001 to 0.0007 ng/g wwt. However, the mean was reported as 0.0008 ng/g wwt, due to several samples with detection limits above 0.002 ng/g wwt. The detection limit issue was even more apparent with BDE 209. For example, the mean concentration for BDE 209 in fish was 0.1 ng/g wwt, but it was not detected in 14 of 24 samples, and the high mean was driven by two samples at 1.27 and 0.68 ng/g wwt; all other positive detections were less than 0.025 ng/g wwt. The detection limits on BDE 209 were highly variable, ranging from 0.011 to 0.17 ng/g wwt. The data regarding the more commonly found BDEs at higher concentrations (congeners 47, 99, 100, 153, and 154) must be assumed to be valid. A second key limiter of this data set is that all samples were purchased from supermarkets in Dallas, TX, so it is unclear to what extent this data represents national trends. A large proportion of the food supply is national in scope, such as non-perishable or frozen food items, but others might be locally produced (fruit and vegetables) or perhaps regionally produced (animal food products). Still, this data set represents the most comprehensive survey of terrestrial animal food products in the United States analyzed for PBDEs. The top four BDE congeners found in fish were BDE 47 at 0.6 ng/g wwt, BDE 99 at 0.17 ng/g wwt, BDE 100 at 0.13 ng/g wwt, and BDE 209 at 0.098 ng/g wwt. The mean concentration of BDE 183, the primary marker for the octa BDE formulation, was 0.002 ng/g wwt, and the mean total concentration over all 24 fish samples was 1.12 ng/g wwt.

Fish, meat, and fowl products were purchased in December 2003 and February 2004 from 3 different food markets in Sacremento and El Dorado Hills in Northern California (Luksemburg et al. 2004). A total of 31 different BDE congeners were measured, although no congener-specific data were provided. Homologue BDE groups were measured, and tables were provided with data for those. The total concentrations found in fish ranged from 0.09 to 4.9 ng/g wwt.

The Norwegian Institute of Public Health in Oslo, Norway conducted an intercomparison laboratory study on the measurement of PBTs including PCDD/Fs, PCBs, and PBDEs (Haug et al., 2005). A total of 73 laboratories participated but only 21 laboratories reported back concentrations of 7 PBDEs in samples of chicken, trout, and palm oil. The 7 BDEs were 28, 47, 99, 100, 153, 154, 183, and 209. Lake trout had the highest concentrations by a large margin: mean concentrations were 0.6; 95.2; 78.6;

39.2; 9.9; 12.0; 0.02; and 0.06 ng/g wwt for the 8 congeners, respectively, totaling 236 ng/g wwt. These results appear anomalous, not only because the concentration were so much higher than anything else in the literature (total PBDEs have been found in the single digit ng/g wwt or less, not in the hundreds of ng/g wwt), but also because their reported concentrations in chicken and palm oil were up to 4 orders of magnitude lower, at concentrations less than 0.01 ng/g wwt.

Tittlemeier et al. (2004) purchased 122 fish and shellfish from retail stores in 3 Canadian cities (Vancouver, Halifax, and Toronto) in the winter of 2002 and analyzed them for 18 BDE congeners. The fish types include salmon, trout, tilapia, Arctic char, mussels, oysters, shrimp, and crab, and the BDEs include 15, 17, 28, 47, 66, 71, 75, 77, 85, 99, 100, 119, 126, 138, 153, 154, 183, and 190. Total BDEs per fish group ranged from 0.02 in shrimp to 1.6 ng/g wwt in trout. Salmon was second with a geometric mean concentration of 1.5 ng/g wwt. Trout and salmon had the highest lipid content at 8 and 11%, respectively, partially explaining the high amount of BDEs in them. The third highest lipid content, at 7.9%, was found in char, and not unexpectedly, char had the third highest total BDE, at 0.6 ng/g wwt. BDE 47 was the most predominant congener, explaining 48% of the concentration, with BDE 99 contributing 24%. Following these two were BDEs 100, 28, 153, 154, and 183. Farmed samples showed higher concentrations than wild samples, particularly for salmon.

Ohta et al. (2002) evaluated the concentration of PBDEs in breast milk and food products, including several fish species, in Japan. The PBDEs evaluated included 28, 47, 99, 153, and 154. Concentrations in 20 fish samples (4 young yellowtail, 4 mackeral 2 natural yellowtail, 3 salon, 3 yellow tuna, and 3 short-necked clams) ranged between 0.02 and 1.65 ng/g wwt in the edible tissues of the fish, with the highest in yellow-fin tuna. The dominant congeners were BDE-47—at over 50% in the samples—and BDE-99— at about 20%. Questionnaires on food consumption were given to the women, and a strong correlation was found between consumption of fish and breast milk concentration. Specifically, in a "high" group of fish consumers (n = 5), the average breast milk concentration of BDEs were 1,724 pg/g lwt, and this was significantly higher than the concentration of BDEs in the "low" group of fish consumers (n = 3), which was 774 pg/g lwt.

Pirard et al. (2005) presented results from salmon, whole trout, and Spanish mussels purchased from a Belgian supermarket. Total concentrations were 8.19, 2.69, and 10.22 ng/g lwt for mussel, trout, and salmon, respectively. BDE 47 dominated the results, comprising over 60% for the three samples, followed by 99 (in mussel and trout) or 100 (in salmon).

Meng et al. (2007) collected samples of 13 fish species from local fish markets and supermarkets from 11 fishery-producing regions in Guangdong Province, China. These include freshwater farmed fish, seawater farmed fish, and wild marine fish. Eleven congeners were measured, including BDE 209. The median and mean of the total of 10 BDEs, not including 209, were 0.16 and 0.23 ng/g wwt, respectively. BDE 209 was found in only 14 of the 390 fish samples, ranging from < 0.1 to 0.57 ng/g wwt, although the detection limit of BDE 209 was the highest, at 0.1 ng/g wwt, compared to 0.001– 0.003 ng/g wwt detection limits of other congeners.

4.6.4. Observations from Fish Data

While this review falls short of a comprehensive review of the literature on brominated flame retardants in fish, certainly it should be clear that sampling for fish is of critical importance, both in the context of sampling fish as food in retail markets and fish farms, and sampling in open aquatic settings as a marker for understanding and tracking the status of PBDEs in the environment. Unfortunately, most of this sampling did not include the higher brominated marker congeners 183 and 209. The very limited sampling for BDE 183 suggested insignificant concentrations, but the market basket sampling by Schecter et al. (2006a) and the marine environment sampling by Johnson-Restrepo et al. (2005) suggested that BDE 209 can be significant and sometimes the highest congener found in fish. Most often, however, BDE 47 dominated the profile, explaining over 50% of the concentration found, with BDE 99 the second highest found explaining around 25%. The ratio of BDE 47 to BDE 99 was about 2.0 in these studies. Generally, total BDE concentrations were highest in open water environments (lakes, rivers, oceans) in contrast to farmed fish or fish obtained from marketplaces. In the wild environment, concentrations ranged above 1,000 ng/g wwt but most often were well above 10 ng/g wwt averaging between 10 and 100 ng/g wwt. Concentrations in farmed and market fish

were generally lower, in the neighborhood of 1–5 ng/g wet weight basis. It is not clear why store-bought fish might be lower than wild caught fish, except possibly that the focus of wild caught fish is from locations historically known to be impacted by contaminants such as dioxins, PCBs, or PBDEs, such as the Great Lakes. Like sediment cores (described in the previous chapter) and body burden measurements (described in the next chapter), temporal sampling of fish has provided evidence of the rise of these compounds in environmental matrices throughout the 1990s into the 21st century.

4.7. FOOD CONCENTRATIONS

United States data on food is highlighted by three market basket surveys: one in Texas (Schecter et al., 2006a), one in California (Luksemburg et al., 2004), and one sampling from several states in the United States (Huwe et al., 2006). Other data include earlier sampling by Huwe et al. (2003) on chicken in a research mode and several studies from abroad. Similar to the fish data, there were inconsistent reports in terms of lipid weight or wet weight. Table 4-4 provides congener-specific data on food concentrations.

Schecter et al. (2006a; derived from data in Schecter et al., 2004, with additional data and analyses) reports on a sampling of 62 food items, split between samples of meat, dairy, and fish. BDEs 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209 were reported, and, as described in Section 4.6.5, there may have been some analytical issues given the high detection limits and a high frequency of non-detects in this data set. In addition to these limitations, all the food originates from Texas supermarkets only. Section 4.6.5 above describes the results for the 24 fish samples. Of 18 meat samples, total BDE ranged from 0.04 ng/g wwt to 1.4 ng/g wwt. BDE 209 was detected in 8 meat samples with concentrations ranging from 0.01 to 0.25 ng/g wwt. Generally, the highest BDE congeners were 47 and 99, with 99 the highest in meat averaging 0.16 ng/g wwt; 47 was at 0.09 ng/g wwt in meat. These two BDE congeners were similar in 15 dairy samples averaging about 0.03 ng/g wwt. The mean of BDE 209 in dairy was 0.04 ng/g wwt, but this was driven by a single sample of cream cheese at 0.48 ng/g wwt; all other samples were either non-detect (8 samples) or had concentrations under 0.02 ng/g wwt. The total average concentrations of BDE 209 in meat and dairy were 0.38 and 0.12 ng/g wwt, respectively.

Fish, meat, and fowl products were purchased in December 2003 and February 2004 from three different food markets in Sacremento and El Dorado Hills in Northern California (Luksemburg et al., 2004). Thirty-one different BDE congeners were measured, although no congener-specific data were provided. Homologue BDE groups were measured, and tables were provided with data for those. The total PBDE concentrations (sum of homologue contrations provided) were higher in fish (from 0.09 to 4.9 ng/g wwt) and fowl (0.09 to 2.5 ng/g wwt) than in beef and deer meat products (from 0.1 to 0.4 ng/g wwt). It was stated that the highest concentrations of individual congeners were 47, 99, and 100, although decaBDE, which is the single congener BDE 209, most often was the highest homologue group found.

A non-statistical sampling of market basket meat and poultry items from large supermarkets was undertaken in 2004, in these states: FL, VA, CT, PA, ND, MT, OR, NM and AZ (Huwe et al., 2005). A total of 65 meat samples including hamburger (n = 11), bacon (n = 11), chicken fat (n = 22), pork fat (n = 11), and beef fat (n = 10) were measured for BDEs 28/33, 47, 85, 99, 100, 153, 154, 183. Beef had the lowest amounts of BDEs (0.25 ng/g lwt for beef fat and 0.67 ng/g lwt for hamburger), while chicken and pork had the highest concentrations— 2.96 ng/g lwt and 2.62 ng/g lwt, respectively. These concentrations were driven up somewhat by a few high samples: One pork and two chicken samples had total BDEs greater than 15.0 ng/g lwt. BDE 99 dominated all food types, thus, accounting for 39%, 36%, 46%, 40%, and 44% for hamburger, bacon, chicken fat, pork fat, and beef fat respectively. BDE 47 was second most dominant, explaining 27%, 27%, 27%, 41%, and 28%, respectively. The ratio of BDE-47 to BDE-99 averaged 0.78, which is similar to the BDE 47/99 ratio of 0.6 found in a penta formulation. This contrasts a BDE 47/99 ratio of sometimes greater than 2, which was identified in several studies in fish as described in Section 4.6.5. As will be described in the next chapter, humans also have more BDE 47 congeners than 99, but about this same factor of 2.0.

Huwe et al. (2003) earlier conducted research-oriented studies on BDEs in poultry. Chicken fat samples from three chicken production sites known to have chickens "contaminated" by consumption of animal feed with ball clay which had high levels of dioxins, and two other chicken fat samples from an uncontaminated production site were

analyzed for BDEs. The congeners analyzed include the following: 17/25, 33, 47, 66, 100, 99, 154, 85, 153, 140, 138, 183, and 209. Four other unidentified congeners were noted, but they are not included in the summary. The samples from two of the "contaminated" sites did not appear to have BDE levels different than the noncontaminated production site. So, for calculation of averages, the nine samples from these three locations were averaged, and the four samples from the contaminated site, where levels were higher, was analyzed separately. The authors conducted a PBDE analysis of the stored contaminated feed but did not find elevated levels of PBDEs (data not provided). This suggested to them that feed was not the cause of high levels of PBDEs in the one site. The authors did state that in the one production site where BDE levels were higher, a factory producing penta-BDE formulations was located in the same city, which may have explained the higher levels found there. The average total BDE concentration of 9 "uncontaminated" chicken was 8.3 ng/g lwt, and the average of 4 "contaminated" chicken was 24.8 ng/g lwt. BDE 99 was the highest congener; it averages 3.1 ng/g lwt uncontaminated and 10.9 ng/g lwt contaminated. BGE congener 47 averaged 2.3 ng/g lwt uncontaminated and 6.9 ng/g lwt contaminated. BDE 209 was generally low in all samples, with actually lower concentrations in the contaminated versus uncontaminated sites; the average over all 13 samples was 1.1 ng/g lwt.

Studies of PBDEs in food abroad mostly originate from Europe, and results are comparable to America in that total PBDEs tend to be less than 1 ng/g, dominated by BDEs 47 and 99, with sparse data on BDE 209. As noted below, however, two studies have recently documented high levels of BDE 209 in food samples. In the United Kingdom study, BDE 209 was found at the highest concentration of all congeners in nearly every sample, and in the study from Spain, BDE 209 was found at the highest level in several food products. In both studies, high concentrations of BDE 209 were 10 times or more higher than other congeners.

A recent study commissioned by WWF (formerly World Wildlike Fund, now just WWF), conducted by the Netherlands Organization for Applied Scientific Research (TNO) (Peters, 2006), measured 30 BDEs in 26 food products from different countries in Europe. The food products ranged from honey in the United Kingdom, to salami in Italy, to pork chops in Poland. BDEs were found in 19 of 26 products: a low total of 0.15 ng/g

found in honey to a high total of 1.3 ng/g wwt in minced beef. BDE 209 was looked for but not found, although it cannot be expected to be found with a high detection limit of 5.0 ng/g wwt. Like other studies, BDEs 47 was found most frequently; it was found in 17 of 26 samples at an average positive concentration of 0.49 ng/g. Interestingly, BDE 32, not measured in other studies, was found next most frequently, in 12 of 26 samples, with an average positive concentration of 0.07 ng/g wwt. BDE 99 was found in 8 of 26 samples at an average positive concentration of 0.12 ng/g.

Another European study included a market basket survey of BDEs, including BDE 209, from Belgium (Voorspoels et al., 2007), although BDE 209 was not quantified in any sample. Concentrations generally were all less than 0.1 ng/g wwt, with fish being the highest generally, and only 2 of 7 food samples contained BDE concentrations greater than 1.0 ng/g wwt, at 1.0 and 1.6 ng/g wwt. Butter was the highest of the non-fish samples, at 0.8 ng/g wwt. The eight meat samples were all under 0.2 ng/g wwt, as were fast food and eggs. Cheese was slightly higher at 0.22 ng/g wwt.

As noted, two recent surveys showed high levels of BDE 209, one from United Kingdom (FSA, 2006) and one from Spain (Gomara et al., 2006). In the dietary survey conducted by the Food Standards Agency of the United Kingdom, composite samples representing 19 food groups were analyzed for a suite of 17 BDE congeners, including BDEs 183 and 209. The meat products concentrations of BDE 209 was exceedingly high at 3.64 ng/g wwt, and in fact, BDE 209 had the highest in all but 2 composite food group samples, although concentrations other than the meat concentration appear more in line with other values in the literature, at less than 0.5 ng/g wwt. For example, BDE 209 was found at 0.29 in "fats and oils" and the next highest congener concentrations were BDEs 49 and 99, both reported at 0.08 ng/g wwt. Although summary statistics were not supplied, generally BDEs 47 and 99 were similar at concentrations between 0.01 and 0.10 ng/g, consistent with other surveys, and other congeners were present but at lower concentrations. Gomara et al. (2006) collected 104 Spanish food samples randomly from local supermarkets all over Spain from 2003 to 2005. The samples encompassed 21 types of food, including milk and dairy products, eggs, sea fish (tuna, sardine, and others), meat and meat products, vegetable oil, and shellfish. A total of 15 BDEs were measured, including for the first time the higher brominated BDE congeners 184, 191,

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196, and 197, in addition to 183 and 209. The highest total median concentration was found in fish, 189 pg/g wwt, followed by oils at 119 pg/g wwt, meats at 76 pg/g wwt, shellfish at 76 pg/g wwt, eggs at 74 pg/g wwt and dairy at 66 pg/g wwt. BDE 209 was the predominant congener in oil and egg samples, at 25 and 37 pg/g wwt, respectively, but it was also found at significant levels in all other food products with medians in dairy products at 4 pg/g wwt, in meats at 11 pg/g wwt, in fish at 5 pg/g fwt and shellfish at 7 pg/g fwt. Otherwise, BDEs 47 and 99 were the predominant congeners in dairy products (11 and 9 pg/g wwt, respectively), meats (17 and 15 pg/g wwt), fish (115 and 15 pg/g wwt), and shellfish (11 and 5 pg/g wwt).

Earlier in 2003, Bocio et al. (2003) evaluated dietary exposure of individuals in Spain to PBDEs. A total of 54 samples were developed as composites of numerous food types, including vegetables, tubers, pulses (peas, beans, and lentils), cereals, fruits, fish, and shellfish, meat and meat products (pork, chicken, beef, lamb) eggs, milk, diary, fats and oils. Samples were analyzed for total homologue groups, so no individual congeners were measured. Because of this, the "total" concentrations here probably should not be compared with totals"from other studies which measured and reported on individual congeners. The highest concentration of total PBDEs was found in oils and fats (0.6 ng/g wwt), followed by fish and shellfish (0.3 ng/g wwt), meat and meat products (0.1 ng/g wwt), and eggs (0.06 ng/g wwt), with essentially none for vegetables and grains. A predominance of the tetra and penta homologues, followed by hexa congeners, was found in the samples.

As described in the section above on fish, the Norwegian Institute of Public Health in Oslo, Norway conducted an intercomparison laboratory study on the measurement of PBTs including PCDD/Fs, PCBs, and PBDEs (Haug et al., 2005). It was noted that fish concentrations were unusually high, totaling 236 ng/g wwt. Results for the other congeners: 28, 47, 99, 100, 153, 154, 183, and 209, were more in line with other values from around with world. Chicken levels of these seven were 0.0007, 0.02, 0.02, 0.007, 0.005, 0.003, 0.003, and 0.07 ng/g wwt, thus totaling 0.14 ng/g wwt, and palm oil levels were 0.005, 0.02, 0.04, 0.02, 0.008, 0.006, 0.007, and 0.39 ng/g wwt, thus totaling 0.49 ng/g wwt. It is noted that BDE 209 was the highest in these chicken and palm oil samples, while it was among the lowest in the fish samples reported earlier.

Irish animal and vegetative food products were surveyed by Tlustos et al. (2005) for the presence of PBDEs. A total of 65 samples, most of them representing pooled samples of 10 or more, were analyzed for PBDEs 17, 28, 47, 47, 66, 71, 77, 85, 99, 100, 119, 126, 138, 153, 154, and 183. Only total concentrations were reported, with no discussion on congener distribution in the food products. Food products included: carcass fat of bovine, avian (including duck), ovine, and porcine; dairy products included butter, cheddar cheese, soft cheese, processed cheese, dairy spread, and yogurt; liver of bovine, avian (chicken and turkey), ovine, and porcine; soup; cereals; fruit; vegetables; vegetable/animal fat; and vegetable oil. Concentrations of total PBDEs ranged narrowly from 0.85 to 1.49 ng/g lwt in all the terrestrial animal food products, and 0.17–0.34 ng/g wwt in the cereals, fruit, vegetables, and vegetable oil.

Levels of 47, 99, 100, 153, and 154 were measured in 33 food items between 2002 and 2004 in Norway (Knutsen et al., 2005). Levels ranged from 0.1–0.5 ng/g lwt for most terrestrial animal food products, but there were high measurements in eggs, 3.85 ng/g lwt, margarine (with 5% fish oil) at 3.08 ng/g lwt, liver paste a 1.17 ng/g lwt, and pork liver at 1.08 ng/g lwt. Levels were much higher in fish, with wet weight concentrations for the majority of fish types above 1.0 ng/g wwt, with a high of 9.73 ng/g wwt for cod liver oil.

Harrad et al. (2004) presented data on BDEs 47, 99, 100, 153, and 154 for indoor and outdoor air, and meat and vegan diets. The purpose of these measurements was to estimate daily exposure to BDEs via inhalation and diet. The total PBDE concentration in the composited samples of vegan and omnivorous diets were 0.15 and 0.18 ng/g dwt, respectively. Similar to air, BDE congeners 47, 99, and 100 were higher in meat diets as compared to vegan diets—BDEs 153 and 154 were very similar. Meat diet samples averaged 0.07, 0.07, and 0.02 ng/g dwt for BDEs 47, 99, and 100, while the analogous concentrations for vegan samples was 0.05, 0.06, and 0.01 ng/g dwt.

Schecter et al. (2006b) looked at the changes in PBDE levels when beef, lamb, and fish were cooked. While they found a reduction in the amount of total BDEs in the samples, they did not provide concentrations before and after cooking, so it cannot be confirmed if, in fact, concentrations decreased. They did show how the percent lipid was reduced by cooking (by fat dripping away), but concentrations are estimated as the mass

of BDEs divided by the mass of lipids (or whole weight), and before and after masses were not provided. Still, the reported amount of total BDEs decreased by over 60% in beef and lamb, by over 50% in catfish, and by smaller amounts (~10%) in trout and salmon. This certainly suggests that concentrations also would have been reduced, and it would have been helpful if the authors had provided concentrations in their article. While their recommendation that exposures consider cooked foods is reasonable, most often exposure via food is calculated by concentrations provided in measurements from food products that are uncooked due to lack of information on concentrations in cooked foods.

In summary, total BDE concentrations in terrestrial food products seem to be lower (less than 1 ng/g wwt) than fish BDE concentrations (1–5 ng/g wwt). Like for fish, a paucity of data on BDE 209 makes it hard to generalize, although when sampled for, it appeared to be present at levels comparable to those for BDE 47 and 99—the congeners generally found at the highest concentration. BDE 99 was typically found at the highest concentration, about twice as high as BDE 47. This contrasts the relationship between these two congeners for fish, where BDE 47 is about twice as high as 99. In very limited sampling, concentrations appear to be much lower in food products of vegetative origin (such as cereals, fruits and vegetables) as compared to terrestrial animal food products. This is to be expected, as these organic PBTs tend to bioaccumulate in fat of animals.

4.8. ASSIGNING EXPOSURE MEDIA CONCENTRATIONS FOR EXPOSURE ASSESSMENT PURPOSES

The challenges with assigning values for individual congeners in exposure media are many:

1) There is no consistent set of measured congeners, as in the case of the 17 toxic dioxin and furan congeners. While the California Air Resources Board studies in air entailed 33 congeners (CARB, 2005, 2006), most studies only measured a handful of congeners. Moreover, the majority of past studies have not measured the deca congener, BDE 209, which remains the primary congener in currently produced and marketed product. The second congener for which limited data exist is BDE 183. The octa formulation of PBDEs contains about 44% hepta congeners, the most of any homologue group, and

BDE 183 is the dominant stable congener within that homologue group. Its presence in an environmental matrix could only have occurred via debromination of higher brominated congeners or because of the presence of the octa formulation. For this reason, BDE 183 is generally considered a primary marker for the possible presence of the octa BDE formulation.

2) There are no statistically designed surveys which would provide a rigorous estimate of exposure media concentrations to which the general population of the United States is exposed. Most studies are non-statistical targeted surveys, conducted within a small geographic area, and driven by a limited budget.

3) Key exposure matrices remain sparsely studied, including indoor air, outdoor soil, and animal food products of terrestrial origin (meat, dairy, and eggs). Food concentration data have only been obtained in the context of a limited number of retail market basket surveys. While these have merit, their coverage is limited: only one in three had any dairy samples, and the one study which did obtain samples from several states (Huwe et al., 2005) did not include measurements for BDE 209.

Therefore, it is not possible to derive media concentrations which are statistically representative of general population exposures. Nonetheless, reasonable assumptions can be made for exposure media concentrations to use in making the exposure estimates (presented in Chapter 5). Decisions needed to be made on which congeners to include in this derivation, and which studies to rely upon. The key congeners that have been measured include the primary congeners of the penta formulation, including BDEs 47, 99, 100, 153, and 154. BDEs 183 and 209 remain of interest for their representation of the octa and deca formulations, as described above. The U.S. EPA PBDE project plan (see http://www.epa.gov/oppt/pbde) identifies BDE 28 as a triBDE of interest, BDE 85 as a tetraBDE of interest, BDE 197 as an octaBDE of interest, and BDE 206 as nonaBDE of interest, so these will be included. Finally, many studies have also included the triBDE 17, the tetraBDE 66, and the hexaBDE 85 in their measurements; so these final congeners will be added, bringing the total to 14 congeners for which profiles will be derived. There are limited concentrations available for other congeners, as displayed in the media-specific tables earlier in this chapter, but only these 14 will be assigned values used in exposure calculations in the next chapter.

Determining final concentrations to represent the general United States population exposure is not straightforward. Some key considerations for this compilation include the following:

1) The studies should come from the United States, or perhaps Canada. Only when North American studies are unavailable will European or other foreign studies be used.

2) Occupational data, while of interest, does not represent general population exposure. For indoor, as well as outdoor, exposures, it should be clear that the data were not taken in the vicinity of known sources, such as recycling facilities, autoshredding facilities, manufacturing facilities, and the like.

3) Studies with a full suite of congeners, and, in particular, BDEs 183 and 209, are preferable to studies which have the limited set of BDEs associated with the penta formulation—BDEs 47, 99, 100, 153, and 154.

4) Attempts to average congener-specific data across studies should be done with caution, if at all. For example, it may be preferable to use data from one geographic area, as long as it is appropriately background and not occupational, if that data has a full congener suite For example, relying on an entire set of urban air data taken by the California Air Resources Board, including the standard BDEs 47, 99, etc, as well as critical BDEs 183 and 209, would be preferable to only using the BDE 183 and 209 data from CARB, while averaging the CARB data on other congeners with data from other urban or rural locations in the United States.

With that as backdrop, Table 4.5 contains the final derived profiles for water, surface soil, indoor dust, indoor and outdoor air, and categories of food products, for use in the exposure calculations in the next chapter. It should be noted that when averages were required that were derived from individual sample data, it was assumed that ND = 0 for these calculations. This was done because often the listed detection limit, such as those from Schecter et al. (2006a), were much higher than the detected quantifications (see Section 4.6 for more detail and examples on the detection limit issue in the Schecter et al., 2006a effort). Following now is a brief justification for each of the media:

1. Drinking Water: Of the three studies found for surface water (none were found for ground water) in the United States, the results from the San Francisco Estuary (Oros et

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al., 2005) were used to represent drinking water exposures. Concentrations of total PBDEs were similar in the three studies, near or below 100 pg/L, but the study of the San Francisco Estuary included BDE 209. The range of total PBDE concentrations ranging from 3 to 513 pg/L, with a mean of 146.2 pg/L. The mean concentration of BDE 209 in 18 measurements was 42.3 pg/L.

2. Surface soil: The only systematic study of surface soil concentrations not associated with an industrial or other contaminated source for United States was from Offenberg et al. (2006), and results from this study will be used in this exercise. The average concentration over the 14 congeners of this assessment was 82 ng/g dry. The only other data on background soils came from Hassanin et al. (2004). It represented European soils and had a much lower total PBDE concentration of only about 1 ng/g dwt.

3. Indoor dust: The Environmental Working Group (Sharp and Lunder, 2004) data set includes samples provided by 10 women who earlier participated in a breast milk sampling program. The concentrations of the congeners track well with other data, as seen in Table 4.2. Because this data originated from 9 different states, it was judged that this might be the most representative data set, although the concentrations may be a bit high. The total of 8,275 ng/g dwt for the 14 congeners compares with 5,811 ng/g dwt from the 17 homes in Washington, DC area (Stapleton, et al., 2005); the 9,271 ng/g dwt from 2 samples from a computer lab in California (CARB, 2005); and the geometric means of three locations (living room, bedroom, and from a household vacuum) within 20 homes in Boston of 13732, 6255, and 4,269 ng/g dwt, ng/g dwt, respectively (Allen et al., 2008).

4. Outdoor air: The CARB data of 84 samples taken in 2004 from 7 monitors on 12 dates from locations in the Bay Area and the South Coast were used for the profile of outside air. While the profile at 158 pg/m^3 might be higher than the profiles measured by Hoh and Hites (2005) or Strandberg et al. (2001), in fact the congener-specific measurements made in these two studies in urban areas are similar to the California measurements. For example, the CARB data included a measurement of 25 pg/m^3 for

BDE 209, while Hoh and Hites (2005) measured 60.1 pg/m³ as an average of 28 samples in 2002/2003. Strandberg et al. (2001) measured 0.30 pg/m³ for BDE 209, but his measurements pertain to 1997-99, which was before the prominent use of the deca formulation. Strandberg et al. (2001) did measure concentrations of 33 and 16 pg/m³ for BDEs 47 and 99, respectively, which compares well with Hoh and Hites (2005), measuring 17 and 7 pg/m³ for these two congeners, and with the CARB data showing 53 and 51 pg/m³ for these two congeners, respectively. Therefore, while possibly a bit high, it would appear that the CARB data captures current urban conditions, and because 84 measurements of 10 of the 14 desired congeners were available (more than any other study), these data were used.

5. Indoor air: The only indoor air measurements in the United States that could be representative of general population exposures were taken in 20 urban residences in Boston, MA (Allen et al., 2007). The geometric mean concentrations were presented for three locations (personal, bedroom, living room), and the average of the 3 geometric means were used as the representative indoor air concentrations. The sum of the geometric mean concentrations for reported congeners for the three locations equaled 605 pg/m³ for the "personal" samples, which were taken near the breathing zone in the bedroom, 392 pg/m³ in the bedroom, and 366 pg/m³ in the living room.

6. Shellfish: The only shellfish data available were the data on clams, oysters, and mussels from the San Francisco Estuary (Oros et al., 2005), so this data was used for the profile. While measurements were made for all 14 congeners, it was stated that non-detects were found for all but BDEs 47, 99, and 100. The wwt total average concentration was 5.7 ng/g wwt, with a range of 2–13 ng/g wwt

7. Finfish: The retail market place data from Schecter et al. (2006a) contained data thought to be most representative for use in this exposure assessment, so it will be used here. A total of 24 samples including tuna, salmon, shark, trout, catfish, and herring, were taken. Measurements and quantifications were made for 12 of the 14 congeners, and the total concentration was 1.17 ng/g wwt. There were some substantially higher

measurements taken from fish in the Great Lakes, including findings by Manchester-Neesvig et al. (2001) on 21 coho and Chinook salmon samples showing an average of 80 ng/g wwt, or the temporal study by Zhu and Hites (2004) showing an average of 120 ng/g wwt for lake trout in Lakes Superior, Michigan, Huron, and Ontario. However, other retail market surveys, such as the one in California, show a range of 0.04 to 4.9 ng/g wwt in fish, and the one in Canada, including 122 fish and shellfish, shows a range of 0.02 ng/g wwt (in shrimp) to 1.6 ng/g wwt (in trout). Other data on farmed fish and fish from abroad, reviewed in Section 4.5, similarly showed concentrations mostly below 10 ng/g wwt and near the value of 1.17 ng/g wwt measured by Schecter et al. (2006a).

8. Beef: The retail market place data from Schecter et al. (2006a) will be used for beef congener data. The total of 0.13 ng/g wwt, the average of 3 beef samples (2 ground, 1 tenderloin), is comparable to the other major retail market basket surveys: Huwe et al.'s (2005) beef samples (n = 11) averaged 0.42 ng/g lwt (roughly 0.08 ng/g wwt) for the five major congeners (47, 99, 100, 153, 154), and Luksemburg et al.'s (2004) beef samples (n = 4) from Northern California averaged 0.15 ng/g wwt total.

9. Pork: The retail market place data from Schecter et al. (2006a) will be used for pork congener data. The total of 0.28 ng/g wwt, the average of 7 pork samples (3 bacon, 1 pork, 2 pork sausage, 1 ground pork), is comparable to the results of Huwe et al. (2005), whose pork samples including 11 bacon and 11 pork fat samples, showed a total of 1.59 ng/g lwt for the five major BDE congeners of 47, 99, 100, 153, and 154. Assuming about a 15% lipid weight of pork, this translates to 0.24 ng/g wwt

10. Poultry: The retail market place data from Schecter et al. (2006a) will be used for poultry congener data. The total of 0.36 ng/g wwt, the average of 3 poultry samples (chicken breast, ground chicken, ground turkey) is comparable to the findings of Luksemburg et al. (2004). Luksemburg et al. (2004) found an average of 0.41 ng/g wwt for 7 poultry samples (4 chicken and 3 turkey samples) and Huwe et al. (2005) found an average of 2.78 ng/g lwt in 22 chicken fat samples. Assuming 15% fat in whole-weight chicken, this translates to about 0.42 ng/g wwt. Interestingly, the duck sample from

Luksemburg at 2.5 ng/g wwt, and the duck sample from Schecter et al. (2006a) at 1.3 ng/g wwt, were the highest of the poultry samples and these were both not included in the displayed average. This high concentration occurred because duck is very fat, listed as 75% lipid in Schecter et al. (2006a). The chicken and turkey samples were listed at between 5 and 11% lipid.

11. Dairy: The retail market place data from Schecter et al. (2006a) will be used for dairy congener data. A total of 15 samples were available, including various cheeses, cow/goat milk, yogurt, ice cream, and infant formula. The average whole-weight concentration was 0.11 ng/g wwt.

12. Eggs: Schecter et al. (2006a) reports on the average of 6 food egg samples. Unlike the meat samples used for this assessment, individual sample data were not presented, so that the presentation of average congener concentrations assuming ND = ½ DL in Schecter et al. (2006a) was used here. The average whole-weight concentration was 0.09 ng/g wwt, the lowest of the terrestrial food products, but it was comparable to the whole-weight concentrations of the other studies, which ranged up to 0.36 ng/g wwt.

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Table 4.1. Congener-specific concentrations of PBDEs in indoor dust in the United States (units in ng/g dry weight)

Congener	Concentration, ng/g dwt	Comment	Citation
BiBDE			
15	11	Mean 10 homes throughout US, but 9 ND, 1 at 109	EWG, 2004
TriBDE			
17	9	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	5.5	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	2.4, 6.4	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	1.4, 0.6, 0.4	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
25	2	Carpet dust (n=2) from computer labs in CA	CARB, 2005
28	21	Mean 17 homes, Wash DC, also listed as congener 33	Stapleton, et al., 2005
	ND (50)	Mean 10 homes throughout US	EWG, 2004
	14.3	Carpet dust (n = 2) from computer labs in CA	CARB, 2005
	3, 20.3	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	25, 14	Mean, geometric mean from homes in Amarillo/Austin, Tex from 2006 (n=20)	Harrad et al. 2008
30	ND	Carpet dust (n=2) from computer labs in CA	CARB, 2005
32	ND	Carpet dust (n=2) from computer labs in CA	CARB, 2005
33	ND (50)	Mean 10 homes throughout United States	EWG, 2004
28/33	16.3, 10.5, 6.4	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
35	ND	Carpet dust (n=2)from computer labs in CA	CARB, 2005
37	ND	Carpet dust (n=2) from computer labs in CA	CARB, 2005
TetraBDE			
47	1,220	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	1857	Mean 10 homes throughout US	EWG, 2004
	ND (400) – 9,860	Range of 89 homes sampled in Cape Cod, MA; 45% detected	Rudel et al., 2003
	430 (230-3,000)	Median; range from 10 homes in Atlanta	Sjodin et al., 2004
	456	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	364, 1621	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	1865, 837, 338	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
	669, 14613	Median, max, from 11 homes in the Boston, MA area	Wu et al. 2007
	810, 470	Mean, geometric mean from homes in Amarillo/Austin, Tex from 2006 (n=20)	Harrad et al. 2008
49	ND	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	29.6, 23.6, 12.4	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
66	28.5	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	21	Mean 10 homes throughout US, but 8 ND, 2 at about 100	EWG, 2004

	20.3	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	5.5, 26.1	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	17.2, 15.3, 6.0	Geo. mean of 3 loc/home from 20 homes in Boston, MA area: liv rm, bdrm, and vac	Allen et al. 2008
	ND, 293.0	Median, max, from 11 homes in the Boston, MA area	Wu et al. 2007
71	ND	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	ND	Carpet dust (n=2) from computer labs in CA	CARB, 2005
75	ND	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	9.3, 5.3, 3.6	Geo. mean of 3 loc/home from 20 homes in Boston, MA area: liv rm, bdrm, and vac	Allen et al. 2008
77	0.8	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	0.1, 0.1	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
PentaBDE			
85	83	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	100	Mean 10 homes throughout United States, but 8 ND, two at 453 and 544	EWG, 2004
	51	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	28.5, 96.4	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	ND, 787	Median, max, from 11 homes in the Boston, MA area	Wu et al. 2007
85/155	124.0, 51.8, 19.2	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
99	1,700	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	ND (400) to 22,500	Range of 89 homes sampled in Cape Cod, MA; 55% detected	Rudel et al. 2003
	2,352	Mean 10 homes throughout United States	EWG, 2004
	880 (69–3,700)	Median; range from 10 homes in Atlanta	Sjodin et al., 2004.
	776	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	612, 2,295	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	2460, 1170, 536	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
	1014, 14979	Median, max, from 11 homes in the Boston, MA area	Wu et al. 2007
	1400, 840	Mean, geometric mean from homes in Amarillo/Austin, Tex from 2006 (n=20)	Harrad et al. 2008
100	274	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	ND (300) to 3,400	Range of 89 homes sampled in Cape Cod, MA; 20% detected	Rudel et al., 2003
	911	Mean 10 homes throughout United States	EWG, 2004
	150 (<15–660)	Median; range from 10 homes in Atlanta	Sjodin, et al., 2004
	135	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	103, 429	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	436, 204, 77	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
	174, 2776	Median, max, from 11 homes in the Boston, MA area	Wu et al. 2007
	240, 160	Mean, geometric mean from homes in Amarillo/Austin, Tex from 2006 (n=20)	Harrad et al. 2008

116	229	Carpet dust (n=2) from computer labs in CA	CARB, 2005
118	70	Carpet dust (n=2) from computer labs in CA	CARB, 2005
119	12	Carpet dust (n=2) from computer labs in CA	CARB, 2005
126	ND	Carpet dust (n=2) from computer labs in CA	CARB, 2005
HexaBDE			
138	17	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	181	Mean 10 homes throughout US, but 8 ND, 1 at 1,668	EWG, 2004
	15	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	8, 23	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	20.9, 12.1, 5.2	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
	ND, 77	Median, ng/g, from 11 homes in the Boston, MA area	Wu et al. 2007
153	181	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	243	Mean 10 homes throughout US, but 7 ND, 1 at 1,510	EWG, 2004
	140 (5–650)		
	144	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	61, 199	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	234.4, 124.2, 47.0	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
	107, 563	Median, max, from 11 homes in the Boston, MA area	Wu et al. 2007
	240, 120	Mean, geometric mean from homes in Amarillo/Austin, Tex from 2006 (n=20)	Harrad et al. 2008
154	156	Mean 17 homes, Wash DC	Stapleton et al. 2005
	156	Mean 10 homes throughout US, but 8 ND, 1 at 1,050	EWG, 2004
	77 (<7–260)	Median; range from 10 homes in Atlanta	Sjodin, et al., 2004
	95	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	54, 189	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	182.8, 94.4, 35.0	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
	93, 455	Median, ng/g, from 11 homes in the Boston, MA area	Wu et al. 2007
	240, 100	Mean, geometric mean from homes in Amarillo/Austin. Tex from 2006 (n=20)	Harrad et al. 2008
155	8	Carpet dust (n=2) from computer labs in CA	CARB, 2005
156	ND	Mean 17 homes, Wash DC	Stapleton, et al., 2005
166	ND	Carpet dust (n=2) from computer labs in CA	CARB, 2005
HeptaBDE			
181	ND	Carpet dust (n=2) from computer labs in CA	CARB, 2005
183	31	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	60	Mean 10 homes throughout United States, but 9 ND, 1 at 604	EWG, 2004
	73 (<8–4,000)	Median; range from 10 homes in Atlanta	Sjodin et al., 2004
	130	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	18.6, 19.3	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	27.9, 32.9, 15.1	Geo. mean of 3 loc/home from 20 homes in	Allen et al. 2008

		Boston, MA area; liv rm, bdrm, and vac	
	28, 16	Mean, geometric mean from homes in Amarillo/Austin, Tex from 2006 (n=17)	Harrad et al. 2008
184	ND	Mean 17 homes, Wash DC	Stapleton, et al., 2005
190	5	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	24	Carpet dust (n=2) from computer labs in CA	CARB, 2005
191	ND	Mean 17 homes, Wash DC	Stapleton, et al., 2005
OctaBDE			
196	15	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	3.6, 2.6, 3.9	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
197	17	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	2.7, 3.3, 5.6	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
203	109	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	3.6, 3.6, 4.9	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
NonaBDE			
206	51	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	76.3, 48.1, 40.5	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm. bdrm, and vac	Allen et al. 2008
207	30	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	45.9, 25.3, 26.6	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
208	35	Mean 17 homes, Wash DC	Stapleton, et al., 2005
	35.6, 17.5, 29.4	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
DecaBDE			
209	2,090	Mean 17 homes. Wash DC	Stapleton, et al., 2005
	2,394	Mean 10 homes throughout United States	EWG, 2004
	2000 (120-21,000)	Median; range from 10 homes in Atlanta	Sjodin et al., 2004
	7500	Carpet dust (n=2) from computer labs in CA	CARB, 2005
	665, 8567	Median, mean (n=9) from vacuum samples in Dallas, TX, in 2004	Schecter et al., 2005
	4502, 1703, 1811	Geo. mean of 3 loc/home from 20 homes in Boston, MA area; liv rm, bdrm, and vac	Allen et al. 2008
	ND, 9020	Median, ng/g, from 11 homes in the Boston, MA area	Wu et al. 2007
	1600, 1300	Mean, geometric mean from homes in Amarillo/Austin, Tex from 2006 (n=17)	Harrad et al. 2008

Table 4.2. Outdoor and indoor congener-specific air concentrations of PBDEs in the United States (units in pg/m³)

Congener	Concentration, pg/m³	Comment	Citation
TriBDE			
17	3.6	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	46	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	1.5-5.6	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	35-106	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	0.3-5.8	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	1.7	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
	7.6, 8.1, 7.0	Geo. mean of 20 homes, 3 loc/home in Boston, MA area; "personal", bedroom, and living room	Allen et al. (2007);
25	2.3	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	29	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	0.4-4.7	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	16-103	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	0.5-3.1	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
28	99	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	2.9-17.8	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	102-372	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	0.4-7.4	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	3.0	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
28 + 33	4.0	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	29.6, 27.3, 25.4	Geo. mean of 20 homes, 3 loc/home in Boston, MA area; "personal", bedroom, and living room	Allen et al. (2007)
30	0.2	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND	Range, n=12 (3 days, 4 samplers) outdoors	CARB, 2005

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		surrounding electronics recycling facility in CA	
	ND	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND-0.9	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
32	ND	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND-0.9	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
33	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND-1.4	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	0.7-35	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
35	0.4	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND-1.1	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND-18.1	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
TetraBDE			
37	2.3	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	1.3-8.4	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND-468	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	0.6-2.4	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
47	6.2	Michigan (n=35), 2002/3	Hoh & Hites, 2005
	17.4	Chicago (n=28), 2002/3	Hoh & Hites, 2005
	7.0	Indiana (n=38), 2002/3	Hoh & Hites, 2005
	9.2	Arkansas (n=30), 2002/3	Hoh & Hites, 2005
	6.9	Louisiana (n=26), 2002/3	Hoh & Hites, 2005
	5.0	3 Rural/remote sites in MI, NY (n=36), 97-99	Strandberg et al 2001

	33	Chicago, n=12, 97-99	Strandberg et al 2001
	175 (671)	Lewes, Del Marva, MD (n=95) geom. mean (max), gaseous phase only	Goel et al., 2006
	9.7 (26)	Horn Point, Del Marva, MD (n=98) geom. mean (max), gaseous phase only	Goel et al., 2006
	17 (52)	Dover, Del Marva, MD (n=47) geom. mean (max), gaseous phase only	Goel et al., 2006
	34.5	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	1,065	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	30-128	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	604-2,850	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; avg = 1,772	CARB, 2005
	30-88	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	53.0	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
	226.8, 157.9, 145.1	Geo. mean of 20 homes, 3 loc/home in Boston, MA area; "personal", bedroom, and living room	Allen et al. (2007)
49	1.2	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	59	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	4.6-36.7	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	449-2860	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; avg = 1764	CARB, 2005
	2.7-10.5	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
49	9.1, 6.0, 7.2	Geo. mean of 20 homes, 3 loc/home in Boston, MA area; "personal", bedroom, and living room	Allen et al. (2007)
65	2.0	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
66	1.6	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	29	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	2.9-10	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	103-750	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	2.3-5.1	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	3.7, 3.5, 3.5	Geo. mean of 20 homes, 3 loc/home in Boston, MA area; "personal", bedroom, and living room	Allen, et al. (2007)
71	1.7	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with	CARB, 2005

		computers off, in CA	
	ND	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
75	0.4	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	6.6	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND-194	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	0.5-2.4	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
77	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	22.1-75.5	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND-2.4	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	2.0	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
PentaBDE			
85	1.1	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	13	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND-6	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	87-284	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	2.1-9.0	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	2.3	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
	3.8, 2.7, 2.5	Geo. mean of 20 homes, 3 loc/home in Boston, MA area; "personal", bedroom, and living room	Allen, et al. (2007)
99	5.1	Michigan (n=35), 2002/3	Hoh & Hites, 2005
	7.4	Chicago (n=28), 2002/3	Hoh & Hites, 2005
	5.1	Indiana (n=38), 2002/3	Hoh & Hites, 2005
	5.4	Arkansas (n=30), 2002/3	Hoh & Hites, 2005
	3.0	Louisiana (n=26), 2002/3	Hoh & Hites, 2005
	3.4	3 Rural/remote sites in MI, NY (n=36), 97-99	Strandberg et al 2001
	16	Chicago, n=12, 97-99	Strandberg et al 2001
	26 (178)	Lewes, Del Marva, MD (n=95) geom. mean (max), gaseous phase only	Goel et al., 2006

	5.3 (26)	Horn Point, Del Marva, MD (n=98) geom. mean (max), gaseous phase only	Goel et al., 2006
	7.7 (17)	Dover, Del Marva, MD (n=47) geom. mean (max), gaseous phase only	Goel et al., 2006
	12.5	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	239	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	9-85	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	402-3210	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; avg=1,771	CARB, 2005
	21-111	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	51.0	N=84 (12 sampling dates, 7 monitors): average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
	110.8, 66.9, 60.3	Geo. mean of 20 homes, 3 loc/home in Boston, MA area: "personal", bedroom, and living room	Allen et al. (2007)
100	1.1	Michigan (n=35), 2002/3	Hoh & Hites, 2005
	1.8	Chicago (n=28), 2002/3	Hoh & Hites, 2005
	1.0	Indiana (n=38), 2002/3	Hoh & Hites, 2005
	1.1	Arkansas (n=30), 2002/3	Hoh & Hites, 2005
	0.7	Louisiana (n=26), 2002/3	Hoh & Hites, 2005
	0.5	3 Rural/remote sites in MI, NY (n=36), 97-99	Strandberg et al 2001
	2.0	Chicago, n=12, 97-99	Strandberg et al 2001
	17 (73)	Lewes, Del Marva, MD (n=95) geom. mean (max), gaseous phase only	Goel et al., 2006
	5.4 (5.4)	Horn Point, Del Marva, MD (n=98) geom. mean (max), gaseous phase only	Goel et al., 2006
	5.3 (5.3)	Dover, Del Marva, MD (n=47) geom. mean (max), gaseous phase only	Goel et al., 2006
	4.8	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	100	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	3-17	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	69-448	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	5.2-21.5	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	13.0	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
	22.2, 14.4, 12.0	Geo. mean of 20 homes, 3 loc/home in Boston, MA area: "personal," bedroom, and living room	Allen et al. (2007)
116	0.2	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	14.7	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005

	ND	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
118	0.3	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND-5.3	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	67-343	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	1.1-3.9	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
119	1.4	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
126	ND	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
HexaBDE			
138	0.9	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND-6.8	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	95-346	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	1.3-3.9	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
153	0.20	3 Rural/remote sites in MI, NY (n=36), 97-99	Strandberg et al., 2001
	0.53	Chicago, n=12, 97-99	Strandberg et al., 2001
	2.0	Outdoors at UC/Davis, 2004; average of 2 days	CARB, 2005

		of filter/PUF and filter/XAD samplers	
	11	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	3-150	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	1,120-8,900	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; avg = 5,623	CARB, 2005
	11.3-33.2	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	3.9	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
	8.6, 4.0, 3.5	Geo. mean of 20 homes, 3 loc/home in Boston, MA area; "personal", bedroom, and living room	Allen et al. (2007)
154	0.12	3 Rural/remote sites in MI, NY (n=36), 97-99	Strandberg et al., 2001
	041	Chicago, n=12, 97-99	Strandberg et al., 2001
	ND	For Lewes, Horn Point, Del Marva, MD (n = 240), gaseous phase only	Goel et al., 2006
	2.8	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	13	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	1.5-86.7	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	1230-5260	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; avg = 3455	CARB, 2005
	3.8-36.9	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	4.0	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
	9.1, 6.1, 5.2	Geo. mean of 20 homes, 3 loc/home in Boston, MA area; "personal," bedroom, and living room	Allen et al. (2007)
155	ND	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND-0.6	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
166	ND	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND-2.5	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	35-463	Range, n=6 (3 days, 2 samplers) indoors in	CARB, 2005

		electronics recycling facility in CA	
	ND	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
HeptaBDE			
181	ND	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	ND	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
183	1.4	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	6-456	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	5610-36700	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; avg = 23813	CARB, 2005
	4.0-32.6	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
	1.4	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
190	<0.06 (DL)	3 Rural/remote sites in MI, NY (n=36), 97-99	Strandberg et al., 2001
	<0.06 (DL)	Chicago, n=12, 97-99	Strandberg et al., 2001
	ND	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
	ND-16	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA	CARB, 2005
	288-1300	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA	CARB, 2005
	ND-3.8	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA	CARB, 2005
NonaBDE			
203	ND	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
	ND	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
DecaBDE			
209	1.4	Michigan (n=35), 2002/3	Hoh & Hites, 2005
	60.1	Chicago (n=28), 2002/3	Hoh & Hites, 2005
	2.2	Indiana (n=38), 2002/3	Hoh & Hites, 2005

9.0	Arkansas (n=30), 2002/3	Hoh & Hites, 2005
2.6	Louisiana (n=26), 2002/3	Hoh & Hites, 2005
<0.10 (DL)	3 Rural/remote sites in MI, NY (n=36), 97-99	Strandberg et al., 2001
0.30	Chicago, n=12, 97-99	Strandberg et al., 2001
10.6	Outdoors at UC/Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB, 2005
58	Indoors in computer facility, average of 2 days, 8 measurements, 6 with computers on, 2 with computers off, in CA	CARB, 2005
140-11400	Range, n=12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA; mean = 2764	CARB, 2005
79700-833000	Range, n=6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; avg = 423,466	CARB, 2005
123-1940	Range, n=9 (3 days, 3 samplers) outdoors in auto shredder facility in CA; avg = 2,403	CARB, 2005
25.0	N=84 (12 sampling dates, 7 monitors); average for 2004 in Bay Area and South Coast of CA	CADAMP, 2006
174, 95, 94	Geo. mean of 20 homes, 3 loc/home in Boston, MA area; "personal," bedroom, and living room	Allen et al. (2007)

Table 4.3. Congener-specific fish concentrations of PBDEs for fish caught in the United States (units in ng/g wet weight, wwt, or lipid weight, lwt, in parenthesis if available)

Congener	Concentration, ng/g wwt (or lwt)	Comment	Citation
TriBDE			
17	0.011	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al. 2006
	0.10 (0.77 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
28	0.01	Average of 2 farm salmon farm composited from Maine; eyeball estimate from graph	Shaw et al., 2005
	0.026	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al., 2006
	1.67 (10.21 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	1.2 (5.4 lwt)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al., 2005
TetraBDE			
47	0.60	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al., 2006
	3.6	N=17 locations in SF Estuary; bivalve (clam, oyster, mussel) average	Oros et al., 2005
	0.43	Average of 2 farm salmon farm composited from Maine; eyeball estimate from graph	Shaw et al., 2005
	52.1	Average of 21 salmon samples from Lake Michigan	Manchester-Neesvig et al., 2001
	63.72 (391.12 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	2–110	Time/spatial trend study for lake trout/smelt in Great Lakes; range for last samples, ~ 2003	Batterman et al., 2007
	46.0 (488.3 lwt)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al., 2005
	1.5, 22, 84%	N=63; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
49	4.78 (35.35 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	ND, 1.3, 33%	N=60; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
66	1.82 (11.29 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	0.021	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al., 2006
	1.7	Average of 21 salmon samples from Lake Michigan	Manchester-Neesvig, et al., 2001
	0.3 (1.8 lwt)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al., 2005
	ND, 1.0, 19%	N=27; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
71	ND	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	ND, <0.5, 3%	N=63; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006

77	0.001	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring; mostly na or nd, with one 3.6 herring	Schecter et al 2006
PentaBDE			
85	0.67 (4.31 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	0.004	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring; mostly na or nd; one catfish at 41.6	Schecter et al 2006
	0.03	Average of 2 farm salmon farm composited from Maine; eyeball estimate from graph	Shaw et al., 2005
	0.1 (0.9 lwt)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al 2005
88	0.03	Average of 2 farm salmon farm composited from Maine; eyeball estimate from graph	Shaw et al., 2005
99	22.96 (142.20 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	0.4 - 15	Time/spatial trend study for trout/smelt in Great Lakes; range for last sampled, ~2003	Batterman et al., 2007
	0.17	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al., 2006
	0.10	Average of 2 farm salmon farm composited from Maine; eyeball estimate from graph	Shaw et al., 2005
	9.3	Average of 21 salmon samples from Lake Michigan	Manchester-Neesvig, et al., 2001
	1.2	N=17 locations in SF Estuary; bivalve (clam, oyster, mussel) average	Oros et al 2005
	5.5 (23.1 lwt)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al 2005
	ND, 17.0, 38%	N=63; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
100	17.50 (109.51 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	0.5 - 15	Time/spatial trend study for trout/smelt in Great Lakes; range for last sampled, ~2003	Batterman et al., 2007
	0.13	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al 2006
	0.08	Average of 2 farm salmon farm composited from Maine; eyeball estimate from graph	Shaw et al., 2005
	9.7	Average of 21 salmon samples from Lake Michigan	Manchester-Neesvig, et al., 2001
	0.9	N=17 locations in SF Estuary; bivalve (clam, oyster, mussel) average	Oros et al 2005
	14.0 (55.4)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al., 2005
	1.0, 5.1, 51%	N=63; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
HexaBDE			
138	ND	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	0.001	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring; mostly nd except 2 catfish at 5.1 and 7.9	Schecter et al 2006
	0.05	Average of 2 farm salmon farm composited	Shaw et al., 2005

		from Maine; eyeball estimate from graph	
	<0.001	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrep et al 2005
	ND, <0.9, 2%	N=63; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
153	0.021	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring;	Schecter et al 2006
	4.43 (27.48 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	0.1 – 3.6	Time/spatial trend study for trout/smelt in Great Lakes; range for last sampled, ~2003	Batterman et al. (2007)
	3.8 (17.0 lwt)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al 2005
	ND, 1.1, 40%	N=63; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
154	0.049	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al 2006
	7.63 (46.84 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	9.2 (2.6 lwt)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al 2005
	0.48, 0.88, 49%	N=63; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
HeptaBDE			
183	0.002	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al 2006
	0.13 (0.73 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
	1.1 (3.1 l2t)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al 2005
	ND, <0.9, 3%	N=63; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
184	ND, <0.9, 2%	N=60; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
190	ND	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu & Hites, 2004
191	ND, <0.9, 0%	N=60; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006
NonaBDE			
203	1.3 (4.3 lwt)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al 2005
DecaBDE			
209	0.092	N=24; consumer fish including tuna, salmon, shark, trout, catfish, herring; majority nd but one catfish at 1269	Schecter et al 2006
	0.5 (96.6 lwt)	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al 2005
	ND, <5.3, 6%	N=63; median, mean, frequency detection in Wash State survey in rivers and lakes	Johnson et al., 2006

Table 4.4. Congener-specific concentrations of PBDEs in food originating from the United States (concentrations in ng/g wwt or specifically identified as ng/g lwt, if that was how reported by the author and conversion to ng/g wwt was not possible).

Congener	Concentration, ng/g wwt or lwt	Food type; comment	Citation
TriBDE			
17	0.0008	N=18 meat (lipid = 26.3%) including pork, chicken, beef; dominated by NDs at DL = 0.7	Schecter et al., 2006
	0.29	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al., 2006
17/25	0.008	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	0.008	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
28	0.005	N=18 meat (lipid = 26.3%) including pork, chicken, beef; dominated by one beef at 59.7	Schecter et al., 2006
	0.79	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al., 2006
33	0.017	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	0.028	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
28/33	0.004 lwt	Market basket hamburger (n=11), 10 locations around the country, sampled in 2004	Huwe & Larsen 2005
	0.004 lwt	Market basket bacon (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen 2005
	0.003 lwt	Market basket chicken fat (n=22), 10 locations around the country, sampled in 2005	Huwe & Larsen 2005
	0.007 lwt	Market basket pork fat (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen 2005
	0.002 lwt	Market basket beef fat (n=10), 10 locations around the country, sampled in 2004	Huwe & Larsen 2005
TetraBDE			
47	2.26	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	6.87	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
	0.093	N=18 meat (lipid = 26.3%) including pork, chicken, beef;	Schecter et al., 2006
	0.032	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al., 2006
	0.18 lwt	Market basket hamburger (n=11), 10 locations around the country, sampled in 2004	Huwe & Larsen 2005
	0.23 lwt	Market basket bacon (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen 2005
	0.81 lwt	Market basket chicken fat (n=22), 10 locations around the country, sampled in 2005	Huwe & Larsen 2005
	1.07 lwt	Market basket pork fat (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen 2005
	0.07 lwt	Market basket beef fat (n=10), 10 locations around the country, sampled in 2004	Huwe & Larsen 2005

66	0.00	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	0.018	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
	1.19	N=18 meat (lipid = 26.3%) including pork, chicken, beef;	Schecter et al., 2006
	0.61	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al., 2006
77	0.83	N=18 meat (lipid = 26.3%) including pork, chicken, beef; mostly ND or NA	Schecter et al., 2006
	0.10	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream; all nd or na	Schecter et al., 2006
PentaBDE			
85	0.11	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	0.52	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
	4.93	N=18 meat (lipid = 26.3%) including pork, chicken, beef;	Schecter et al., 2006
	1.08	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream; most nd or na except one cheese at 5.52	Schecter et al., 2006
	0.022 lwt	Market basket hamburger (n=11), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
	0.023 lwt	Market basket bacon (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.047 lwt	Market basket chicken fat (n=22), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.034 lwt	Market basket pork fat (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.014 lwt	Market basket beef fat (n=10), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
99	3.11	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	10.87	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
	0.16	N=18 meat (lipid = 26.3%) including pork, chicken, beef;	Schecter et al., 2006
	0.028	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al., 2006
	0.26 lwt	Market basket hamburger (n=11), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
	0.30 lwt	Market basket bacon (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	1.38 lwt	Market basket chicken fat (n=22), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	1.04 lwt	Market basket pork fat (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.11 lwt	Market basket beef fat (n=10), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
100	0.45	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	1.47	4 chicken fat (90% lipid) samples from a	Huwe, et al., 2003

		contaminated production site	
	0.023	N=18 meat (lipid = 26.3%) including pork, chicken, beef;	Schecter et al., 2006
	0.005	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al., 2006
	0.042 lwt	Market basket hamburger (n=11), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
	0.041 lwt	Market basket bacon (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.28 lwt	Market basket chicken fat (n=22), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.18 lwt	Market basket pork fat (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.02 ng/g lwt	Market basket beef fat (n=10), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
HexaBDE			
138	0.02	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	0.16	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
	0.002	N=18 meat (lipid = 26.3%) including pork, chicken, beef;	Schecter et al., 2006
	0.0003	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream; most nd or na with high DL	Schecter et al., 2006
140	0.003	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	0.015	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
153	060	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	2.90	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
	0.021	N=18 meat (lipid = 26.3%) including pork, chicken, beef;	Schecter et al., 2006
	0.004	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al., 2006
	0.10 lwt	Market basket hamburger (n=11), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
	0.078 lwt	Market basket bacon (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.22 lwt	Market basket chicken fat (n=22), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.12 lwt	Market basket pork fat (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.019 lwt	Market basket beef fat (n=10), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
154	0.18	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	0.55	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
	0.014	N=18 meat (lipid = 26.3%) including pork, chicken, beef;	Schecter et al., 2006
	0.002	N=15 dairy (lipid = 10.2%) including cheese,	Schecter et al., 2006

		milk, infant formula, yogurt, ice cream	
	0.029 lwt	Market basket hamburger (n=11), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
	0.042 lwt	Market basket bacon (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.088 lwt	Market basket chicken fat (n=22), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.087 lwt	Market basket pork fat (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.011 lwt	Market basket beef fat (n=10), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
HeptaBDE			
183	0.19	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	0.34	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
	10.1	N=18 meat (lipid = 26.3%) including pork, chicken, beef;	Schecter et al., 2006
	1.86	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al., 2006
	0.029 lwt	Market basket hamburger (n=11), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
	0.12 lwt	Market basket bacon (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.13 lwt	Market basket chicken fat (n=22), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.084 lwt	Market basket pork fat (n=11), 10 locations around the country, sampled in 2005	Huwe & Larsen, 2005
	0.012 lwt	Market basket beef fat (n=10), 10 locations around the country, sampled in 2004	Huwe & Larsen, 2005
DecaBDE			
209	1.24	9 chicken fat (90% lipid) samples from three production non-contaminated sites	Huwe, et al., 2003
	0.72	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe, et al., 2003
	0.053	N=18 meat (lipid = 26.3%) including pork, chicken, beef; dominated by 1 turkey at 245	Schecter et al., 2006
	0.041	N=15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream; dominated by 1 sample at 481; others at < 20	Schecter et al., 2006

Table 4.5. Exposure media concentrations (note: --- means no data available; ND means data available but not detected.)

Exposure Media	17	28	47	66	85	99	100	138	153	154	183	197	206	209	Total	Reference; n
Water, pg/l	6.6	3.3	42.7	2.2	1.3	27.6	7.2	0.3	3.9	2.9	4.4	0.1	1.4	42.3	146.1	Oros et al. (2005); n=33[a]
Surface soil, ng/g dtwt	---	---	1.9	<0.1	<0.1	3.6	0.4	---	5.7	4.8	37.4	12.4	0.8	15.3	82.3	Offenberg et al. (2006)
Indoor dust, ng/g	---	ND	1857	21	100	2352	911	181	243	156	60	---	---	2394	8275	EWG (2004); mean 10 homes
Outdoor air, pg/m³	2	3	53	---	2	51	13	---	4	4	1	---	---	25	158	CARB (2005); n=84
Indoor air, pg/m³	8	27	177	4	3	79	16	---	5	7	---	---	---	121	447	Allen et al. (2006); Webster (2006); n=8[b]
Shellfish, ng/g wwt	ND	ND	3.6	ND	ND	1.2	0.9	ND	ND	ND	ND	ND	ND	ND	5.7	Oros et al. (2005); n=17[c]
Finfish, ng/g wwt	0.01	0.03	0.60	0.02	0.004	0.17	0.13	0.001	0.02	0.05	0.002	---	---	0.09	1.17	Schecter et al. (2006); n=24
Beef, ng/g wwt	0.0005	0.02	0.05	0.0002	0.0006	0.04	0.006	0.0001	0.006	0.004	0.001	---	---	0.003	0.13	Schecter et al. (2006); n=3
Pork, ng/g wwt	0.0001	ND	0.08	ND	0.005	0.12	0.015	0.001	0.02	0.01	0.009	---	---	0.02	0.28	Schecter et al. (2006); n=6
Poultry, ng/g wwt	0.0001	0.0002	0.06	0.0003	ND	0.12	0.03	0.002	0.02	001	002	---	---	0.12	0.36	Schecter et al. (2006); n=3
Dairy, ng/g wwt	<.0001	0.0002	0.03	0.0003	0.0008	0.03	0.005	<0.0001	0.004	0.002	0.002	---	---	0.04	0.11	Schecter et al. (2006); n=15
Eggs, ng/g wwt	0.0001	0.0002	0.02	0.0002	0.002	0.04	0.006	0.0001	0.004	0.003	0.0001	---	---	0.01	0.09	Schecter et al. (2006); n=6

a: for Oros et al. (2005), concentrations reported as "Q" meaning "detected, but not reportable because outside QA limits" not counted in averaging; "bdl" meaning below detection limit counted as 0; b: Allen et al. (2006) and Webster (2006) sampled 3 locations within 20 urban residences, and presented geometric means for the three locations over all 20 homes. Results presented are the average of the 3 geometric mean values; c: Oros et al. (2005) claims that only BDEs 47, 99, and 100 were detected in sampling of clams, oysters, and mussels - that others were measured but not detected - from SF Estuary.

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Chapter 5 HUMAN EXPOSURE

5.1. INTRODUCTION

The rise in PBDEs in breast milk in the United States throughout the 1990s in the 2000s, coupled with the finding that American, breast milk concentrations exceed those of European women by factors of 10 or more, has served to focus attention on United States exposures to PBDEs. The first section of this chapter reviews the data on body burdens of PBDEs, with a focus on data from the United States. Robust data are available on blood and breast milk, while limited data are available on other matrices—including adipose tissue and liver. The section on body burdens concludes with assignment of representative PBDE congener background profiles in blood and mother's milk. The next section reviews estimates of dose of PBDEs currently available in the literature. This is followed by development of an estimate of background dose using the environmental media concentration profiles developed in Chapter 4, in combination with exposure contact rates. The dose estimate made here is compared to the literature estimates of dose. The next section of the chapter attempts to use a simple pharmacokinetic framework to see if the dose estimates can explain body burdens of the individual congeners. Figure 5-1 depicts this approach. In combination with mother's milk concentrations, the same PK model was used to evaluate the impact of breast-feeding on the PBDE body burden of infants. The chapter concludes with a series of findings from this examination of United States exposures to PBDEs.

5.2. BODY BURDEN DATA

Data from around the world on PBDE body burdens, with an emphasis on data from the United States are reviewed in this section. Subsections on blood and breast milk conclude with a table displaying individual congener data; data on adipose and other tissues are too sparse to warrant a table of raw data. This section on body burden data concludes with a table suggesting representative profiles in blood and mother's milk for selected congeners.

5.2.1. Blood Data

Twelve studies that contained data on PBDEs in blood in the United States were located. Table 5-1 shows congener-specific data from these.

The most statistically rigorous and expansive study of background exposures to PBDEs is a recent analysis of 2003/4 NHANES data by Sjodin et al. (2008). Unfortunately, BDE 209 was not measured in this NHANES study. A total of 2,040 serum samples from individuals 12 years of age and older were analyzed for 10 BDE congeners, including BDEs 17, 28, 47, 66, 85, 99, 100, 153, 154, and 183. The geometric mean concentrations over the entire population were, in ng/g lipid weight (lwt), descending order: 20.5 for BDE 47, 5.7 for BDE 153, 5.0 for BDE 99, 3.9 for BDE 100, and 1.2 for BDE 28. Geometric means were not provided for the other BDE congeners because they were detected at less than 60%, including the low frequencies of 5, 15, 21, and 23% for BDEs 17, 183, 66, and 85, respectively. The sum of these geometric means was 36.3 ng/g lwt. The 95th percentile for the sum of PBDEs was 291 ng/g lwt, and the maximum found was 3,680 ng/g lwt, with BDE 47 at 2,350 ng/g lwt for this individual. A statistically significant relationship between age and concentration was found for BDEs 28, 47, 99, 100, and 153. Specifically, the highest concentrations were found in the age group 12–19 years, with lower concentrations for 20-39, and 40-59, but then a rise for the category >60 years. For example, the geometric mean concentration of BDE 47 for these four age categories, respectively, was 28.2, 21.5, and 17.7, and then a rise to 19.7 ng/g lwt. Although not statistically significant, the geometric mean concentrations for males were higher than females for BDEs 47, 99, 100, 153, and 154. Although BDE 47 was found most frequently and at the highest concentrations for the survey as a whole, 10.5% of participants had BDE 153 concentrations that were higher than BDE 47. Race was identified as a statistically significant factor for BDE 99; the geometric mean for non-hispanic blacks was the highest at 6.2 ng/g lwt, Mexican Americans were at 5.9 ng/g lwt, and non-hispanic whites were at 4.7 ng/g lwt.

Other than NHANES, the most comprehensive study of PBDEs in blood was conducted by Schecter et al. (2005). Although limited geographically, this study contained two pooled samples of 100 individuals each and 39 samples of blood from individuals in Mississippi and New York. Also, the study included an archived blood

sample from 1973, which was from a pool of 100 individuals from Dallas, Texas. All samples were analyzed for 13 BDEs including 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209. All congeners were non-detects for the 1973 sample. The 2 pooled samples collected in 2003, one serum and one whole blood, both also n = 100 as noted, were from discarded samples from the University of Texas Southwestern Medical Center in Dallas. The 39 individuals sampled in 2003 included 29 from Mississippi and 10 from New York. The results from the three groups of samples from 2003 were fairly similar: total concentrations of the 13 congeners were 61.8 ng/g lwt for the serum pool, 79.7 ng/g lwt for the whole blood pool, and 52.6 ng/g lwt as the mean for the 39 individuals. The congener-specific trends were similar as well: BDE 47 dominated the profile by encompassing between 44 and 53% of the total, with BDE 99 and 153 comprising about the same amounts – BDE 99 was between 14 and 21% and 153 was between 11 and 20%. From the individual samples, an interesting trend was that women had higher concentrations than men: the range and mean of total BDE in 22 men were 4.6-192.8 ng/g lwt and 25.1 ng/g lwt., and for 17 women, the range and mean of total BDE were 5.6-365.5 ng/g lwt and 74.1 ng/g lwt. Although women had a higher level of PBDEs in their blood than men in this study, Schecter et al (2005) stated that their results were not statistically significant. BDE 209 was found at low levels in the pooled blood, 1.4 ng/g lwt, and in the individual samples, it averaged 1.7 ng/g lwt with non-detects in 19 of 39 individual samples.

Sjodin et al. (2004a) conducted a more rigorous temporal evaluation by collecting samples that represented different time frames from the mid-1980s until the early 2000s. Specifically, serum pools were collected in the southeastern United States from a blood bank in Memphis, TN, representing years 1985-1997, and 2002, and serum pools were collected in Seattle, WA, representing years 1999-2002. The pooled samples were clustered to represent the following time periods: 1985-89 (n=9), 1990-94 (n=14), 1995-99 (n=10), and 2000-02 (n=7), and the samples were measured for BDEs 47, 85, 99, 100, 153, and 154. A clear trend was seen with total concentrations rising from 9.6 ng/g lwt in the 1985-89 time frame, to 48 ng/g lwt in 1990-94, 71 ng/g lwt in 1995-99, and 61 ng/g lwt in 2000-02. BDE 47 comprised 55-65% of total in the 4 time frames. In contrast to BDEs, BB-153 (a marker for polybrominated biphenyls) and CB-153 (a marker for polychlorinated biphenyls) decreased in the samples over time.

Sjodin et al. (2001) provided another data set from the latter part of the 1980s showing similarly low concentrations of PBDEs. Twelve samples from United States donors who provided blood at a commercial blood collection facility in the state of Illinois in 1988, and then stored at -70°C, were retrieved for analysis. Seven BDEs were quantified: 47, 99, 100, 153, 182, 203, and 209. Three unidentified octa BDE and three unidentified nona BDEs were noted. The data was reported in units of pmol/g lw, converted to ng/g lwt by multiplying by the congener's molecular weight in pg/mole, and then pg converted to ng by a multiplication of 0.001 ng/pg. Although total concentrations were not provided—only median and range of concentrations for individual congeners were provided—an estimate of a median total concentration developed as a sum of the medians of individual congeners was 2.7 ng/g lwt. This is much lower than the 50-80 ng/g lwt total found by Schecter et al. (2005) from samples taken in 2003. Of interest is the finding of BDE 209 in 5 of 12 samples, with positives ranging from 1.5 – 33.6 ng/g lwt. It was stated that BDE 209 has a short half-life in humans, 6.8 days, so the presence here suggests continual exposure near the time of blood sample collection. Although not stated by the authors, its presence could also be laboratory contamination. Also of note is that the congener most consistently found of the other 6 was BDE 153, found at a range of 0.1 – 2.0 in all 12 samples.

Adipose tissue and serum was sampled from two disparate cohorts of women who were sampled in the late 1990s, and it was compared with a third cohort of women sampled between 1959 and 1967 (Petreas et al., 2003). One set of 32 adipose tissue samples were from women undergoing surgery for breast cancer between 1996 and 98. The second set was serum from a group of 50 Laotian women of reproductive age living in the SF Bay area, taken in the 1997–99 time frame. The final set was serum from a study of pregnant women enrolled in a case-control study of cryptorchidism and hypospadias as part of the Child Health and Development Studies (CHDS), taken between 1959 and 1967. Only BDE 47 could be quantified in this study, so it was the only congener measured and reported. Like the Schecter et al. (2005) finding of mostly non-detects in blood sampled from 1973, the entire set of 420 serum samples from 1959-1967 did not have any detections of BDE 47 at its high quantitation limit of 10 ng/g lwt. The mean and median from the adipose samples was 29.9 and 16.5 ng/g lwt, respectively, with 100% quantifiable measurements. The reproductive study revealed a mean and

median of 50.6 and 10 ng/g lwt with 48% quantified. There was no relationship between BDE 47 concentrations and age in both the adipose tissue and reproductive studies. This contrasts their finding of an increase of PCB 153 (which was also measured in these samples) with age in the adipose and reproductive studies.

Ninety-three anglers who had sufficient blood volume and who completed a fish consumption questionnaire were sampled between 2001 and 2003 (Morland et al., 2005). The urban anglers were from New York and New Jersey. Analysis was conducted for BDEs 47, 85, 99, 100, 153, 154, and 183. BDE 209 could not be reported because of high background contamination during the processing of the unknown samples. Fish eaters were categorized as "none" (eating no locally caught fish) or "any" (eating some locally caught fish), and "any" was further subcategorized based on amount of fish meals per month of locally caught fish. The highest congener found was BDE 47, at a geometric mean of 13.3 ng/g lwt, followed by BDE 99, at 3.2 ng/g lwt, BDE 153 similarly at 3.2 ng/g lwt, and BDE 100 at 2.7 ng/g lwt. All other congeners were not detected or very infrequently detected with geometric means less than 1 ng/g lwt. Although total concentrations found were not discussed, a sum of the geometric means of the congeners was 24.5 ng/g lwt. The straight mean might be higher because of the presence of a few very high concentrations. BDE 47 was found at a high of 1,388 ng/g lwt, and the high BDE 99 concentration was 546 ng/g lwt, for example. There were moderate, but statistically insignificant, increases in BDE concentrations from no local fish intake to > 1 meal/week.

Focant et al. (2004) describe a unique methodology for analysis of PBDEs and other PBTs. This method uses comprehensive two-dimensional gas chromatography and isotope dilution time-of-flight mass spectrometry (GC/GC-IDTOFMS) for the simultaneous measurement of selected polychlorinated biphenyls (PCBs), organochlorine pesticides (OCPs), and brominated flame retardants. Unlike classic GC/MS, this method evaluates all contaminants simultaneously with one injection into the GC column. Three milk samples and one blood sample were analyzed by Focant et al. (2004). The blood sample was from a pooled sample collected from 15 individuals in 2002 in three cities: Philadelphia, Memphis, and Miami. BDEs 17, 28, 47, 66, 85, 99, 100, 153, and 154 were analyzed. The blood sample results were expressed in pg/g fwt from blood serum, and were only presented on a graph. The results were estimated from the graph, and

converted to ng/g lwt, assuming 0.65% lipid in the serum. The total BDE from the one sample was 52.7 ng/g lwt, dominated by BDE 47 at 28.1 ng/g lwt, with comparable contributions by 99, 100, and 153 at 9.2, 6.9, and 6.2 ng/g lwt, respectively.

Twelve paired samples of maternal and cord blood were obtained from a hospital in Indianapolis during Aug-Dec, 2001, and analyzed for BDEs 47, 99, 100, 153, 154, and 183 (Mazdi et al., 2003). Results for maternal and cord blood were essentially identical: the range and median of total PBDE for mother's blood was 15 to 480 ng/g lwt and 37 ng/g lwt, respectively, and the corresponding range and median for infant blood was 14 to 460 ng/g lwt and 39 ng/g lwt, respectively. BDE 47 accounted for 53-64% of total PBDEs; BDEs 99, 100, and 153 each contributed 10-15% or total. BDE 154 and 183 were found rarely and at low levels. There was no age or BMI relationship with total BDEs. The authors claimed that the blood concentrations were 20-100 times higher than a similar population of Swedish mothers and children.

Wolff et al. (2005) conducted a study of exposures among mothers who were pregnant near the World Trade Center (WTC) Site on September 11, 2001. The study involved a complex evaluation of exposures, including measurement of key persistent contaminants including polycyclic aromatic hydrocarbons, polychlorinated biphenyls, dioxins, and PBDEs in blood. The authors did not find an association of PBDEs and measures of potential exposure to WTC contaminants, and they generally found low levels of PBDEs. Of 100 mothers, they found median levels for BDEs 28, 47, 99, 100, and 153 at 0.65, 9.7, 1.5, 1.8, and 1.8 ng/g lwt respectively.

Bradman et al. (2006) reports on a sampling of blood from pregnant Latina women, primarily from Mexico, living in Salinas Valley, California. The serum specimens were collected between September 1999 and January 2001. Seven BDEs were measured, including 47, 85, 99, 100, 153, 154, and 183. The resulting concentrations were fairly low, although not as low as the Wolff et al. (2005) study on pregnant women during the World Trade Center attacks. The median total from the 24 women was 21 ng/g lwt—with a high of 320 and a low of 5.3 ng/g lwt. Like other studies, the dominant congener was BDE 47. The authors show that the levels are highest among women who have spent less than 5 years in the United States, compared to women who have spent more than 5 years in the United States.

Fischer et al. (2006) present a case study of PBDEs in a family that showed somewhat elevated levels in the parents but higher levels in one child and still higher levels in the toddler of the family. Samples were collected from a family of 4, including 35 and 37 year-old parents, a 5 year-old daughter, and an 18-month old son in September and December of 2004. The sum of BDEs 47, 99, 100,153, and 154 in the parents ranged between 64 and 147 ng/g lwt in the two sampling dates, and BDE 209 contributed a relatively small addition at between 2 and 23 ng/g lwt. The story was much different with the children. The 5 year-old daughter had concentrations of 237, 239/249 ng/g lwt (the last two were duplicates of the same December sample) of the 5 congeners for the September and then December samples but a disparate range of 143 ng/g lwt of BDE 209 in the September sample and 9/12 ng/g lwt in the December sample (duplicates). The toddler had the highest concentrations of all: 418 and 488/476 ng/g lwt for the five congeners and 233 and 19/26 ng/g lwt of BDE 209 in the September and December samples, respectively. The authors discounted laboratory error and attributed the higher concentrations in the children to exposure to house dust. While the authors have discounted laboratory error, it would appear that a decline by an order of magnitude in both the toddler and infant is substantial, and could be due to some difference in the two laboratories. A decline of this magnitude is plausible because of the short half-life of BDE 209 in humans – it has been quantified on the order of 15 days, while the half-lives of lower brominated congeners has been quantified on the order of years. Whether due to differences in laboratories or differences in exposures, BDE 209 was quantified by both laboratories and this in itself is worthy of reporting. The higher levels of the other congeners in the toddler were attributed to his consumption of breast milk, although Fisher et al (2006) suggest that it might also be due to exposure to house dust. Of the non-209 congeners, the typical trend of seeing BDE 47 at the highest concentrations was true for all participants and sampling dates; BDE 153 was second most prevalent, ranging from one-thrid to one-half of the concentration of BDE 47.

Schecter et al. (2006a) measured the concentrations of 12 BDEs (17, 28, 47, 66, 85, 99, 100, 138, 153, 154, 183, and 209) in the blood of 8 vegans (no animal food products including dairy). They found a range of BDE concentrations of 12.4 to 127 ng/g lwt total, with a median of 23.9 and a mean of 53.3 ng/g lwt. BDE 47 was the most abundant congener, with the highest mean concentration of 23 ng/g lwt. The second

highest was BDE 153 at 14 ng/g lwt. BDEs 99 and 100 totaled about 6 ng/g lwt. BDE 209 was not detected, although detection limits were relatively high at between 2 and 7 ng/g lwt. The authors characterize these findings as lower, but not substantially lower, than other studies of BDEs in the blood of Americans, and they suggest that this could be the result of not consuming food of animal origin. They note that for dioxins and other POPs, foods of animal origin have been attributed as the main source of exposure, and because the concentrations of BDEs in vegans, who have been so for a minimum of 5 years, is not that much lower than other populations, exposures other than food may be important for this class of compounds.

These data suggest a range of approximately 30–100 ng/g lwt total BDEs is representative of blood in the general population of Americans in the 2000s, with occasional measurements in the 100s and 1,000s of ng/g lwt. Sampling of cord and maternal blood, as well as mother's milk, in Canada suggests somewhat lower levels. Ryan and Oostdam (2004) collected small amounts of individual maternal and cord blood samples from the North West Territories of Canada that were part of a "Northern Contaminants Program", composited them, and measured the composites for BDE congeners including 28, 47, 85, 99, 100, 153, 154, and 183. These samples pertained to years 1994 to 1999. In ten maternal composite samples, the mean total PBDE concentration was 23.3 ng/g lwt, with a range of 13.1 to 46.5 ng/g lwt, and the cord blood averaged 12 ng/g lwt (range or individual composite samples not provided). In the mother's blood plasma, BDE 47 comprised about 40% of total whereas in cord blood, BDE 47 comprised about 77%.

In contrast to the North American studies above, most studies from Europe, Asia, and elsewhere suggest concentrations of total BDEs in blood less than 10 ng/g lwt. Studies below include the countries of Sweden, Norway, New Zealand, the United Kingdom, Spain, the Faroe Islands, Japan, and Nicaragua.

Blood from 37 Swedish men were sampled in 1991 and 2001, and measured for PBDEs 28, 47, 99, 100, 128, 154, 153, 183, 196, 197, 203, 206, 207, 208, and 209 (Jakobbson et al., 2005). An additional 10 men were sampled in 1988 and 2002. These men were specifically selected to represent levels of fish consumption, so they do not necessarily represent a cross-section of the average population. The median and range of total concentrations from 1991 and 2001 was 11 (3.3-59) and 14 (4.2–57), respectively,

expressed in units of pmol/g lwt. Because individual results were not provided, these totals could not be converted to ng/g lwt. However, the conversion would result in concentrations that are somewhere between about one-fourth and three-fourths the listed concentrations as the conversion factors for individual congeners range from 0.486 [ng/g]/[pmol/g] for BDE 47 to 0.959 [ng/g]/[pmol/g] for BDE 209. BDE 47 was the dominant congener in 1991, which explains about 19% of total concentrations. BDEs 154 and 209 explained 12% and BDE 153 accounted for 8%. BDE 153, however, was the dominant congener in 2001: 21% of the mean. BDE 47 explained 11%, and BDEs 209 and 154 accounted for 10% and 8% respectively. The finding of an increase in BDE 153, and the meaningful contribution of BDE 209 are noteworthy in this study. The authors note a decline of other POPs like CB-153, p,p'-DDE and hexachlorobenzene between 1991 and 2001 of between 30-50%, while no decrease and a small increase was noted for BDEs. A second study in Sweden (Karlsson et al, 2007) looked at levels in air, dust, and blood from individuals in five households. Concentrations of the tri-hexa brominated congeners (BDEs 28, 58, 66, 99, 100, 153, and 154) were near the detection limit for 4 of the 5 individuals, with concentrations below 10 ng/g lwt for all measurements and the tri-hexa total at less than 15 ng/g lwt for all individuals. BDE 209 was detected in 4 of 5 individuals, at concentrations ranging from about 9.4 to 17.4 ng/g lwt. One individual also had quantified measurements of BDEs 197/204 (co-eluting), 196, 206, and 207 at concentrations ranging from 3.5 to 9.7 ng/g lwt. While the levels overall are generally low compared to levels found in the United States, they are noteworthy like the other study from Sweden discussed here in that the profiles had significant contributions from BDE 209 and even other nona and decaBDE congeners.

Pooled samples of about 20 individuals each from 5 hospitals in Norway (total number of samples analyzed was not provided) were analyzed for 11 BDEs: 28, 37, 47, 85, 99, 100, 119, 138, 153, 154, and 183 (Thomsen et al., 2005a). Samples for the years 1977, 1982, 1988, 1991, 1994, 1997, 1998, 1999, 2000, 2001, 2002, and 2003 were obtained. The sum of the seven most abundant congeners (28, 47, 99, 100, 153, 154, and 183) showed a concentration range of 0.5 to 5.0 ng/g lwt, with a clear trend of low concentrations for the early years: 0.5 in 1977, 1.3 in 1982, etc, which consistently rose to high levels between 3.6 to about 5.5 ng/g lwt between 1997 and 2003. A spike of about 5 ng/g lwt in 1991 could not be explained. BDE 47 was found in the highest

concentration except for one of the samples; the relative amount of BDE 153 appears to be increasing over time. Results for BDE 209 were presented, but the article is not consistent in its reporting of BDE 209 concentrations. The text suggests a median BDE 209 of 8.7 ng/g lwt, and the figures in the article show a remarkably high measurement of 35 ng/g lwt in 2000. These levels appear so much higher than others that even the authors suggest, "contamination of the sample cannot be totally excluded."

Harrad and Porter (2007) report on concentrations of BDEs including 47, 99, 100, 153, 154, and 183 in the blood of 23 individuals (10 males, 13 females age 20 to 64) sampled in 2001 in Wellington, New Zealand. The mean concentration of total BDEs was similar to other European studies at 7.17 ng/g lwt. Also similar to other studies was the finding that BDE 47 concentrations dominated, accounting for over 50% of the concentration. The second most found congener was BDE 153, explaining between 15 and 20% of concentrations. The authors claim the exposure was due to imported consumer goods, because these products are not produced in New Zealand.

Thomas et al. (2006) measured BDEs in the blood of 154 volunteers in 13 locations in the United Kingdom in 2003. They measured 22 congeners including BDE 209. The median total BDE was 5.6 ng/g lwt, with a range of 0.6 to 420 ng/g lwt, and only 5% greater than 30 ng/g lwt. BDE 209 was quantified in only 11 samples, with a high of 240 ng/g lwt, although the detection limit was high at 15 ng/g lwt. BDEs 47, 99, 100, 153, 154, and 183 were regularly detected, similar to other studies, but the median concentration of BDE 153 was the highest at 1.7 ng/g lwt, followed by BDE 47 at 0.82 ng/g lwt. This is atypical because BDE 47 is most often the highest found in profiles.

Gomara et al (2007) report on the sampling of PBDEs in human umbilical cord serum, maternal and paternal serum, placentas, and breast milk from individuals living in two locations (Vallecas and Getafe) in Madrid, Spain. The sampling occurred between October 2003 and May 2004, and involved 391 individual samples including 113 of maternal serum, 104 of paternal serum, 92 of umbilical cord serum, 30 of placenta, and 52 of breast milk. Fifteen individual congeners were measured in all samples, including BDE 209. The maternal, paternal, and umbilical cord serum samples had medians which ranged narrowly between 9.7 and 17 ng total PBDE/g lwt. BDE 47 was the predominant congener in the serum samples.

Blood concentrations of pregnant Faroese (the Faroe Islands are between Shetland and Iceland) women were determined from samples taken in 1994, and then their children's blood was sampled and measured 7 years later, in 2002 (Fangstrom et al., 2005). Fifty-seven mothers and 42 children were sampled, of which 41 were mother/child pairs. BDEs 47, 99, 100, 153, 209, and 153/154 were measured. Concentrations were low, with a median total concentration of just over 5 ng/g lwt for both mothers and children. The predominant congener for the mother was BDE 47 and the co-eluting BDEs 153/154, accounting for 26% each. For children, the predominant congener was 153, explaining about 46% of the total concentration. BDE 209 was present in both mothers and children at low concentrations of 0.8 and 1.0 ng/g lwt, respectively.

Samples of maternal blood plasma, cord blood plasma, and breast milk were taken in 2000-2001 from 15 mothers living in Stockholm (Guvenuis et al., 2003). They were opportunistic samples from women between 28 and 38 years old. For 55% of the women, this was their first child; for 33% of the women, this was their second child; and for 14%, this was their third. Ten BDEs were measured, including 17, 28, 47, 66, 85, 99, 100, 153, 154, and 183. The median and range from the three matrices, in ng/g lwt, were as follows: maternal blood—2.1 and (0.7–8.4); cord blood—1.7 and (0.5-4.3); breast milk—2.1 and (0.6 -7.7). BDE 47 was the predominant BDE in all matrices (46-70% in breast milk, 31-61% in maternal blood, and 45-94% in cord blood), followed by BDEs 153, 99, and 100. BDE 47 correlated in maternal blood and cord blood, but the levels of 153, 99, and 100 were higher in maternal blood as compared to cord blood (BDE 153 at 0.56 ng/g lwt in maternal blood but only 0.17 ng/g lwt in cord blood, e.g.). The authors suggested this might indicate that the higher brominated congeners do not pass thru the placenta to the same extent as do the lower brominated congeners.

Fukata et al. (2005) measured 27 BDE congeners including the key ones (47, 99, 100, 153, 154, 183, and 209) in umbilical cord tissue, maternal blood serum, and cord blood serum in Japan. Samples from eight volunteers were obtained and split into pool A and pool B (maternal blood was only available from pool B since there was insufficient volume). Umbilical cord samples were uniformly lower for all congeners as compared to cord serum and maternal serum, which were similar to each other. Total PBDEs in the two pools were as follows: 5 and 1.7 ng/g lwt in umbilical cord A and B respectively, 35

and 18 ng/g lwt in cord serum A and B, and 20 ng/g lwt in maternal serum from pool B. BDE 209 was not detected in umbilical cord, but it was found at 23 and 10 ng/g lwt in cord serum and 10 ng/g lwt in maternal serum. PCBs and CDD/Fs had a slightly different trend. While umbilical cord was always lower than cord serum (like PBDEs), they were close in magnitude and in fact maternal serum was significantly higher than either organ.

The one study which presented blood data outside of the United States with concentrations comparable to those found in the United States was a study in Nicaragua (Athanasiadou et al., 2008; results there are expressed in pmol/g lwt; results expressed in ng/g lwt are found in an earlier publication: Faldt et al., 2005). Five pools of serum from teenagers lived and/or worked near a waste disposal in Managua, Nicaragua, and then four pools of serum from women in different settings (urban areas, fishing villages, etc.) were analyzed (one analysis per pool). BDEs 47, 99, 100, 153, 183, 203, and 209 were analyzed. The pool of teenagers who both worked at the disposal site and lived nearby (no other pool was that exposed) had the highest concentrations, with a total over 600 ng/g lwt. The average of the other 8 pools was 38 ng/g lwt. BDE 47 was the most prominent congener, contributing just under 50% of the total concentration, with BDE 99 second at about 20%, BDE 100 at 11%, and so on. BDE 209 was present at equal levels in the teenagers living near and working at the disposal site, and all other groups, at about 5 ng/g lwt.

In summary, the review of literature on PBDEs in blood has revealed these trends:

1) Total PBDEs in the general population in the United States appear to be in the range of 30 – 100 ng/g lwt, although the studies have included outliers at the low end and at the high end. The most valid study of national trends, an evaluation of NHANES 2003/2004 (2040 serum samples, all greater than 12 years of age), had geometric mean concentrations of key congeners 28, 47, 99, 100, 153, and 154 at 1.2, 20.5, 5.0, 3.9, 5.7, and 2.3 ng/g lwt. One study of pregnant women in New York associated with 9/11 evaluations showed levels below 10 ng/g lwt, while outliers of studies with reasonably large sample size show concentrations in the 100s and even the 1000s of ng/g lwt;

2) one case study from the United States (presumably Fischer et al. 2006?) on 4 individuals in one family showed much higher levels in the children as compared to the adults, with exposure to house dust (because of elevated BDE 209 in the children) and mother's milk suggested to explain the levels in the 100s in the children as compared to around 100 ng/g lwt for the parents. This was the only study found with children;

3) United States levels are higher than levels found in nearly every other study done outside of the United States, with most non-United States data suggesting total PBDEs to be less than 10 ng/g lwt;

4) the predominant congener is BDE 47, explaining about 50% of the total concentration. The second most found congeners are 99 and 153, both explaining in the range of 10-20% of total concentrations.

5) Most of the studies have not measured BDE 209, but, when measured, it was found in about half the samples at low levels near 1-2 ng/g lwt. The exception of the one case study of the family of 4, which showed levels above 100 ng/g lwt in children; low levels of BDE 209 have been attributed to the rapid half-life of 15 days in humans, and the higher levels in children attributed to dust exposures in the house.

5.2.2. Breast Milk Data

While blood data suggested concentrations of total PBDEs in the range of 30–100 ng/g lwt, data on PBDEs in breast milk suggest possibly higher concentrations, with medians or means in some studies in the United States above 100 ng/g lwt. Table 5-2 shows congener-specific milk concentrations of BDEs from studies in the United States. Interestingly, in one blood study, analysis of results by individuals suggested that females could have meaningfully higher concentrations than males. As described above, Schecter et al. (2005) found that, in 39 samples from 22 males and 17 females, the range and mean in males were 4.6-192.8 ng/g lwt and 25.1 ng/g lwt, and, for females, the range and mean were 5.6-365.5 ng/g lwt and 74.1 ng/g lwt. However, Schecter et al (2005) does note that these differences are not statistically significant.

The Environmental Working Group (EWG) sampled 20 primaparae women from around the country for 35 PBDEs (Lunder and Sharp, 2004). Total BDEs averaged 159 ng/g lwt, ranging from 9.5 to 1,078 ng/g lwt, with 6 having levels above 100 ng/g lwt, and 2 exceeding 700 ng/g lwt. These results were the highest found to date, and significantly higher than European women. The most common BDE was 47, which accounted for about half of the total PBDEs in each participant. The variability in PBDE levels could not be explained by the diet, occupation, age, body mass, or the amount of time they had breastfed their infants.

Another environmental organization, the Northwest Environment Watch (NEW), conducted a study in 2003 (NEW, 2004). Between April and November of 2003, 40 first-time breastfeeding mothers from the Pacific Northwest, 10 each from Washington, Oregon, British Columbia, and Montana, were sampled for the presence of BDEs 32, 28/32, 47, 66, 71, 85, 99, 100, 153, 154, 1803, and 209. Levels ranged from 6 to 321 ng/g lwt in the breast milk, with median and mean levels of 50 and 97 ng/g lwt, respectively. These levels are comparable to blood and other measurements in the United States, but 20 to 40 times higher than levels measured in Sweden and Japan, according to the authors. BDE 47 was the highest found, with a median and mean level of 26 and 50 ng/g lwt, respectively, with BDE 153 at 4.8 and 16 ng/g lwt, BDE 100 at 5.2 and 12 ng/g lwt, and BDE 99 at 5.4 and 10 ng/g lwt. BDE 209 was found in 24 of 40 samples; it had a high concentration of 4 ng/g lwt and median and mean concentrations of 0.4 and 0.8 ng/g lwt, respectively.

Schecter et al. (2003) collected milk from 47 volunteer donors between August and December 2002; 24 donors were from Austin, TX, and 23 were from Dallas, TX. Samples were measured for 13 BDEs including 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209. No information was available as to whether these women were primaparae. It is, however, reasonable to assume they were randomly selected because milk banks were the source of milk in Austin and clinics were the source in Dallas. The mean total BDE was 74 ng/g lwt (median = 34; max = 419). The dominant congener was BDE 47, comprising 54% of this total, with BDE 99 comprising 19% and BDE 153 comprising 7%. BDE 209 was quantified in only 7 samples, with 16 NDs and the remaining samples not measured for BDE 209. Schecter et al (2003) notes a mean

concentration for the 23 samples of 0.9 ng/g lwt. Measurements were not correlated with age or length of time in nursing.

Focant et al. (2004) evaluated two different analytical methods on both blood and milk samples. They analyzed three pooled samples of mother's milk: one pool from two mothers in Denver, one pool from 10 samples collected in 2003 in California, and the third pool from 10 individuals in North Carolina, also in 2003. BDEs 17, 28, 47, 66, 85, 99, 100, 153, and 154 were analyzed. The average total BDE from these three was 315 ng/g lwt, with BDE 47 the highest at 193 ng/g lwt (61% of total), followed by BDE 99 at 55 ng/g lwt (17%), and BDE 100 at 34 ng/g lwt (11%). Results from the more traditional GC-IDHRMS analysis were compared with the newer GCxGC-ID TOFMS, revealing reasonable agreement. Because often the number of samples (derived from the different pools) analyzed by GC-IDHRMS was higher than the newer method, the summary results are from these analyses.

Two studies have been conducted in the Boston, Massachusets area. In one, breast milk was collected from 46 women, with total BDE concentrations ranging from 4 to 263 ng/g lwt, with a median of 28 ng/g lwt (Wu et al., 2007). Only one sample, the highest at 263 ng/g lwt, was higher than about 130 ng/g lwt. BDE 47 dominated most samples, although BDE 153 predominated in three samples, and BDE 209 was above detection limits in 11 samples. Questionnaire data suggested that concentrations in house dust as well as the consumption of frozen dairy products provided the strongest associations with log-transformed total PBDE in breast milk. The second study in the Boston area was conducted by Johnson-Restrepo et al. (2007), who measured breast milk in 38 volunteer donors between June and November 2004. They measured 17 congeners including BDE 209, and found a median total concentration of 19.8 ng/g lipid, with a range of 0.06 to 1910 ng/g lwt and a mean of 75 ng/g lwt. BDE 209 was not detected in any sample, although the detection limit appeared high at 204 ng/g lwt. The most abundant congener found was BDE 47, explaining about half the concentrations found.

A limited set of longitudinal data (i.e., data on changes over time) was available for 3 women for BDEs 47 and 99 (Sjodin et al., 2005). These data originate from an ongoing study a Pennsylvania State University College of Medicine, where a cohort of 30 participants who seek prenatal and pediatric care are being enlisted for a longitudinal study of PBDEs, pesticides, and PCBs in milk. Contrary to expectations, levels of all of

these contaminants suggested increases over time. BDE 47 increased from 30 to 40 ng/g lwt in 2 of 3 women from postpartum day 40 to 120, and from 10 to about 15 ng/g lwt from day 40 to day 60 in the other woman. BDE 99 increased from 50 to100 ng/g lwt in one participant from day 40 to day 120, increased slightly from approximately 5 to 6 ng/g lwt from day 40 to day 60 in another, and decreased from 8 to about 5 ng/g lwt from day 40 to day 90 in the third participant.

Ryan et al. (2006) reported on the sampling of human milk from two locations in North America: in Hamilton, Ontario, Canada and in Austin, Texas, of the United States. The Canadian milk samples included 34 that pertained to 2005, 13 that pertained to 2003, 14 that pertained to 2002, and 26 that pertained to 1992. Specific congeners were not identified; only total PBDEs was reported. The lowest concentrations found were in 1992, with a median of 3.1 ng/g lwt total PBDEs. Concentrations rose to a median of 33 ng/g lwt in 2003, with another rise to a median of 39 ng/g lwt in 2003, and then a decline to a median 20 ng/g lwt for 2005 samples. The Texas samples pertained to years 2004 and 2002, and the median concentrations for those two years was higher than these Canadian samples at medians for 2004 and 2002 of 43 and 44 ng/g lwt

Like the blood data, the mother's milk data suggests much higher concentrations in America compared to European and Asian countries. Extracted milk fat from the 3rd round of the WHO-coordinated exposure study was evaluated for the presence of BDEs (Kotz et al., 2005). As of the writing of the study, samples from 17 different locations (of 24 total) were used. Nine BDEs were quantified, including congeners 15, 28, 47, 77, 99, 100, 126, 153, and 183. The highest level by far was the level found in a sample from the United States at 373.6 ng/g lwt; the second highest was 10.3 ng/g lwt from a sample from Ireland. The predominant congener was BDE 47, at 63% in United States samples (233 ng/g lwt), followed by BDE 99 at 16% (60 ng/g lwt) and BDE 100 at 11% (41 ng/g lwt).

Schuhmacher et al. (2007) report on concentrations of 15 BDE congeners (although the specific congeners were not identified except to note that samples were fortified with BDEs 28, 77, 99, 153, and 183; no information on whether BDE 209 was included). Their study included 15 women, sampled in 2002, that lived in an urban area (7–10 km from a hazardous waste incinerator; 7 women) and near an industrial zone (8 women). There did not appear to be a distinction in the two small groups, with means of 2.2 and 2.5 ng/g lwt in the urban and industrial zones, respectively.

Thomsen et al. (2005b) sampled breast milk of 151 women representing the northern, southwestern, and eastern parts of Norway. Samples were analyzed for BDEs 28, 37, 47, 85, 99, 100, 119, 138, 153, 154, and 183. The sum of the seven most abundant BDE congeners (28, 47, 99, 100, 153, 154, and 183) ranged from 0.95 to 21.05 ng/g lwt, with a median of 2.35 ng/g lwt, which is comparable to other European countries. BDE 47 was the most abundant; it had a median of about 1.15 ng/g lwt. BDE 153 was the second most abundant: a median of about 0.50 ng/g lwt. By use of a multiple linear regression model, it was shown that there was a statistically significant positive correlation with age (older women had higher concentrations) and a negative correlation with parity (number of children) and education. The mean concentrations of total BDEs for women <28, 28-31, and >32 was 3.24, 3.39, and 5.17 ng/g lwt, respectively. The mean concentration for women with one child was 4.33 ng/g lwt. Women with more than one child had a lower concentration: 3.64 ng/g lwt. The authors assert that this was the first study to show statistically significant correlations with age and number of children.

Gomara et al (2007) report on the sampling of PBDEs in human umbilical cord serum, maternal and paternal serum, placentas, and breast milk from individuals living in two locations (Vallecas and Getafe) in Madrid, Spain. The sampling occurred between October 2003 and May 2004, and involved 391 individual samples including 113 of maternal serum, 104 of paternal serum, 92 of umbilical cord serum, 30 of placenta, and 52 of breast milk. Fifteen individual congeners were measured in all samples, including BDE 209. Breast milk samples had median concentrations in the two locations of 6.1 and 5.5 ng total PBDE/g lwt, which was a bit lower than the blood samples, which had medians ranging from 9.7 to 17 ng total PBDE/g lwt in the various blood matrices. BDE 209 dominated the breast milk samples, with medians of 2.8 and 2.9 ng/g lwt (ranging as high as 52 ng/g lwt) in the two locations.

A total of 89 lactating mothers in four towns in Japan provided both serum and milk samples for analysis of 13 BDEs, including BDEs 15, 28, 47, 99, 100, 153, 154, 183, 196, 197, 206, 207, and 209 (Inoue et al., 2006). The geometric means for the total amounts of the 13 PDEs in human milk and serum was 1.56 and 2.89 ng/g lwt, respectively. BDE 209 was the predominant congener in serum, accounting for 38% of the total amount of BDEs, but it was a minor component in milk, accounting for 8%. In

milk, BDEs 47 and 153 were the major contributors, accounting for 28 and 23% of total BDEs, respectively. Nursing duration was found to be correlated with PCB concentrations in the infants, but not so for PBDEs. In contrast, geography was tied to PBDEs, but not to PCBs.

Fangstrom et al. (2008) conducted a temporal study on Swedish mother's milk. Fourteen pooled milk samples representing 1980 (116 mothers pooled), 1984/5 (102 mothers), several of the years between 88 and 2002 (20 mothers), 2003 (15 mothers), and 2004 (20 mothers) were sampled for BDEs 47, 77, 99, 100, 153, and 209. It was not possible to quantify BDE 209 in milk, and the authors suggest this could be due to the short half-life of this compound that has been noted for serum. From the middle of the 1990s, the concentrations of the lower brominated BDE congeners (47, 99, and 100) are decreasing, while 153 appears to be retaining its levels reached towards the latter 1990s. BDE 47 was near or less than 0.5 ng/g lwt prior to 1990, reached levels above 2.0 ng/g lwt by mid-1990s, and dropped sequentially from 1.8 (in 2001) to 1.4 (2002) to 1.2 (2003) to 0.9 (2004). Very similar trends were seen for 99 and 100, with peaks between 0.5 and 0.8 in the mid-1990s, dropping to 0.3 ng/g lwt in the early 2000s. Meanwhile, the highest concentration of BDE 153 till the 1999 sampling was the BDE 99 sample at 0.82. Afterwards, BDE 153 ranged from 0.7 to 1.3 between 2001 and 2004. This trend of dropping lower chlorinated BDEs and increasing higher BDEs was seen in the United States, Norway, the Netherlands and the Faroe Islands. This might be due to declines in use of the pentaBDE formulation and/or the debromination of BDE 209 and other higher brominated BDEs.

In a temporal study of organohalogen compounds, including PCBs, PCDD/Fs, and PBDEs, in the breast milk of German women, Furst (2006) found an increase in the mean concentration of PBDEs from a pooled sample (n=300) in 1992 to a sampling of 79 women in 2002. Specifically, the mean total concentration (including BDEs 28, 47, 66, 85, 99, 100, 153, 154, and 183) increased from 1.87 ng/g lwt to 3.75 ng/g lwt. This contrasted with the other organohalogen compounds studied, which showed reductions during this time period.

Ohta et al. (2002) determined the concentration of PBDEs in breast milk of 12 primaparae women at one month after delivery in Japan. The PBDEs evaluated included 28, 47, 99, 153, and 154. Concentrations ranged between 0.7 and 2.8 ng/g lwt in the

breast milk. Samples were also taken of numerous food products, including 20 fish samples, spinach, potato, carrots, pork, beef, and chicken (see Chapter 4 for a summary of the results). Questionnaires on food consumption were given to the women, and a strong correlation was found between consumption of fish and milk concentration. In a "high" group of fish consumers (n=5), the average breast milk concentration was 1.7 ng/g lwt, while the concentration in the "low" group of fish consumers (n=3) was 0.8 ng/g lwt.

Toms et al. (2007) measured BDEs in pooled milk samples from Australia. A total of 157 milk samples were collected between 2002 and 2003, and they were pooled to create 17 regional samples. Eighteen congeners were measured; BDE 209 was not measured. Total PBDE averaged 11.1 ng/g lwt (median = 11.0 ng/g lwt) with a narrow range of 6.1–18.7 ng/g lwt. BDE 47 dominated the profile, explaining over 50% of the total, followed by 99, 100, and 153—all of which contributed between 10 and 20%.

In summary, data suggests concentrations of total BDEs in women's breast milk in the United States exceeds that in blood, perhaps averaging near 100 ng/g lwt, in contrast to a range more like 30–100 ng lwt in blood generally. Part of this trend could be a gender issue, as one blood study (Schecter et al. 2005) suggested higher concentrations in women than in men, although this difference was not statistically significant. Similar to blood data, concentrations in breast milk of United States women exceeded that of women outside of the United States. Breast milk concentrations of total PBDEs outside of the United States appears to mostly below 10 ng/g lwt. As for blood, BDE 209 was usually not measured or when measured, not found in most instances (only one study found significant levels of BDE 209 in blood, and that was in a case study and it was found in one child and one toddler). BDE 47 was the predominant congener, followed by either BDEs 99 or 153.

5.2.3. Adipose and Other Tissue

Most of the body burden data originates from either blood or human milk. The very limited data on adipose and other tissue is generally consistent with blood and milk data – United States data are higher than data from other countries; BDE 47 predominates, and BDE 209 is either not measured for or not found when measured.

Johnson-Restrepo et al. (2005) measured BDEs in adipose tissue from 52 individuals who were undergoing liposuction during Oct, 2003–Oct, 2004 in New York

City. BDEs sampled included 28, 30, 47, 85, 99, 100, 153, 154, and some unidentified di, tri, and penta-BDEs. The occurrence of BDE 209 was examined qualitatively, but it was not detected. Total BDEs ranged from 17.4 to 9,630 ng/g lwt, with a median of 77.3 ng/g lwt and a mean of 399 ng/g lwt. The mean dropped to 141 ng/g lwt when two outliers of 9,630 and 4,060 ng/g lwt were dropped. The authors claimed this was 10 to 100 times higher than concentrations reported for human adipose tissue collected from several European countries. The data showed no correlation with age, which is consistent with the theory that most exposure was only in recent years. BDE 47 was the predominant congener, explaining roughly 33% of the total BDEs (at a mean of 132 ng/g lwt), followed by BDE 153 (23%; 91.8 ng/g lwt), BDE 99 (18%; 74.4 ng/g lwt), and BDE 100 (17%; 67.7 ng/g lwt).

Data on 11 adipose tissue samples from 32 women in the industrial port town of Porto Alegre, Brazil, showed a range of 0.73 to 3.69 ng/g lwt, with BDE 47 dominating the profile (median = 0.52 ng/g lwt), followed by BDE 99 (median = 0.34 ng/g lwt), BDE 153 (median = 0.19 ng/g lwt) and BDE 100 (median = 0.12 ng/g lwt). BDE 153 was found in only 27% of samples with a median of 0.07 ng/g lwt (Kalantzi et al., 2005).

Naert et al. (2006) measured BDE congeners 28, 47, 99, 100, 153, 154, and 183 in abdominal adipose tissue from 53 individuals (31 men, 22 women) who died from natural causes or accidents in Belgium. The mean age of the individuals was 53 when they died, and no information on diet or exposures was available. Consistent with blood data from Europe, total BDE concentrations ranged between 1.23 and 57.2 ng/g lwt, with a median of 5.32 ng/g lwt. The most predominant congener was BDE 153, at a median of 2.40 ng/g lwt, with all other congeners having medians under 1.00 ng/g lwt.

Schecter et al. (2007) measured levels in fetal liver tissue samples that were obtained from 4 stillborn fetuses and 7 liveborn infants, all of whom died shortly after birth and before any feeding. Thirteen congeners were evaluated, although BDE 209 was not found in any samples. The mean total concentration was 19.5 ng/g lwt (assuming ND = 0, at ND = ½ DL, the total went up to 23.1 ng/g lwt owing mainly to an average of 3.5 ng/g lwt assuming ND = ½ DL for BDE 209). The congener profile was fairly similar to blood and breast milk profiles, being dominated by BDE 47 at 55% of the total, BDE 99 at 23%, and BDE 100 and 153 at 10 and 6%, respectively. One sample was uniquely high, 96 ng/g lwt, while all other samples were under 33 ng/g lwt. This one high sample

was from the infant who lived the longest, 7 days. The authors did not clarify why this infant was characterized as having died after 7 days before any feeding could occur.

Gomara et al (2007) report on the sampling of PBDEs in human umbilical cord serum, maternal and paternal serum, placentas, and breast milk from individuals living in two locations (Vallecas and Getafe) in Madrid, Spain. The sampling occurred between October 2003 and May 2004, and involved 391 individual samples including 113 of maternal serum, 104 of paternal serum, 92 of umbilical cord serum, 30 of placenta, and 52 of breast milk. Fifteen individual congeners were measured in all samples, including BDE 209. The concentrations were lowest in the placentas, with a median value of 1.9 ng total PBDEs/g lwt. This compared to medians in the breast milk around 6 ng total PBDE/g lwt, and medians in the blood matrices ranging narrowly between 9.7 and 17.0 ng total PBDE/g lwt.

5.2.4. Selection of Representative Body Burden Profiles

In Chapter 4 (Section 4.8), exposure media concentrations were assigned for a select group of PBDE congeners in order to conduct the exposure assessment. Specifically, these assigned media concentrations were used to estimate exposure intake dose (see Section 5.4 below.). Also, simple pharmacokinetic exercises were applied to those intakes and used to predict body burden levels (see Section 5.5 below). In order to evaluate the merit of that exercise, those body burden predictions were compared with measured body burdens. This section will assign representative central tendency body burdens for that purpose.

As also discussed in Chapter 4 in the context of exposure media concentrations, assignment of these representative body burdens should have these characteristics: 1) they should originate from the United States, or maybe Canada, as representative of industrial North American patterns, 2) they should be representative of background and not occupational exposures, and 3) preferably, they should originate from a single study that has the ideal characteristics of having a large sample size, being from a diverse geographic area, and containing the appropriate set of congeners. Table 5.3 has those assignments, one set each for blood and mother's milk.

The blood data are from Sjodin et al. (2008). Of all the United States blood data available, this evaluation of NHANES 2003/4 is the most representative of recent, national trends. Geometric mean concentrations, shown in Table 5.3, are provided for BDEs 28, 47, 99, 100, and 153, but not for BDEs 17, 66, 85, 154, and 183, because these were only quantified in 40% of the samples. Arithmetic means were not provided in Sjodin et al. (2008) and medians might have been selected over geometric means as most representative of central tendency, but the median of BDE 99 was provided as less than detection limit, while the geometric mean was provided for BDE 99. There are five studies on PBDEs on United States breast milk that could be used to represent background conditions. These include two by public interest research groups—the Environmental Working Group (EWG; Lunder and Sharp, 2004) and the Northwest Environment Watch (NEW, 2004), two conducted in the Boston, MA area (Wu et al., 2007; Johnson-Restrepo et al., 2007), and a study of 47 samples taken in Texas by Schecter et al. (2003). None of the studies have a large sample size. The EWG study includes 20 samples from around the United States, so it has broad coverage, but it also has the highest concentrations of any United States study on breast milk. The NEW study represents several states in the Northwest, has a sample size of 40, and its concentrations track well with EWG, but are consistently lower. The Schecter et al. (2003) data on 47 samples are all from Texas. Their data also tracks with the two studies, but it is the lowest of all the data and originates from one state only. The two studies from Boston, MA, found the lowest concentrations, with median levels at 28 ng/g lwt total (Wu et al., 2007) and 19.8 ng/g lwt (Johnson-Restrepo et al, 2007). The NEW median data from the Northwest will be used to characterize breast milk; it is displayed in Table 5.3. It was chosen because it represented several states, it had as large a sample size as others, and the concentrations were consistent among the US studies other than the EWG study which had the highest concentrations. Mean concentrations were also provided in the NEW study, but they were meaningfully higher than median concentrations: the median total PBDE concentration was 50 ng/g lwt (this median contained more congeners than the median of 44 ng/g lwt shown in Table 5.3) while the mean total concentration was 97 ng/g lwt. Because geometric means were chosen to represent blood concentrations, the most analogous statistical representation of breast milk concentrations, median concentrations, were chosen.

5.3. STUDIES ON INTAKE, OR EXPOSURE, DOSE

Several researchers who measured PBDEs in exposure media then went on to estimate the exposure dose associated with that media. Their approach was simply to associate the media concentration with a contact rate. For example, a given concentration in dust times an amount of dust ingested per day provides an estimate of the daily dose via dust ingestion. This was done most often for studies on house dust and food, but researchers also considered inhalation exposures when measuring air. A second approach used to estimate intake dose from house dust was to measure a surface loading (not a bulk concentration) on the hands in units of mass/unit area, and then use an empirical model to correlate that loading with an intake rate, in units of mass/time. Other researchers took a more studied approach to evaluating exposure to PBDEs, using models, statistical approaches, and other avenues to provide estimates and insights on exposure to PBDEs. Table 5-4 provides estimates of these exposure doses.

As discussed in Chapter 4, studies have shown indoor dust to contain much higher concentrations of PBDEs compared to outdoor soils. Subsequently, the "soil ingestion" pathway has focused on indoor dust measurements. Stapleton et al. (2005, 2008b) have conducted the most comprehensive dust pathway analyses with their studies on house dust coupled with exposure modeling. Using estimates of inadvertent ingestion of dust by young children (ages 1-4), 0.02–0.2 g/day, they used their measurements of PBDEs in dust to estimate a total ingestion of PBDEs range from 120 to 6,000 ng/day (Stapleton et al., 2005). They list an adult exposure of 3.3 ng/day, but this is based on a low estimate of 0.56 mg/day of dust ingestion. This value was found in EPA's *Exposure Factors Handbook* (EPA, 1997), where Hawley (1985) is cited for using a value of 0.56 mg/day to characterize adult exposure to housedust from normal activities in the house (higher exposures of over 100 mg/day resulted from "work in the attic"). Stapleton et al. (2008b) later approached the dust ingestion pathway from a different angle. They measured PBDEs in hands using sterile gauze pads soaked in isopropyl alcohol. They measured PBDEs on the hands of 33 individuals residing in Durham, North Carolina, and found a median total load per hand of 130 ng, or when normalized to surface area, a surface loading of total PBDEs of 135 pg/cm^2. They measured 13 congeners including BDE 209, which was found in 22 of the 33 samples, with a median total load of 25.5 ng, and a high

of 270 ng. Using an empirical approach based on contact events per day, transfer efficiency, the hand loadings, and the fraction of hand coming in contact with the mouth, they estimated median exposures to the adult and child to be 154 ng/day and 1380 ng/day.

In contrast to the low dust ingestion rate of 0.56 mg/day assumed by Stapleton et al. (2005) in their earlier estimates of PBDE ingestion via dust, Sjodin et al. (2004b) assumed an upper limit ingestion rate of 100 mg/day, and using their median concentration of 4,200 ng/g in house dust from Atlanta (range of 530–29,000 ng/g), they suggest that this pathway could add up to 400 ng/day of PBDE exposure. Such a total would dwarf estimates of 40 to 100 ng/day from food ingestion they cite from the literature. Harrad et al (2008) used "mean" and "high" dust ingestion rates of 20 and 50 mg/day to model adult ingestion of PBDEs based on measurements from homes in Texas. Using arithmetic mean concentrations from dust samples (n=28), they estimate a "high" total intake of 228 ng/day, which includes BDEs 28, 47, 99, 100, 153, 154, 183, and 209. They also provide estimates for toddlers (6-24 months) assuming 50 mg/day as a "mean" and 200 mg/day as a "high"ingestion rate. Again using average dust concentrations, a high toddler intake estimate was 91 ng/day. They also provide estimates for toddlers and adults in the UK, New Zealand, and Canada, based on dust sampling from those countries. The most interesting finding was that UK dust was substantially higher in BDE 209 compared to all other countries, and median intakes of BDE 209 were double those from the US. Using data that originated from Kuwait, Gevao et al. (2005) used standard exposure assumptions for dust ingestion for children (100 mg/day) and adults (10 mg/day) and found that the mean ingestion of total PBDEs averaged 2.0 ng/day for children, and 0.2 for adults. Harrad et al. (2006) used indoor dust measurements from 8 homes in the United Kingdom to estimate a possible range of adult dust ingestion exposures from 0.9 to 22 ng/day total BDEs, and a range for toddlers of between 12 and 43 ng/day.

Similar to the relationship between outdoor soils and indoor dust, outdoor air concentrations were typically lower than indoor air measurements, and, hence, literature estimates of inhalation exposures to PBDEs have focused on indoor air concentrations. Using data from passive indoor air in a Canadian study, Wilford et al. (2004) assumed standard resting respiration rates and found that the median exposure via inhalation was

1.9 ng/day for females and 2.0 ng/day for males, which compares to a Canadian estimate of 44 ng/day by dietary intake. Hazrati and Harrad (2005) used passive air measurements in 12 homes, 10 offices, and 1 private car, and an inhalation rate of 20 m^3/day, to estimate a mean daily intake via inhalation of 4.3 ng/day in the United Kingdom. Harrad et al. (2006) used other air data to conclude that average inhalation intakes were 2 ng/day total PBDEs and less in the United Kingdom. Using air concentration data from a study in Kuwait, Gevao et al. (2005) estimated inhalation doses of 0.4 ng/day for adults and 0.2 ng/day for children (with inhalation rates of 20 m^3/day for adults and 8.3 m^3/day for children. However, Meng et al. (2007) estimated inhalation exposures to PBDEs using data on outdoor urban air concentrations from China (see Chen et al., 2006). They found median exposures doses to range from 2.7–9.2 ng/day. They note this is higher than the estimates provided in Wilford et al. (2004) and Harrad et al. (2006), but they claim this is because they included BDE 209 in their estimates unlike these other two studies, and this congener dominated the air profiles. Mandalakis et al. (2008) studied air concentrations in cars in Greece, and combining their measurements with inhalation rates and time in cars per day, they estimated inahaltion of total PBDEs while driving to range between 0.0005 to 2.9 ng/day, with a median of 0.2 ng/day. This exposure was dominated by BDE 209, explaining about half of all exposure. They also found that, despite the small amount of time in the car, that this activity contributed 29% of overall daily inhalation exposure.

Most direct dose estimates in the literature pertain to dietary dose, and they are developed mostly by individuals who also measured food concentrations. Estimates of dose originate from the United States and overseas, with United States studies summarized here first. Schecter et al. (2006b) combined their measured average food concentrations with food consumption rates to calculate intakes for various age ranges (2-5, 6-11, 12-19....>= 60) and for males and females. Total intakes ranged from about 0.9–1.5 ng/kg body weight/day for males/females above the age of 12. For ages 2-5 (males & females), the intake was estimated at 2.7 ng/kg-d, and for ages 6-11 (males & females), the intake was 1.8 ng/kg-d. Individual intakes between 47 and 99 were nearly identical for males and females, ranging from 0.4 to 0.7 ng/kg-d after age 12, 0.9-1.5 ng/kg-d for the two earlier age ranges. Huwe et al. (2005) estimated a dietary intake of PBDEs from meats for a consumer of "lean meats" (5% lipids) was 0.3 ng/kg-d, while a "higher fat

meats" consumer had an intake of 0.8 ng/kg-d. The estimates are based on an average body weight of 53 kg. Fish, meat, and fowl products were purchased in December 2003 and February 2004 from 3 different food markets in Sacremento and El Dorado Hills in Northern California (Luksemburg et al., 2004). Using average daily intake by adults and children taken from the *Exposure Factors Handbook* (EPA, 1997), dose estimates were provided with their data. Using the highest and lowest concentrations measured in wild and farm-raised fish, the theoretical average daily intakes of PBDEs through fish ingestion ranged between 0.1 and 1.0 ng/kg-day in children and between 0.02 and 1.0 ng/kg-day in adults. Assuming the highest and lowest concentrations measured in beef and chicken products, theoretical average daily intakes of PBDEs through ingestion ranged between 0.4 and 20 ng/kg-d in children and between 0.4 and 10 ng/kg-d in adults.

Ten studies were found providing dietary dose estimates in Europe and Asia. Bocio et al. (2003) estimated that dietary intake equaled 97.3 ng/day for total PBDEs for adults in Spain, based on a total diet survey. This was based on homologue group concentrations; a later survey of Spanish foods evaluating 15 individual congeners arrived at an estimate of 38.5 ng/day for BDEs (Gomara et al., 2006). Schuhmacher et al. (2007) used the food concentration data of Bocio et al. (2003), in combination with food consumption rates for an urban and industrial area of Spain to calculate dietary intakes of total BDEs of 72 (urban) and 63 (industrial) ng/day. Harrad et al. (2004) measured PBDEs in duplicate diet samples from both a vegan and omnivorous diet. They estimated a dietary exposure average 107 ng/day (median = 91 ng/day) using consumption data from the survey, and although they did not estimate an exposure intake for the vegans, they provided the omnivorous and vegan concentrations, and it is noted that the vegan concentrations were about one-half the omnivorous concentrations. They note this discrepancy is not as large as the discrepancy for other POPs, like dioxins, which bioconcentrate substantially more in animal fat. They could not explain this trend but noted other literature showing significant concentrations in vegetative food products. Knutsen et al. (2005) combined concentrations from a market basket survey with a comprehensive food consumption survey to estimate a mean daily exposure of 62.5 ng/day, a median exposure of 48.6 ng/day, and a 95% exposure of 149.0 ng/day for Norway. Without fish, these numbers were about one-third as much, with mean at 20.0 and median at 19.2 ng/day. When a different survey more specific to fish types was used,

the median rose to 74.2 ng/day, and then even higher when a recommended daily additional intake of cod liver oil was assumed (which is recommended by the Norwegian government as a healthy supplement)—median intake rose to 122.9 ng/day. A similar combination of a comprehensive market basket consumption survey with composite food samples measured for PBDEs was conducted by Bakker et al. (2008) for the Netherlands. They found a median dietary intake of 0.79 ng/kg-day, with a 95% of 1.62 ng/kg-day, dominated by dairy and fish at 39 and 28%, respectively. An estimate of 51 ng total BDEs/day was derived for diet only for Swedish general population (Darnerud et al., 2001). Concentrations in a market basket survey were combined with dietary intakes from fish and fish products, meat, dairy, and fats/oils. Fish products contributed about half of total. Meat, dairy, and fats/oils contributed about 15% each. Using mother's milk concentration of 4.2 ng/g lwt, they estimated an infant dose of 110 ng/day.

It is noted that BDE 209 was not included in any of these dietary intakes and surveys; the standard suite of BDE congeners (47, 99, 100, 153, and 154) were included. Three studies were found that included BDE 209 as well: one on fish consumption in China, one in Belgium, and one was in the United Kingdom. Fish consumption intakes were determined for a surveyed group of individuals in China, and the median dose ranged from 1.7–12.9 ng total/day for several age ranges (Meng et al., 2007). While including BDE 209, it was found in only 14 of 390 fish samples upon which the intake estimates were based. This could have been due to the high detection limit, 0.1 ng/g wwt, of this congener compared to detections limits of 0.001–0.003 ng/g wwt of 10 other congeners measured. The one in Belgium did not include individual congener breakouts, but it was stated that BDE 209 was never found above LOQ in any food sample. The average total adult intake was only 35 ng/day, generally low but in line with other European surveys which did not include BDE 209 (Voorspoels et al., 2007).

However, the presence or absence of BDE 209 could very well be a major issue for these literature estimates of European exposures, as a dietary estimate based on a survey which did include BDE 209 arrived at substantially different results. The United Kingdom Food Surveillance Agency (FSA, 2006) recently published results of a food survey including BDE 209 and quite alarmingly found BDE 209 at the highest level of all BDEs (as noted in Chapter 4 in the section on food concentrations). They estimated exposure doses in conjunction with their food concentrations and found average intakes

totaling 5.9 ng/kg-day, of which 4.5 ng/kg-day was due to BDE 209. These results either are questionable themselves, or alternately, throw into question other European surveys on "total" dietary dose of BDEs which have not measured BDE 209 in the food. It is noted that, in a study on BDEs in dust in Europe, BDE 209 dominated substantially over other congeners (Fabrellas et al., 2005), providing support to this finding in food, and suggesting that much of the European literature on exposure to BDEs does not tell the complete story by not considering BDE 209.

Jones-Otazo et al. (2005) used models, in combination with reported food concentrations, to provide a comprehensive evaluation of exposure to PBDEs pertinent to the Toronto urban environment. A regional, multi-media fate model termed Multi-media Urban Model (MUM-Fate) was used to predict outdoor soil, outdoor air, indoor, and residential dust concentrations of PBDEs. The results were combined with measured concentrations from food and mother's milk to determine potential exposures of PBDEs in this complex exercise. Exposure scenarios included the following: elevated indoor sources, fish eater, occupational exposure (from an electronics recycling plant), and exposures experienced by four younger age-ranged individuals (0-6 months), toddler (6 months–4 years), child (5-11 years), and teen (12-19 years). They modeled "total PBDE," which included 47, 99, 100, 153, and 154. BDE 209 was excluded due to a lack of data in most media. Their results suggested first that 100-422 g/day of PBDEs were released into the 470 km^2 modeled area; these emission rates resulted in modeled air concentrations which matched measured concentrations. Their results suggest a range of average daily intake from all sources to be 155 ng/day for the adult to 1,965 ng/day for the infant (2 to 280 ng/kg bw). Nearly 100 ng/day of this adult exposure is modeled to come from soil/dust exposures, mainly indoor dust ingestion (dermal and inhalation minimal). For toddlers, over 90% of their daily intake of 264 ng/day comes from dust ingestion. Following household dust, "dairy, meat, and eggs" exposure contributed 16% of total exposures. Other exposure estimates include 227 ng/day for the fish eater and 2,190 ng/day for the occupational exposure. While the finding that house dust contributed the most to human exposure for all scenarios, this has to be considered carefully in light of the data input into the exercise, particularly the food concentrations. It is noted that their assumption of total concentration for dairy, meat, and eggs was 101 pg/g wwt, while Schecter et al. (2006b) found that total BDE ranged from 39 ppt wwt to 1426 ppt wwt in

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18 meat samples collected in Texas. Therefore, it seems quite possible that the food concentrations were lower than would be assumed had the estimates been developed for United States conditions.

McDonald (2005) developed a dose estimate starting from body burdens and working backward using pharmacokinetic (PK) modeling. Examining 6 studies that evaluated PBDE body burdens in individual women (serum, milk, and adipose tissue studies), McDonald found that the median concentration was about 48 ng/g lwt, a mean of 90 ng/g lwt, and a 95% of 302 ng/g lwt. These totals were the sum of congeners 47, 99, 100, 153, and 154. McDonald used a simple first-order, single compartment (lipid compartment) PK model to determine the dose required to obtain congener specific body burdens. He assumed congener-specific adsorptions ranging from 0.78–0.94. Congener-specific half-lives were based on rat data and a correlation between rat and human half-lives, and he came up with half-lives of 3 yrs for BDE 47, 5.4 yr for BDE 99, 2.9 yrs for BDE 100, 11.7 yrs for BDE 153, and 5.8 yrs for BDE 154. The estimated total dose of PBDEs (sum of 5 congeners) was 8.5, 16.0, and 53.6 ng/kg-d for the median, the mean, and the 95% concentrations, respectively. He went further to evaluate this dose level in terms of potential for health impact, based on rat testing. He found that rodent-to-human body burden concentrations of concern were < 1 for alterations of male and female reproductive organs in rats, < 10 for neurodevelopmental effects in mice, and <100 for neurodevelopmental effects in rats. They also looked at other intake studies and noted that an intake estimate of 0.4–11 ng/kg-day was derived in a forward manner in another United States study on food intakes only.

Webster et al. (2005) conducted a monte carlo exercise on exposures to BDE 47 using concentrations of this congener in air, food, dust, and using traditional exposure factors from the EPA's *Exposure Factor's Handbook* (EPA, 1997). They derived estimates of exposure dose (in ng/kg-day) to infants, young children, and adults. Then, using a simple 1-compartment, steady-state approach and assuming a 4-year half-life, they estimated body burdens resulting from median doses and compared that to some tissue data. From their monte carlo simulations, the mean dose to infants, children, and adults were 123.9, 7.7, and 0.9 ng/kg-day, respectively. The infant dose was dominated by breast milk ingestion, explaining 95% of dose. The children's dose was dominated by the dust-related exposures of dermal (35%) and dust ingestion (36%), with diet being the

third most important (28%). For the adult, diet dominated at (63%), while dust ingestion (23%) and dermal (11%) comprised a significant portion of the remainng exposures. Several uncertainties were identified: absorption fractions, concentrations in exposure media, and so on. Based on the median adult dose (0.8 ng/kg-day), the steady-state lipid concentration was predicted to be 5 ng/g lwt. Assuming breast milk lipid concentrations were similar to overall body lipid concentrations, this was lower than the average BDE concentration found in other United States studies, which the author claimed to average about 20 ng/g lwt. The authors suggested possible issues with exposure dose and PK modeling assumptions (4-year half-life too short).

She et al. (2005) used the ratio of BDE 99 to BDE 47 (99/47) as a way of understanding the possible sources of exposure to humans. The study evaluated data from numerous studies which provided data to develop ratios in the penta formulation, in house dust, and in mother's milk, and made observations in each. For the penta formulation, BDEs 99 and 47 are present at about the same amount, 40% each, with BDE 100 at 7%. The ratio of 99/47 in United States house dust ranged from 0.5 to 2.0, with an average ratio of about 1, which would be consistent with the penta BDE formulation with use information. This suggests the widespread use of penta in North America. The variation in the ratio could be due to debromination and different rates of volatilization between the congeners, but overall, United States dust appears consistent with expectations. United Kingdom dust showed high levels of BDE 209, which is consistent with the deca formulation. Mother's milk, on the other hand, showed a 99/47 ratio of 0.2 to 0.3 in three studies, with the highest levels in milk to have a ratio more like 0.6. Their explanations and discussions focused on these points: 1) BDE 47 is more bioaccumulative than BDE 99, so that alone could lead to a downward shift in the ratio (i.e., more prominence of the more bioaccumulative BDE 47 in mother's milk), 2) this bioaccumulation trend extends not only to direct dust exposures by the mother, but maybe more importantly to exposures by animals and then exposures by humans by animal food products, 3) this latter point suggests that on average, a 0.2-0.3 ratio would suggest a predominant pathway by food, and 4) the higher mother's milk concentrations, when the ratio was more like 0.6, might suggest that dust is an important pathway as well (because dust is a direct exposure with one bioaccumulation step, while food is a secondary pathway).

The highest estimates of exposure dose, on a body weight, are for infants via breast milk. Schecter et al. (2005) calculated infant intakes to be 307 ng/kg-d, dominated by BDEs 47 at 169 ng/kg-d, followed by BDE 99 at 49 ng/kg-d, and BDE 100 at 36 ng/kg-d. As noted above, Webster et al. (2005) calculated an intake of 123.9 ng/kg-day for BDE 47 alone, and Jones-Otazo et al. (2005) modeled an intake of 280 ng/kg-day for total BDEs for the infant ages 0–6 months. The lowest intake estimates were for breast-feeding infants in China, where Meng et al. (2007) calculated a median intake of total BDEs to be about 6–7 ng/kg-day (ages 0 – 1 year).

In summary, some of the key observations from exposure and exposure dose estimation include:

1) on a body weight basis, adult exposures for the dietary pathway alone in the United States appear to be estimated in the range of 0.5–2.0 ng/kg-day;

2) although no United States studies have estimated inhalation exposures, estimates for the UK, Canada, and one study in Kuwait suggest much lower exposures in the range of 0.4 – > 4.0 ng/day (on a body weight basis assuming a 70 kg adult, 4 ng/day = 0.06 ng/kg-day);

3) adult exposures to dust have been estimated to be higher than this range, perhaps more like 3.0 – 6.0 ng/kg-day; these estimates are made based on vacuum dust sampling combined with dust ingestion rates of 100 mg/day;

4) by these two findings, the suggestion is that dust would dominate adult exposures, and in fact, the majority of studies looking at multiple pathways conclude that dust exposures may dominate. However, there is some uncertainty in the literature on this point. One study using monte carlo techniques found that adult exposures to BDE 47 were dominated by diet, 63%, as compared to dust, 23%, and dermal contact with dust, 11% (this was the only study found which looked at dermal impacts). Another study assumed indoor dust ingestion was < 1 mg/day, so that this pathway resulted only in a BDE intake of about 3 ng/day;

5) there seems to be uniform agreement that child and toddler exposures are higher than adult on a body weight basis, > 7 ng/kg-day, and are dominated by dust exposures;

6) infant exposures are dominated by breast milk ingestion, with body weight-based exposures above 100 ng/kg-day, with one estimate over 300 ng/kg-day.

5.4. ESTIMATES OF BACKGROUND INTAKES OF PBDES FOR ADULTS

The procedures to estimate individual intakes of PBDEs for adults were developed in a manner similar to that done in the USEPA Dioxin Reassessment (EPA, 2003), with one important addition, as will be described shortly. Intakes are a function of contact rates in combination with exposure media concentrations. As in the Dioxin Reassessment, intakes are defined as the amounts of contaminants crossing the body boundary but not yet absorbed into the blood stream. For this reason, absorption fractions are not applied to inhalation and ingestion intakes; they are later applied to estimate body burden in the use of the pharmacokinetic model. An absorption fraction was, however, used for the dermal contact pathway, as this will result in the estimation of an amount crossing the skin boundary. This amount is considered "absorbed" and no further absorption fraction is required for this dermal dose. Table 4-5 provides the exposure media concentrations that were developed Chapter 4, and Table 5-5 in this chapter provides the contact rates, and for dermal exposure, the full dermal contact algorithm.

The important addition to the procedures originally laid out for dioxin-like compounds, applied here to PBDEs, relates to the importance of the indoor versus the outdoor environment. The Dioxin Reassessment used air concentrations from outdoor ambient air measurements for the inhalation pathway, and measurements of dioxins in background soils for soil ingestion and soil dermal contact. In the case of dioxins, the primary sources are emissions from combustion sources into the open environment, with subsequent accumulation in outdoor soils and, of primary importance to dioxin exposure, in the terrestrial and aquatic food chains. In contrast, the primary cause for PBDE exposures are their use in commercial products that are part of the indoor environment (PC circuitry, foam cushions, etc), and, as described in Chapter 4, indoor air and indoor dust concentrations of PBDEs are orders of magnitude higher than outdoor air and soil.

Therefore, the use of outdoor measurements in air and soil does not appear appropriate for inhalation and soil/dust pathways for PBDEs. Sjodin et al. (2004b) and Stapleton et al. (2005, 2008b) recognized the importance of exposure to indoor dust when calculating dust ingestion intakes in the hundreds to thousands of ng/day total PBDEs for adults and children. Their intake calculations used "soil ingestion" contact rates, applying them in total to their indoor dust measurements, as though the entire contact from "soil" is, in fact, from "indoor dust." In contrast to their calculations of dust exposure intakes in the 100s of ng/day, their diet intakes (described in the previous section) were always under 100 ng/day total PBDEs. This shows the importance of exposures to indoor dust and indoor air.

The approach taken in this assessment is to estimate a weighted average concentration of "dust/soil" and air, which, in theory, considers what portion of total soil ingestion/dermal contact and inhalation comes from indoor dust and indoor air. The surrogate used to estimate this portion will be "time spent indoors." The *Exposure Factors Handbook* (EPA, 1977) provides tables on hours/day spent indoors, and for adults the recommended number of hours per day is 21, which is 87.5% (or, expressed as a fraction, 0.875) of the time. Therefore, a weighted average concentration, C[avg], of "soil/dust" and air that will be used in the adult soil ingestion, soil dermal contact, and inhalation pathways is C(indoor dust/indoor air) * 0.875 + C(outdoor soil/outdoor air) * 0.125. This approach simplistically assumes that exposures are proportional to time indoors versus outdoors, which could be totally incorrect for soil pathways if, in fact, the actual exposures to dust/soil all were to take place outdoors. In contrast, this is a reasonable approach for inhalation, for obvious reasons. According to the *Exposure Factors Handbook*, children under 11 years of age spend 19 hours per day indoors, so their fraction indoors will be 0.792. Children ages 12 and higher spend the adult number of 21 hours per day indoors.

Table 5-5 provides all of the exposure parameters and a brief description of the pathways. Table 5-6 provides the final adult intake estimates for the 13 PBDEs for which environmental media concentrations were derived in Chapter 4. The total dose to adults was 547 ng/day, or on a body weight basis assuming a 70-kg adult, 7.8 ng/kg-day. The following are key observations from Table 5-6, and some accompanying discussion:

1) Predominance of the Dust Pathways: Given the procedures and parameters in Table 5-5, coupled with the media concentrations provided in Table 4-5, it would appear that the pathways of dust ingestion and dust dermal contact overwhelm the exposure of adults to PBDEs. Dust ingestion and dermal contact together account for 82% of the total exposure. The estimate of 363 ng/day of PBDEs via dust ingestion is consistent with Sjodin et al. (2004b), who calculated an intake of 400 ng/day assuming a dust ingestion rate of 100 mg/day and comparable PBDE dust concentrations as were used in this exercise. It is higher than the median estimate of 154 ng/day for adults estimated by Stapleton et al. (2008b) based on measurements of PBDEs on hands and then an empirical model to calculate intakes. The estimate of the PBDE intake by dust ingestion is linearly related to the dust ingestion rate and the PBDE concentration. Therefore, use of a lower rate of dust ingestion, such as the 0.56 mg/day used by Stapleton et al (2005), would lead to a much lower ingestion of PBDE by the dust ingestion pathway. Further discussions on the uncertainty of this pathway are provided in Section 5.6 below.

2) Prevalence, or Lack Thereof, of Food Exposures: After dust exposures, about 18% of exposures are due to inhalation and food/water ingestion. In contrast to this finding, the literature has an abundance of dietary surveys of PBDEs and discussions on exposure via food consumption. A significant portion of this literature deals with food exposures in Europe and Asia. Studies on indoor dust concentrations for locations outside of the United States tend to show lower concentrations of PBDEs (except for BDE 209; see discussions in Chapter 4), and so for Europe and Asia, food very likely dominates overall exposure. For the United States, the literature is now beginning to recognize the importance of the dust pathway and much more work on dust exposures has occurred since about 2005 (see summaries in Section 5.3).

3) Distribution of Dose Among BDE Congeners: The percentage of total dose attributed to BDEs 47, 99, and 209 are about equal at 25%, 28%, and 27%, respectively, followed by BDE 100 at 11%, for a total of 91% among those four congeners. Needless to say, dust-related exposures dominated for the individual congeners, but mostly for BDE 209, where dust ingestion and dust dermal contact explained 88% of the dose, while it explained 68% of exposure to BDE 47 and 82% to BDE 99. Exposures to BDEs 138, 153, and 154 were all low at between 2-3% of total. BDE 183, generally considered to be a marker for the presence of the octa PBDE formulation, although it could be present in

environmental media as a result of debromination of higher brominated BDEs such as BDE 209, was a small contributor to overall dose, at 1%. Although at least some measurements were made of all the congeners on Table 5-6, it would appear that from a dose perspective, perhaps BDE congeners 17, 28, 66, 197, and 206 can be neglected. However, from a body burden perspective, BDE 28 makes up between 2-4% of total body burden, while BDE 138, which makes up 2% of the dose, is virtually absent in body tissues. This exemplifies the importance of the interplay between dose and body burden when understanding and quantifying exposure to this class of compounds.

5.5. CONVERTING ADULT INTAKE DOSE TO BODY BURDEN

The intake doses in the previous section will be converted to body burdens in this section. Assuming first-order kinetics and that PBDEs accumulate in body lipids, the equation for the change in lipid concentrations over time is

$$\delta C_{BDE}/\delta t = (D_{BDE}(t)*ABS_{BDE})/BL(t) - k*C_{BDE}(t) \qquad (2)$$

where

C_{BDE} is the congener-specific lipid-based concentration over time (ng/g lwt)

D_{BDE} is the daily dose of BDE (ng/day)

ABS_{BDE} is the congener-specific and route-specific absorption fraction

$BL(t)$ is the body lipid mass over time (g)

k is the first-order elimination rate of the congener in the body (day^{-1}).

As presented here, k is assumed to be a constant, but it too could vary over time. The solution to this partial differential equation is

$$C_{BDE}(t) = C_{BDE}(0) * e^{(-kt)} + [(D_{BDE}(t) * ABS_{BDE}) / BL(t)] * [(1 - e^{-kt})/k] \qquad (3)$$

where

$C_{BDE}(0)$ is the initial body burden at time 0

Assuming a constant BL and a constant dose over time, the steady state lipid concentration (ie., when t approaches infinity) is easily calculated as:

$$C_{BDE} = (D_{BDE} * ABS_{BDE})/(k * BL) \qquad (4)$$

Equation (4) is used here to estimate an adult body burden, using the congener-specific adult doses provided in Table 5-6. It is assumed that bodies are 25% lipid, leading to a BL value of 17,500 g. Estimates of absorption for other POPs (dioxins and PCBs) when ingested on soil were in the range of 0.30 to 0.70, and it was noted that dioxins might be more bioavailable on house dust as compared to soil (Paustenbach et al., 1997, 2006; ATSDR, 2004). Huwe et al. (2008) studied the retention and excretion of BDE congeners administered to male rats in corn oil and household dust (NIST reference material). The rats were dosed at a "high" dose of 6 μg/kg-day or a "low" dose of 1 μg/kg-day. They were fed this amount for 21 days and then killed 24 hrs after the last feeding. Fifteen BDE congeners were measured in adipose tissue and liver, and feces collected during the experiment were also measured to provide a mass balance. By calculating the amounts "retained" from the dose in the body by this mass balance, they could surmise absorption by this amount retained. The retention amounts for the high dose group exposed to PBDEs in dust, expressed as a fraction of total exposure, ranged from 0.04 (for BDE 209) to 0.78 for BDE 100. For key congeners BDE 47, 99, 138, 153, 154, and 183, the fractions retained were 0.69, 0.44, 0.67, 0.73, 0.19, and 0.48, respectively. Results were similar for corn oil and for the lower exposure amounts. Although not ideal because it is not human data (although human data are understandably rare) and it is not corroborated else, these values will be used in the modeling of this study for congener-specific values of ABS. McDonald (2005) assumed absorption fractions of 0.78 to 0.94 for BDEs 47, 99, 100, 153, and 154 based on experiments with rats, and the values he used will be used for these congeners for all other pathways in this study, including inhalation and water/food ingestion. There is no additional absorption assumed for dermal contact since an absorption fraction was already included in the dose estimates for that pathway. McDonald (2005) also cited Geyer et al. (2004) to assign half-lives of 2.9 to 11.0 yr to this same group of congeners, and these values will be used here. Thuresson et al. (2006) provided estimates of 0.26 yr (94 d) for BDE 183 and 0.041

yr (15 d) for BDE 209, and these will be used here. The first-order elimination rates, k_{BDE}, are easily calculated as 0.693/hl (where hl = half-life). For lack of better information, average values of absorption and half-life, 0.90 and 6 years, respectively, will be assigned to congeners BDE 28 and 138, two other congeners which are present in exposure media and in measurements of blood or milk. BDE congeners 17, 66, 85, 197, and 206 will be neglected in this example as they are rarely found in exposure media or body measurements. The final set of doses, pharmacokinetic parameters, predicted lipid-based concentrations using these parameters in Equation (3) above, and then the observed concentrations of the 9 PBDE congeners are shown in Table 5-7.

Overall, the total concentration was predicted at 35.9 ng/g lwt, while it was observed at 36.3 ng/g lwt in blood and 44.1 ng/g lwt in milk. Predictions appear reasonably close to measurements for 6 of the 8 congeners (for which there are predictions and at least one observation). The prediction of BDE 47 at 10.0 ng/g lwt does not appear to match well with the observed measurements of 20.5 ng/g lwt in blood and 26.0 ng/g lwt in milk. Conversely, the prediction of BDE 99 is higher than both measurements, with a prediction of 14.6 ng/g lwt, while it was found at 5.0 and 5.4 ng/g lwt in blood and milk, respectively. Otherwise, BDE 100 was predicted at 4.2 ng/g lwt while it was found at 3.9 and 5.2 ng/g lwt. BDE 153 was predicted at 4.6 ng/g lwt, while it was found at 5.7 and 4.8 ng/g lwt.

Obviously there are uncertainties with use of this pharmacokinetic model and the assignment of parameters; these are discussed below in Section 5.6. Even with uncertainties, one can make some valid and important observations about exposure of Americans to PBDEs using this framework. For one, indoor exposures of soil/dust ingestion, dermal contact, and inhalation dominate total exposures. While this might be a valid observation based on intake estimates alone, it is strengthened significantly by this simple pharmacokinetic exercise. If one calculates body burdens of these nine congeners based on water/food ingestion alone (the dose is estimated at 92 ng/day or 1.3 ng/kg-day with a body weight of 70 kg), the body burden is modeled to be 10 ng/g lwt, which is much less than the observed medians of 36 and 44 ng/g lwt in blood and breast milk. With 23.8 ng/g lwt predicted to occur from soil/dust ingestion, dermal contact, and inhalation, the suggestion is that 70% or more of the United States body burden could be explained by non-food exposures occurring in the indoor environment.

A second observation is that while there may be significant exposure to BDE 209, it does not appear to be showing up in large concentrations in adults, although it might be a different story for children, as discussed below. The calculated intake dose is 27% of the total, equal essentially to intakes of BDEs 47 and 99 (calculated at 25 and 28%, respectively). However, because of a rapid half-life in the body (15 days), tissue levels are predicted to be as low as 0.05 ng/g lwt, which is numerically close to the observed 0.4 ng/g lwt in breast milk (some blood studies other than NHANES quantified BDE 209 in blood, also, at around 0.1 ng/g lwt). Said another way, concentrations of BDE 209 are much lower in both observed measurements and predicted concentrations than the 5–30 ng/g lwt found and predicted for BDEs 47 and 99.

However, to observe that BDE 209 has not been quantified at concentrations approaching that of BDEs 47 and 99 in blood lipid may not be fully informative. The presence of lower brominated BDE congeners in blood could have resulted from debromination of BDE 209. Also, it may be that BDE 209 is not as lipophilic as other BDEs. Measuring BDE 209 in extracted blood lipids might underestimate its presence in blood because some BDE 209 may be present in the unanalyzed portion of a blood sample.

5.6. EXPOSURE OF SPECIAL POPULATIONS OF INTEREST TO PBDES

This section discusses exposures of special populations to PBDEs, and develops intake and body burden estimates for some of the populations. Specifically, this section provides an overview of body burden studies on occupational populations, it derives intake estimates and resulting body burden estimates for infants, it derives intake estimates for children within specific age ranges, and it discusses an important observation that there appears to be a proportion of individuals at the high end of the general population who are experiences significantly higher exposures than the remaining general population.

5.6.1 Impacts to Infants from Consumption of Breast Milk

Using the profile of PBDEs in mother's milk, the dose to the infant was modeled as follows:

$$D = C * f * IR \qquad (1)$$

where D is the ingested dose of PBDEs (ng/day)

C is the concentration in milk fat (ng/g lwt)

f is the fraction of fat in breast milk

IR is the ingestion rate of breast milk (g whole weight/d)

The dose term, D, can easily be converted to a body-weight-based dose term by dividing by infant body weight, BW. The rate of ingestion of mother's milk and the fraction of fat in the mother's milk were assumed to be constant over the duration of breast-feeding. Smith (1987) reported that studies in Britain and Houston found that the breast milk ingestion rate for 7- to 8-month-old infants ranged from 677 to 922 mL/d and 723 to 751 mL/d, respectively, and that breast milk ingestion rates remain relatively constant over an infant's life. Smith (1987) also assumed that mother's milk has a 4% fat content. These assumptions were adopted for the purposes of the modeling exercise described here: IR = 800 g/d (which assumes 1 L milk weighs 1 kg) and f = 0.04. Given the total PBDE concentration of 44.1 ng/g lwt in mother's milk (see Table 5-7), the total dose to infants is 1,411 ng/day. Assuming an average body weight of 10 kg for an infant during the months of breast-feeding, a dose is calculated as 141 ng/kg-day. This is about half the intake as the 307 ng/kg-day estimated by Schecter et al. (2005).

Infant impacts to breast milk and children's body burdens were handled in a different manner than adult body burdens. The approach mirrors what was done for dioxins by Lorber and Phillips (2002), who modeled the impact of dioxin-like compounds in infants resulting from consumption of breast milk. The procedures in Lorber and Phillips (2002), as applied to PBDEs instead of dioxin toxic equivalents (TEQs), include the following: 1) total PBDEs were modeled instead of individual congeners; one half-life (variable as noted below) and absorption (constant at 0.80) were used to characterize this surrogate measure of exposure; 2) the dynamic solution to Equation 2 above, shown in Equation 3, was used to be able to characterize changes in dose, elimination half-life, body lipid fractions, and body weight over time, and 3) the elimination half-life for total PBDEs in infants will be more rapid than had been assumed for individual congeners for adults.

Lorber and Phillips (2002) cited the pharmacokinetic modeling work of Kreuzer et al. (1997) in their assignment of the overall elimination rate for dioxin TEQs from

infancy into childhood. Kreuzer et al. (1997) found that the overall elimination rate of 2,3,7,8-TCDD in infants was driven by the non-metabolic process of fecal elimination. Specifically, the overall elimination half-life of 2,3,7,8-TCDD was modeled to be about 0.4 yrs at birth, compared to 5 years or more at adulthood, because of the magnitude of lipid loss in fecal elimination in infants. Since 2,3,7,8-TCDD accumulates in lipids (as do the PBDEs), the rapid loss of lipids via fecal elimination results in a comparable rapid loss in 2,3,7,8-TCDD. Lorber and Phillips (2002) assumed this intial, rapid half-life at birth would rise to a half-life of 5 years by 18 years of age. They verified their approach by showing how infant body burdens of TEQs were predicted to be much closer to measurements in the literature when assuming this rapid half-life as compared to assuming the longer half-life that would be appropriate for adults. Similar data are not available for PBDEs, but given the similar lipophilicity of PBDEs and CDD/Fs, this approach seems reasonable. Specifically, this same half-life profile used in Lorber and Phillips (2002) will be used here: a rapid half-life at birth rising to an overall, representative half-life of 6 years by age 11. Drawing on information in the *Exposure Factors Handbook* (EPA, 1997), Lorber and Phillips (2002) also assigned temporally varying body lipid contents and body weights. Absorption of PBDEs will be assumed to be 80% (absorption fraction = 0.8), and the initial body burden at birth will be 37 ng/g lwt, similar to the adult total body burden from blood measurements. Derivation of intake dose was provided above, and it was 1,411 ng total PBDE/day for one year of breast feeding, followed by child intakes from 1 year on (see Section 5.6.2 below). Table 5-8 shows the final PK parameters, including dose estimates, from birth until age 19. Figure 5-2 shows the final results of this exercise, including the impact on body burden assuming the overall half-life of 6 years.

With the assumption of more rapid elimination earlier in life, the infant body burden rises to about 125 ng/g lwt at age 1 and continues to rise to above 200 ng/g lwt through age 5. At this point the concentration begins to drop, ultimately decreasing to 100 ng/g lwt by age 19. If total BDEs had an overall half-life of 6 yrs from birth on, than the body burden would rise to near 200 ng/g lwt by age 1, and continue to rise to about 325 ng/g lwt by age 5, only then to slowly dissipate to levels below 100 ng/g lwt by age 19.

The validity of these predictions cannot be easily verified because of the lack of data in the literature. However, there is one study described above in Section 4.2 on blood levels of four individuals within a family in California. Fischer et al. (2006) present data on the two parents, a 35 and 37 year old, a 5-year-old daughter, and an 18-month-old son in September and December of 2004. The sum of BDEs 47, 99, 100,153, 154, and 209 in the parents ranged between 64 and 147 ng/g lwt in the two sampling dates, but the concentrations in the children were much higher. The 5 year-old daughter had concentrations of 237, 239/249 ng/g lwt (the last two were duplicates of the same December sample) of the 5 congeners for the September and then December samples. The toddler had the highest concentrations of all: 418 and 488/476 for the five congeners. Also of interest were very high initial concentrations of BDE 209, which dropped significantly in both the 5 year old and the toddler: the 5 year old had concentrations of 143 ng/g lwt of BDE 209 in the September sample and 9/12 ng/g lwt in the December sample. The toddler had 233 and 19/26 ng/g lwt in the September and December samples, respectively. Discounting laboratory error, the authors attribute the higher concentrations in the children to exposure to house dust, and the drop in 209 levels between the September and December samples to the short half-life of BDE 209 in humans. While the authors have discounted laboratory error, it would appear that a decline by an order of magnitude in both the toddler and infant is substantial, and could be due to some difference in the two laboratories. However, a decline of this magnitude is plausible because of the short half-life of BDE 209 in humans, as noted by the authors. The higher levels of the other congeners in the toddler were attributed by Fisher to consumption of breast milk, although it could also be due to exposure to house dust. These high measurements in children support the simple PK modeling done here, but are not sufficient to verify it. Body burden measurements in children remain an uncertainty, although the modeling and this single family provide some evidence that body burdens in children are higher than adults, with the suggestion that they could be very high. Further, the analysis here suggests that these higher levels could persist through childhood into early adulthood.

5.6.2. Childhood Intakes

The total dose to adults was 547 ng/day, or 7.8 ng/kg-d (assuming an adult body weight of 70 kg). Using the intake rates provided in Table 5.5 and the exposure media concentrations developed in Chapter 4 (shown in Table 4.5), the total dose for the three age ranges of children were as follows: 751 ng/day for the 1-5 age range, 439 ng/day for the 6-11 range, and 536 ng/day for the 12-19 age range. On a body weight basis, the doses are 50.1 ng/kg-d for ages 1-5 (assuming 15 kg bw), 14.6 ng/kg-d for 6-11 (30 kg), and 9.2 ng/kg-d for 12-19 (58 kg). The much higher dose for the child age 1-5 was due to the doubling of soil/dust ingestion from 50 mg/day to 100 mg/day. Otherwise, the trends as elaborated above for adults in Section 5.4, such as the predominance of the soil ingestion and dermal contact pathways, were similar for children.

5.6.3. Body Burden Data to Characterize Occupational Exposures

Limited studies from Sweden and from China suggest that PBDE concentrations are elevated in occupational groups exposed to likely sources of PBDEs. One study, which looked at incinerator workers in comparison to general population exposures, did not find a difference. The only study of occupational exposures in the United States found a significantly higher (p < 0.05) level of PBDEs in workers in foam recycling facilities and individuals who installed carpet padding manufactured from recycled foam.

This study from the United States (Stapleton et al., 2008a) included 12 foam workers from two foam recycling facilities (one in Maryland and one in California), 3 carpet layers who worked in association with the California facility, and 5 control group individuals comprised of spouses and clerical workers from the facilities. The median total PBDE concentrations (comprised of BDEs 17, 28, 47, 66, 99, 100, 153, 154, and 183) in the foam workers, carpet installers, and control were 160, 178, and 19 ng/g lipid, respectively. The body burdens of the workers were dominated by BDE 47, which explained 50-60% of the total concentration, followed by 99 and 153, which both contributed 13-20% of the total.

Thirteen PBDE congeners were quantified in serum in a group of 19 PC technicians (PC techs), and the results were compared against hospital workers, and PC clerks in Sweden (Jakobbson et al., 2002). Moreover, within these two comparison groups, refined characterizations included women who had never breast fed (NBF), hospital cleaners (HC), and computer clerks (PCC). Results were provided for BDEs 47,

153, 154, 183, and 209. There were distinct differences among the groups. The median value of BDE 47 was fairly similar among the groups: PC techs had a median of 1.3 ng/g lwt, HCs had a median of 1.6 ng/g lwt, PCCs had a median of 1.5 ng/g lwt, and NBFs had a median of 2.1 ng/g lwt. BDE 153 was the highest in the PC techs, and significantly higher than the other three groups: 2.6 ng/g lwt for the PC techs, while it was 0.6, 0.8, and 0.8 ng/g lwt for the other three groups. While these other three groups did not have BDE 209, it was quantified at a median of 1.5 ng/g lwt for the PC techs. There were no correlations with age and BDE levels, but a correlation was found between time on the job and BDE 153 concentrations in the PC techs.

Eight Swedish employees at a recycling plant and 4 rubber mixers volunteered to donate blood samples during their summer vacations, in 1998 and 2000, respectively (Jakobsson et al., 2003). The first blood sample was drawn at start of vacation, 3-4 days later, and then at additional time periods during the 4-5 week vacation. The samples were analyzed for BDEs 47, 153, 183, and 209. Assuming there was a constant baseline of BDEs in these workers, which they obtained from serum measurements on non-exposed individuals, the authors calculated half-lives. In addition to these 8 temporally followed workers, the authors measured blood from 60 other subjects; in all, their study had 107 observations from 68 subjects. They found distinct exposure levels and patterns of BDE congeners as a function of which workers they measured. The rubber workers, who were exposed only to decaBDE, had markedly elevated levels of BDE 209, which averaged 27–35 ng/g lwt, with a high of 278 ng/g lwt. Unexposed individuals had levels at ND-2.4 ng/g lwt. The electronic dismantlers were expected to be exposed to all BDE congeners, and they were, with concentrations of BDEs 47, 153, 183, and 209 averaging 2.9, 4.5, 7.9, and 4.8 ng/g lwt for 47, 153, 183, and 209. While they found that BDE 209 is bioavailable based on finding high levels in an occupationally exposed group (the rubber workers), they also found it had the shortest half-life. There was, overall, an inverse relationship between half-life and degree of bromination–lower brominated congeners had longer half-lives: BDE 203 was 37 days, BDE 183 was 110 days, BDE 153 was 680 days, and BDE 154 was 270 days.

Qu et al. (2007) measured levels of 14 BDEs (including BDE 209) in electronic waste dismantling workers, in residents living within 50 km of the dismantling area, and a reference group with no exposure near an occupational setting in Quongdong, South

China. The median concentrations of each congener were determined, and the sum of these individual medians from the three groups were as follows: 126 ng/g lwt (dismantling workers), 35.1 ng/g lwt (residents living within 50 km), and 9.4 ng/g lwt (reference group). BDE 209 was the highest among all groups, explaining between 50 and 70% of total concentration. Interestingly, BDE 207 was the second highest in the electronic recycling group and the residents living within 50 km, explaining 8 and 15% of the total, respectively. Exceedingly high concentrations were found in one 18-year-old male electronic waste worker: BDEs 28, 183, 208, and 209 were found at 148.3, 60.2, 66.2, and 3,436 ng/g lwt, respectively.

Lee et al. (2007) measured 13 congeners (not including BDE 209) in 92 blood samples, including 30 from incinerator workers, 51 from nearby residents, and 11 from controls in 2001 and 2002. The average total concentration was 16.84 ng/g lwt, and there was only a slight difference between the incinerator workers, who had the highest concentrations at an average of 19.24 ng/g lwt, and the other two groups: residents at 15.22 ng/g lwt and controls at 17.74 ng/g lwt. The difference between the groups was not significant, and there was no other correlations found, including to age, weight, dietary habits, and others, with the exception of sex: males had 15% higher levels compared to women. BDE 47 dominated the profile, explaining 33% of the profile, followed by BDEs 153 (24%), BDE 183 (17%), BDE 99 (15%), and BDE 100 (7%).

5.6.4. Elevated Exposures at the High End of the General Adult Population

The analysis of the 2003/2004 NHANES blood concentration data described by Sjodin et al. (2008), shown in Table 5.7 and used as a primary comparison to the predicted concentration in the model, did not include statistics on total concentration, just statistics on individual congeners. The congener-specific median concentrations are shown in Table 5.7, and, when added together, they lead to a total concentration of about 36 ng/g lwt. The statistics provided for individual congeners show that the 90th percentile concentration of all the key congeners in United States citizens are about 4-6 times higher than the 50th percentile and the 95th is near or more than 10 times higher than the medians of the congeners. Perhaps more noteworthy was the finding that the highest total concentration found in an individual in NHANES 2003/2004 was 3680 ng/g lipid, which included BDE 47 at 2350 ng/g lipid (Sjodin et al., 2008). This highest individual

had concentrations about 100 times higher than the median in the population. Clearly, there are individuals with very much higher concentrations than the central tendency median selected for the point estimate exercise of this chapter.

In comparison, this is not the same trend as generally found for dioxin. Ferriby et al (2006) statistically evaluated the concentrations of polychlorinated dibenzo-p-dioxin (PCDD) and dibenzofuran (PCDF; the combination abbreviated PCDD/F) concentrations from NHANES 2001/2002. Determining the toxic equivalent (TEQ) concentrations of the 17 toxic PCDD/F congeners for individuals in the survey, they provided population statistics for PCDD/F TEQ concentrations. The median concentration in the population was 14.4 pg/g lwt, the 95% was 45.2 ng/g lwt, and the maximum found was 139.2 ng/g lwt. In this case, the maximum found was 10 times and not 100 times the median. To illustrate the difference in these NHANES results, Figure 5.3 shows the comparison of NHANES percentiles for PCDD/F TEQ and BDE 47 findings at the 25%, 50%, 75%, 95%, compared to the maximum found in the NHANES surveys (dioxin data from Ferriby et al., 2006, and PBDE data from Sjodin et al., 2008). While the percentiles of dioxin concentrations within the general adult population, expressed as a fraction of the maximum found, range from between about 0.10 at the 50th % and 0.30 at the 95th % for PCDD/Fs, they range much more narrowly at <0.01 at the 50th % to 0.07 at the 95% for BDE 47.

Other studies show a similar disparity between the median (or geometric mean, if that is what the study authors provided) and high value of their body burden measurement study. For example, Moreland et al. (2005) measured the blood of 93 individuals, 79 of which were anglers. The highest congener found was BDE 47, at a geometric mean of 13.3 ng/g lwt, followed by BDE 99, at 3.2 ng/g lwt. Although they did not report total concentrations, BDE 47 was found at a high of 1,388 ng/g lwt, which was 100 times the geometric mean for this congener, and the high BDE 99 concentration was 546 ng/g lwt, about 20 times the geometric mean. Petreas et al (2003) measured BDE 47 in adipose tissue from 32 women in the San Francisco area, and found a median of 16.5 ng/g lwt, with a high of 510 ng/g lwt, 30 times the median.

Evidence suggests that these elevated exposures at the very high end of the population could very well be due to dust exposures. Nearly every study that has been conducted measuring dust concentrations from different locations very similarly finds a

reasonably log normal range until the very last few samples which are substantially higher than the rest of the population. Following is a bullet summary showing this trend from several dust studies:

a) Stapleton et al (2008b) measured BDEs on hands using hand-wipes. Their results and exposure estimates were described earlier in Section 5.3. The key finding of note for this discussion is that they measured BDEs in 33 individuals, and that for 32 of them, the measurement was under 500 ng/hand, but the 33rd individual had about 2000 ng on his hand. The median from this population was 128 ng/hand.

b) Allen et al (2008) collected 108 bulk dust samples from about 20 homes in the Boston, MA area, during two sampling events in 2006. Samples were characterized as having come from the "living room", "bedroom", and "vacuum bag" (meaning location unspecified). The geometric mean total concentrations found in the living room, bedroom, and vacuum bag were13,732; 6,255; and 4,269 ng/g dwt, respectively. The two highest total concentrations were 544,000 and 269,000 ng/g dwt, found in vacuum bag samples from a single individual's home. The first high measurement was dominated by BDE 209, explaining 97% of the 544,000 ng/g dwt found; the dominant congener was not identified for the second highest sample.

c) Harrad et al (2008) measured 78 samples in four cities in Canada, New Zealand, the United Kingdom, and the United States. The median total concentration of 28 samples from the United Kingdom was 2,900 ng/g dwt, but one sample was found at 520,000 ng/g dwt, with essentially all of it (>99%) explained by BDE 209. In 20 United States samples (from 2 cities in Texas), the median was 3,500 ng/g dwt, with a maximum of 17,000 ng/g dwt, dominated by BDEs 47, 99, 154, and 209.

d) Two studies of house dust in the United States showed similar trends of having a relatively consistent finding for most of the homes, and then one or two having substantially higher concentrations. In one, Stapleton et al (2005) analyzed dust samples from 17 homes in the Washington, DC area. They found total concentrations (including BDE 209) to be less than or near 7,000 ng/g dwt in 15 homes, but in two homes they

found 14,990 and 30,100 ng/g dwt, respectively. These samples were dominated by BDEs 47, 99, and 209 in comparable amounts. In the other, Sharp and Lunder (2004) analyzed dust samples in 10 homes from 9 states. Concentrations found in 8 of the homes were near or less than 6,000 ng/g dwt total, but in two homes, the total concentrations were 16,000 and 41,000 ng/g dwt. These high samples were dominated by BDEs 47, 99, and 209.

Perhaps a certain behavior within a household or office that puts one in close contact with a product containing BDEs or in close contact with dust heavily laden with BDEs explains this tendency in populations to have very high exposures at the high end. This is clearly an uncertainty that requires further investigation. Besides dust, the cause for these higher exposures could also be a high consumption rate of a particular food product that may be impacted by local conditions. For example, Sjodin et al (2000) found that Swedish fisherman consuming large amounts of Baltic Sea fish had a median median BDE 47 level five times higher than that of nonconsumers. However, only a small statistically insignificant difference was found between urban anglers and non-anglers in a study of 93 individuals (79 anglers, 14 non-anglers) in the United States (Morland et al, 2004). Discussions below focus on the question of whether dust-related exposures are likely to dominate the median exposures. It should also be noted that findings of PBDE body burdens in other United States studies are not very different from the NHANES study. As described in Section 5.2., the means or medians from essentially all United States studies are within a reasonably narrow range of PBDE concentrations: 30 to 100 ng/g lwt. The consistency in central tendency findings in these studies suggests consistency in exposure of Americans to PBDE.

5.7. UNCERTAINTY AND VARIABILITY IN ESTIMATING INTAKE DOSE AND CONVERTING THAT DOSE TO A BODY BURDEN

The analysis in this chapter has taken a "point estimate" approach in the intake estimation, the pharmacokinetic modeling, and the comparison with measured body burdens. In so doing, it has arrived at a fairly broad-reaching finding that the bulk of exposures are indoor-dust related. The exercise revealed that between 7 and 8 ng/kg-day of total PBDE exposure appears necessary in order to reproduce the median body burdens

seen in the adult population. This finding is based on pharmacokinetic modeling—given the model and parameters chosen for BDE congeners, the median body burden would not be duplicated unless the intake dose were in the 7–8 ng/kg-day range. The point estimate approach suggested that over 80% of this total intake came from dust ingestion, inhalation, and dermal contact with dust. However, there are uncertainties throughout this exercise, as well as variabilities in exposure. As discussed in the previous section, there appears to be exposures at the very high end of the general population that are substantially higher than the central tendency exposures that are characterized by a dose in the range of 7 to 8 ng/kg-day, dominated by dust exposures. This construct of the exercise does not guarantee that the finding of the importance of the dust pathways is proven. The purpose of this section is to lay out all of these uncertainties and variabilities so that this primary finding and other findings in this chapter can be understood in their proper context.

a) Uncertainties With Estimates of Dust Intakes of PBDE:

   1. soil/dust ingestion amount: The amount of 50 mg/day assumed for adults is a classic uncertainty. Described as an "average" value for soil ingestion in the *Exposure Factors Handbook* (EPA, 1997), the actual amount of ingestion of indoor dust + outdoor soil could very easily be much lower. While Sjodin et al. (2004b) assumed a dust ingestion rate of 100 mg/day, Stapleton et al. (2005), on the other hand, assumed an adult housedust ingestion rate of 0.56 mg/day, over two orders of magnitude lower, explaining their estimate of 3.3 ng/day of exposure to PBDEs via ingestion exposure to household dust. As noted earlier in the chapter, this value of 0.56 mg/day housedust was listed in EPA's *Exposure Factors Handbook* (EPA, 1997), which cited Hawley (1985) for using a value of 0.56 mg/day to characterize adult exposure to housedust from normal activities in the house (higher exposures of over 100 mg/day resulted from "work in the attic"). As noted, Stapleton et al. (2005) estimated an exposure to total PBDEs of 3.3 ng/day based on this assumption. It would seem that their subsequent studies, and those of others, essentially prove that this is much too low an estimate for indoor dust ingestion, at least in the context of estimating exposure to indoor dust. Specifically, their recent study (Stapleton et al., 2008b) directly measuring BDEs on hands using wipes, and then using

an empirical model to estimate hand-to-mouth exposures, found a median exposure estimate for adults of 154 ng/day.

   2. soil/dust fraction: The procedure here to characterize ingestion of housedust is to multiply a total amount ingested by a fraction that comes from the house. The assumption that 0.90 for this fraction, based on data suggesting 90% of the time is spent indoors, is conservative. Paustenbach et al. (1997) looked at data suggesting that 50% of indoor dust originates from outdoor soil. This may be less important for the current exercise because dust concentrations were taken from indoor dust measurements, so the origin of the dust is not relevant when using measured dust concentrations directly. Still, the assumption that 90% of the ingested amount is indoor dust is really not substantiated in the literature. It is appropriate for "time spent indoors" and, for obvious reasons, would be reasonable for inhalation exposures.

   3. bioavailability of PBDEs in dust: Bioavailability was considered in the context of *absorption fractions* for dust ingestion and dust dermal contact. In the dermal contact pathway, an *absorption fraction* of 0.03 (3% absorbed) was assumed for all congeners (Table 5.5), and in the dust ingestion pathway, different absorption fractions for each congener ranging from about 0.20 to 0.80, were assumed. There is a subtle difference in the way in which results are presented in this study, in that for all pathways except dermal contact, the *intake dose* is calculated just by the contact rate (food ingestion rate, inhalation rate, etc) multiplied by the concentration of BDEs in the contact media (in food, air, etc). For the dermal contact pathway, this *intake dose* has already considered this absorption fraction of 0.03. For the other pathways, the absorption fractions are used in the PK modeling of body burdens due to intake doses; the absorption fractions reduce the intake dose to consider absorbed dose. In any case, all of the absorption fractions are uncertain. The value of 0.03 for dermal contact was used in the modeling of background exposures to dioxin based on literature showing that this tightly sorbed contaminant would not desorb readily from soil contacting the skin and then penetrate the skin surface to a great extent (EPA, 2003). However, this may not be true for PBDEs, or at least true for the case where the vehicle is house dust rather than soil. Only a few studies were found that have measured dermal absorption of PBDEs. Although none of these used PBDEs sorbed to dust, soil or other solid matrices, they provide some indication of the dermal absorption potential of these chemicals. Hughes et

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al (2001) examined the in vitro dermal absorption of [^{14}C]decabromodiphenyl oxide (DBDPO). Skin from the adult hairless female mouse was removed and mounted in flow-through diffusion cells. The chemical was applied to the skin at three dose levels (6, 30 and 60 nmol) in a volatile vehicle (tetrahydrofuran). The 24-h cumulative percent of the dose in the receptor fluid was 0.07–0.34%. The percent of the applied dose detected in the skin after 24 hr ranged from 2 to 20%. Staskal et al (2005) conducted a mouse in vivo study to measure dermal absorption of 2,2',4,4'-tetrabromodiphenyl ether (BDE 47). A single dose was applied to the skin in an acetone solution. About 62% of the administered dose was absorbed over a 5 day period. Roper et al (2006) studied the dermal absorption of 2,2',4,4'-tetrabromodiphenyl ether (BDE 47) using rat and human skin in vitro. A single dose was applied to the skin in an acetone solution. The total absorbed dose in human skin after 24 hr was 3.13% (1.93% in the receptor solution and 1.2% in the skin). The total absorbed dose in rat skin after 24 hr was 17.94% (14.81% in the receptor solution and 3.13% in the skin). Roper et al (2006) showed that rat skin was more permeable than human skin to TBDE and this has also been observed for other chemicals and other rodents in multiple studies (i.e. van Ravenzwaay et al., 2004). All three studies applied the chemical in a solvent which evaporated rapidly leaving a residue of pure chemical on the skin. This would tend to increase the absorption relative to a similar dose which has been absorbed to dust. Thus, the 3% absorption based on human skin in vitro testing by Roper et al (2006) is probably the most relevant data, but may be high for dust exposures. USEPA (2004) recommends 3% absorption for TCDD in soil. The similarity of TCDD to PBDEs in terms of lipophilicity and molecular size, adds support to the assignment of 0.03 for PBDEs. The absorption fractions for dust ingestion were derived from a study by Huwe et al (2008), where BDE congeners were administered to male rats in corn oil and household dust. It was found that absorption amounts were similar for corn oil and dust, and the congener-specific results from that study, showing absorption fractions ranging between 0.18 and 0.78, were directly used for the dust ingestion pathway in this study. The absorption fractions for food ingestion and inhalation were higher at between 0.78 and 0.94. It is known that organic compounds sorbed to soil, or dust, are less bioavailable than when ingested in food or inhaled. Paustenbach et al. (2006) examined the literature on the bioavailability of 2,3,7,8-TCDD. In their monte carlo simulations on contaminated soil exposures, they

assumed that the oral absorption of this compound in contaminated soil followed a lognormal distribution with a range of 0.5 to 63% and a mean value of 35%. ATSDR (2004) reviewed the literature on bioavailability of PCBs in soil and concluded that a range of 40-65% was appropriate. Paustenbach et al. (1997) did comment on the fact that the bioavailability of contaminants on house dust, in general, is much greater than that in outdoor soil, because house dust particles are finer than soil particles. In a backward pharmacokinetic modeling exercise in which McDonald (2005) derived intake estimates that would correspond to measured body burdens, he assumed absorption fractions of 0.78 to 0.94 for BDEs 47, 99, 100, 153, and 154. This was not pathway-specific and was based on absorption experiments in rats, and although the carrier in the experiments was not noted, it was unlikely to be soil. EPA (2003) assumed that the absorption fraction of dioxin from a total intake dose was 0.80, but dioxin intakes are dominated by food and not dust/soil. In general, the point is made that while an intake dose of PBDEs could be dominated by dust ingestion and dermal contact with dust, there remains uncertainty as to how much of the doses by these pathways get absorbed to eventually appear in blood.

c) Uncertainties and Variabilities With Other Pathways:

It is reasonable to conclude that there is less uncertainty in characterizing central tendency for water ingestion, food ingestion, and inhalation, as compared to soil/dust ingestion and soil/dust dermal contact. Food intake quantities as developed in the *Exposure Factors Handbook* (EPA, 1997) were derived from the U.S. Department of Agriculture Continuing Survey of Intakes by Individuals (CSFII), which is a survey of high quality used in many assessments, such as the U.S. Food and Drug Administration's market basket surveys, which are used to determine intakes from measurements of contaminants in sampled food. Water ingestion and air inhalation are also comprehensively studied and the contact rates chosen are characterized as very reasonable central tendency point estimates. Furthermore, most food surveys arrive at comparable concentrations in food products. One exception described in Chapter 4 was the finding by the United Kingdom's Food Standards Agency of high BDE 209 in all food sampled (FSA, 2006). The quality of this data are unknown, and might be questioned since it differs from essentially all other studies of PBDEs in food. Interestingly, BDE 209 was found at high levels also in dry cat food (Dye et al, 2007).

While BDE 209 was found at low concentrations, <0.01 ng/g wwt, and comprised only a small percent of total concentration, <10%, in canned "wet" cat food products, it was found at levels between 0.42 and 2.28 ng/g wwt in dry cat food and comprised 73 – 87% of total concentrations in four types of dry cat food (chicken, salmon, poulty and fish, and adult dry food). When combining food concentrations of PBDEs with food intakes, most studies around the world, including the United States, have arrived at adult food intakes in the 1-2 ng total BDE/kg-day. Inhalation and water ingestion have mostly arrived at adult exposures less than 1 ng/kg-day. These findings can easily be seen in Table 5-4, which lists intake estimates from all pathways from studies around the world. Only the UK FSA study showing unusually high BDE 209 concentrations arrived at food intakes greater than this 1-2 ng total BDE/kg-day range.

It should also be noted that there may be pathways not considered in this assessment. For example, while the assessment considered dermal contact with dust, it did not consider dermal contact with PBDE-treated products. Dermal absorption of PBDEs may occur via direct contact with treated materials such as clothing, carpeting, upholstery, etc. Wester et al (1996) has shown that chemicals in fabric can transfer from fabric into and through human skin. This was based on human skin in vitro experiments with glyphosphate and malathion applied to cotton sheets. Absorption from dry cloth was found to occur but it was less than the chemicals in aqueous solutions. When the cloth was wetted with water to simulate sweating, absorption increased. For example, absorption of malathion from aqueous ethanol solution was 8.77 ± 1.43%. This decreased to about 0.60% for dry cotton sheets. However, absorption from cotton sheets increased to 7.34 ± 0.61% when wetted with aqueous ethanol.

d) Uncertainties With Pharmacokinetic Modeling

The choice of the simple 1-compartment 1st order model is reasonable for PBDEs. Like dioxins, for which the model has been extensively and successfully used (EPA, 2003, and other citations not provided), PBDEs are lipophilic and persistent in body. The application of the model at steady state instead of in a temporally variable mode could introduce uncertainties. For dioxins, it was found that application of the model at steady state using dose estimates developed for current conditions would underestimate average adult body burdens by about one-half (EPA, 2003). This is because of high

dioxin intakes in the middle decades of the twentieth century (the 1960s until about 1980; much higher than current intakes), such that body burdens of older adults are higher than younger adults, driving up the current average adult population body burden. Similar age trends were not found in BDE population studies. In fact, the younger age ranges were found to have higher body burdens in some studies. Unlike dioxins, PBDEs were not in the environment prior to their introduction into household products beginning in the 1970s and body burdens were first found to contain PBDEs in the 1980s. With the voluntary withdrawal of pentaBDE and octaBDE formulations in 2004, there may be declines seen in the key congeners of these formulations in the most recent surveys. Generally, though, it may be reasonable to assume that exposures have at least remained steady if not increased (because of perhaps rising levels in dust as more products using the PBDEs were incorporated into modern living) between 1980 and the present. This provides a reasonable justification for use of the simple PK model in a steady-state mode, at least in comparison with dioxin, where steadily declining exposures leads to an underestimate of population body burdens if using a steady state model

The uncertainties associated with the dose estimates were discussed in the previous section. Absorption was also discussed in the previous section in the context of absorption of PBDEs from ingested dust. There was limited choice in the literature from which to make selections of elimination half-lives. Only one study, Geyer et al. (2004) was found to assign half-lives to BDEs 47, 99, 100, 153, and 154 of 2.9 to 11.0 yr. Only one study was found, Thuresson et al. (2006), which provided estimates for BDE 183 of 0.26 yr (94 d) and for BDE 209 of 0.041 yr (15 d).

With these selections, it was found BDE 47 appears to be underpredicted. The prediction was 10.0 ng/g lwt, compared to observations of 20.5 ng/g lwt in blood and 26.0 ng/g lwt in breast milk. The cause for this underprediction is not known, but it could very easily be the assumed half-life in humans. At 3.0 years, it dissipated nearly twice as fast as BDE 99, which was assigned a half-life of 5.4 years. Had it dissipated at a half-life of 10 years, the prediction would jump to greater than 30 ng/g lwt, now more than twice the prediction for BDE 99, and more in line with current measurements. In the same vein, BDE 99 was overpredicted. It was predicted at 14.6 ng/g lwt, while is measured as 5.0 ng/g lwt in blood and 5.4 ng/g lwt in breast milk. Perhaps the half-lives of both congeners should be reversed. However, other evidence in the literature supports

the assumption that BDE 47 is eliminated more rapidly than BDE 99. In an experiment where BDEs 47, 99, 100, and 153 were all administered intravenously to mice, Staskal et al (2006) found that tissue concentrations were highest for BDE 153, followed by BDEs 100, 99, and 47. Similar to the human data, this mice data suggests a more rapid elimination of BDE 47 as compared to BDE 99 and also other key congeners. On the other hand, the human body burden data show that BDE 47 concentrations are the highest of all congeners and about four times higher than BDE 99 concentrations. The modeling in this study (given uncertainties of course) suggests that the dose of the two congeners is about equal. Logically, therefore, one would speculate that BDE 99 would be more rapidly eliminated as compared to BDE 47, but that is not what the human toxicokinetic data, or the rodent data cited, has found. This trend of higher BDE 47 concentrations in humans may suggest debromination of BDE 99 (or higher brominated congeners) to form BDE 47 in the body. In other words, the modeling may correctly determine body burdens due to intakes, but the half-lives assigned might not reflect full elimination from the body but rather higher brominated congeners are being transformed to lower brominated congeners. However, this is much too speculative given current information on human and animal metabolism. It might be possible that the one study measuring the elimination of these congeners in an occupational cohort was not appropriate for the half-lives of these two congeners in the background population. In any case, it would be premature to use this framework to "calibrate" congener-specific half-lives in humans. Still, it gives an indication of where key information gaps are.

With regard to the final total concentrations, they were predicted at 35.9 ng/g lwt and measured at 36.3 ng/g lwt in blood and 44.1 ng/g lwt in breast milk. Other studies in breast milk showed different concentrations, some lower and some higher. Most other blood measurement studies were somewhat higher than this NHANES result, but most studies found central tendency total concentrations to be well under 100 ng/g lwt, so the prediction is about within a factor of 2 of most measurements, and in the case of NHANES blood and the selected breast milk concentrations, predictions came very close to measurements.

The results of this simple PK modeling exercise are consistent with those of McDonald (2005), although he took the opposite approach: he started with body burdens and used reverse PK modeling to derive the intake doses which would explain the body

burdens. He used the same PK parameters (absorption fractions and congener-specific half-lives) as were used in this exercise, although he did not separately consider dust ingestion with a different absorption fraction. Compiling data on PBDEs in breast milk (including some of the same data summarized above), he found a median concentration of 48 ng/g lwt, a mean of 90 ng/g lwt, and a 95% of 302 ng/g lwt. These totals were the sum of BDE congeners 47, 99, 100, 153, and 154. Modeling these congeners individually, he found that the doses of total PBDEs that would explain these body burdens were 8.5, 16.0, and 53.6 ng/kg-d for the median, mean, and 95%. In the exercise above, dose was forward calculated from exposure media concentrations and contact rates, and the adult dose was estimated at 547 ng/day total, or on a body weight basis assuming a 70 kg adult, 7.8 ng/kg-day. This dose was estimated based on "representative" central tendency media concentrations and average contact rates, so the consistency of 7.8 ng/kg-day derived by a forward calculation for "average" conditions with McDonald's 8.5 ng/kg-day derived by a retrocalculation from the median body burden is very encouraging.

What is equally interesting is that McDonald (2005) cites European body burdens which are more like 10 ng/g lwt, as well as estimates of dose made by Europeans based on food alone, which are in the range of 1.0 ng/kg-day, and using his PK framework. McDonald (2005) found quantitative consistency in those two quantities (i.e., the dose he reverse-calculated from body burdens matches the forward calculated dose by food ingestion developed by European researchers). In essence, he is implying that the primary pathway of BDE exposure to Europeans is through food consumption. It is unclear why house dust and other indoor sources of PBDEs were not considered. These pathways were considered by Harrad et al. (2006) in a study conducted in the United Kingdom. They observe that dust concentrations are much lower in the United Kingdom as compared to the United States, while food concentrations are comparable in the two countries. They suggest that the higher body burdens found in the United States are likely to indoor dust exposures. If not entirely due to indoor dust, there may be other pathways of exposure Americans have that Europeans do not. In any case, the prevalence of lower brominated BDEs in dust in the United States and the subsequent impact on United States body burdens is the key issue identified in this assessment, and, needless to say, it requires further research.

5.8. OVERALL FINDINGS OF EXPOSURE OF AMERICANS TO PBDES

Examination of literature data and exposure exercises in this chapter support these general findings:

1. PBDEs bioaccumulate in lipids, and body burden measurements are expressed on a ng/g lipid weight (lwt) basis. Nearly all body burden data are from blood and breast milk. Body burdens of Americans are higher than body burdens of individuals in other countries; most of the non-United States data are from Europe. Data suggests total PBDE body burdens in the range of 30 to 100 ng/g lwt in Americans, while it is less than 10 ng/g lwt for people in other countries. There is some suggestion that body burdens are higher in women than men; breast milk data suggest lipid-based concentrations that are higher than the lipid-based blood concentrations (and blood measurements are from both men and women).

2. The predominant congener found in body burden studies is BDE 47, explaining about 50% of the total concentration. The second most found congeners are 99 and 153, both explaining in the range of 10-20% of total concentrations. Most of the studies have not measured BDE 209, but when measured, it was generally found in about half the samples at low levels near 1-2 ng/g lwt

3. The limited data on adipose tissue are consistent with these findings for blood and breast milk. Limited occupational data support the observation that individuals in occupations which would lead to higher exposures to specific congeners have higher concentrations of those congeners than the general population. Very limited data are available on body burdens of children and infants, but in one study of a family including 2 parents and 2 young children— the children's body burdens were in the 200 – 400 ng/g lwt range, while the parents had blood measurements near 100 ng/g lwt. An important trend that warrants further investigation is that, even in background adult populations, there are individuals experiencing very high exposures. This has been seen in studies of PBDEs in blood as well as indoor dust measurement studies, suggesting that dust exposures could explain these unusually high exposures.

4. Intakes have been expressed as a straight-dose basis, ng total PBDE/day, or on a body-weight basis, ng total PBDE/kg-day. Intake estimates in the literature have tended to focus more on intake by food than by housedust, although this has changed in recent years as researchers recognize the importance of indoor dust in the overall exposure paradigm for PBDEs. Several of the researchers measuring PBDEs in dust also estimated intakes by soil/dust ingestion using their measurement data, and others have more directly measured potential hand-to-mouth intakes by sampling the hands of individuals using alcohol wipes (see Stapleton et al., 2008b). Some estimates of exposure via housedust were high, up to 400 ng/day, but other estimates were as low as 3 ng/day; the latter assumed less than 1 mg/day dust ingestion while the former assumed a conservative 100 mg/day dust ingestion rate. Stapleton et al. (2008b) estimated a median adult hand-to-mouth intake of 154 ng/day (> 2 ng/kg-day), based on hand wipe data in conjunction with a model on exposure to contaminants on hands. Literature estimates of intakes from food ingestion were in the range of 0.5 to 2.0 ng/kg-day.

5. Intake estimates derived in this study, based on exposure media concentrations derived in Chapter 4 combined with average contact rates, arrived at total daily intakes in the range of 450 to 600 ng/day for children and adults. These intakes were driven by indoor exposures via soil/dust ingestion, dermal contact with dust, and inhalation of indoor air; those three pathways accounted for about 83% of total intakes, with food and water ingestion explaining the remaining 17%. Infant intakes via breast milk exceeded 1,400 ng/day total PBDEs.

6. Using a simple pharmacokinetic (PK) model parameterized with available literature values, lipid-based concentrations (not specific to blood or milk) were predicted, starting with the intake values as summarized above. On a total PBDE basis, the prediction was low at 35.9 ng/g lwt, while it was observed at 36.3 ng/g lwt in blood and 44.1 ng/g lwt in milk as central tendency values (geometric mean in blood and median in breast milk) values in studies selected as representative of the general population. Predictions were reasonably close to measurements for 7 of 9 congeners. The prediction of BDE 47 at 10.0 ng/g lwt did not match the observed measurements of 20.5 ng/g lwt in blood and

26.0 ng/g lwt in milk, and the prediction of 14.6 ng/g lwt of BDE 99 was judged meaningfully higher than the observed concentrations of 5.0 ng/g lwt for blood and 5.6 ng/g lwt for breast milk. The causes for these discrepancies in the BDEs 47 and 99 are not known, but it could very easily be the assumed half-lives in humans. At 3.0 years, BDE 47 was eliminated nearly twice as fast as BDE 99, which was assigned a half-life of 5.4 years. Had BDE 47 been assigned an elimination half-life of 10 years, the prediction would jump to over 30 ng/g lwt, now twice as high as BDE 99 prediction, and more in line with measurements. Similarly, if the BDE 99 half-life was half as much as it was, predictions of BDE 99 in lipid would better match the observed lipid concentrations.

7. While these predictions encouragingly match observations, uncertainties exist in the exercise, starting from development of dose estimates based on limited environmental measurements, to indoor contact rates with housedust, to the PK parameters of absorption and elimination half-life. There is also variability in United States body burdens. Contact rates for food/water ingestion and inhalation are fairly well established, and the exposure media concentration summaries suggest similarities among different studies in food and air concentrations. It was assumed that the remainder of the exposures came from house dust through the pathways of ingestion and dermal contact. Circumstantial evidence supporting this hypothesis was the high concentrations found in United States house dust, and other researchers have also identified house dust as a key matrix of exposure concern for these compounds. The overall weight-of-evidence of this exercise supports the finding that the bulk of United States exposures occur in the indoor environment through contact with house dust. The exercise suggests these exposures account for between 80 and 90% of total exposures, with the remainder due primarily to food ingestion.

8. This PK model was used to model infant body burden impacts from consumption of breast milk. Lipid concentrations were modeled to rise to 200 ng/g lwt through age 5, to then drop gradually to below 100 ng/g lwt by age 19. However, this result was very sensitive to assumed elimination half life for total BDEs. When an adult-like half-life of 6 years was used in this infant model, concentrations rose to nearly 200 ng/g lwt by age 1 to continue to rise to 325 ng/g lwt by age 5, to then drop to below 100 ng/g lwt by age 19.

Only one study was available with which to compare these results. Studying a family of four – two adults, a child, and a toddler, concentrations in the 18-month old toddler rose to above 400 ng/g lwt, and concentrations in the 5 year-old were above 200 ng/g lwt. At the same time, the adult concentrations in the family were near 100 ng/g lwt. While this study was insufficient to provide verification of either modeling assumption on half-life in infants and children, this ancillary data supports the conclusion that infant and childhood body burdens appear likely to be significantly higher than that of adults.

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Table 5-1. Blood concentrations of PBDE congeners in the United States

Congener	Concentration, ng/g lwt	Comment	Citation
TriBDE			
17	0.3	N=1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. 2004
	0.1, 0.03, 0.05	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals – 29 MS, 10 NY	Schecter et al., 2005
	0.013	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	0.003	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
28	1.2	N=2040; NHANES 2003/4; geometric mean	Sjodin et al., 2008
	0.8	N=1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. 2004
	1.3, 1.9, 1.1	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals – 29 MS, 10 NY	Schecter et al., 2005
	0.65	N = 100; median serum level in 100 mothers who were pregnant and near WTC on 9/11	Wolff et al et al., 2005
	0.92	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	0.58	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
TetraBDE			
47	20.5	N=2040; NHANES 2003/4; geometric mean	Sjodin et al., 2008
	0.6 (<0.4-23.8)	Median, range; 6 of 12 blood donor samples collected in Ill in 1988 quantified	Sjodin et al., 2001
	13.2; .7-1388.6	Geometric mean; range of 93 urban angler samples in NY and NJ; 93% detected	Morland et al et al., 2005
	28.1	N=1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. 2004
	34 (29-98)	Median from 2000/2 (range) in trend study from pooled blood from around U.S.	Sjodin et al., 2004
	28; 9.1-310	Median; range from maternal/cord blood (n=12); 2001; cord blood was identical	Mazdai et al., 2003
	50.6 (<10-511)	Mean, range from Laotian reproductive age women in San Francisco area, 97-99	Petreas et al. 2003
	32.5, 44.2, 25.0	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals – 29 MS, 10 NY	Schecter et al., 2005
	0.6 (<0.4-23.8)	Median, range (n=12) from individual donors collected in 1988 in Illinois	Sjodin et al., 2001
	23 – 60; 94 – 137; 186 – 245	Range from 2 parents, 1 5 year-old, and 1 18-month old from case study in CA in 2004	Fischer et al., 2006
	9.7	N = 100; median serum level in 100 mothers who were pregnant and near WTC on 9/11	Wolff et al et al., 2005
	22.6	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	13.1	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	11; 205	N=24; median and max from a cohort of pregnant Latina women in CA	Bradman et al., 2006
66	0.3	N=1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. 2004

	0.3, NA, 0.4	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals – 29 MS, 10 NY	Schecter et al., 2005
	ND	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	0.1	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
77	ND, NA, 0.01	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals – 29 MS, 10 NY	Schecter et al., 2005
PentaBDE			
85	1.0; 0.2-109.1	Geometric mean; range of 92 urban angler samples in NY and NJ; 27% detected	Morlandet alet al., 2005
	0.8	N=1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focantet al. 2004
	0.7 (0.5-1.4)	Median from 2000/2 (range) in trend study from pooled blood from around U.S.	Sjodin et al., 2004
	NA, 1.1, 1.2	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals – 29 MS, 10 NY	Schecter et al., 2005
	0.4	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	0.3	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	0.3; 5.0	N=24; median and maximum from a cohort of pregnant Latina women in CA	Bradman et al., 2006
99	5.0	N=2040; NHANES 2003/4; geometric mean	Sjodin et al., 2008
	0.3; <0.2-3.7	Median, range; 8 of 12 U.S. blood donor samples collected in Ill in 1988 quantified;	Sjodin et al., 2001
	3.2; 0.3-545.5	Geometric mean; range of 93 urban angler samples in NY and NJ; 66% detected	Morlandet alet al., 2005
	9.2	N=1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focantet al. 2004
	11 (6.8-26)	Median from 2000/2 (range) in trend study from pooled blood from around U.S.	Sjodin et al., 2004
	5.7; 2.4-68	Median; range from maternal/cord blood (n=12); 2001; cord blood was identical	Mazdai et al., 2003
	8.4, 12.8, 11.1	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals – 29 MS, 10 NY	Schecter et al., 2005
	4 – 16; 28 – 34; 37 – 45	Range from 2 parents, 1 5 year-old, and 1 18-month old from case study in CA in 2004	Fischer et al., 2006
	1.5	N = 100; median serum level in 100 mothers who were pregnant and near WTC on 9/11	Wolffet alet al., 2005
	6.0	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	2.7	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	2.9; 54.0	N=24; median and maximum from a cohort of pregnant Latina women in CA	Bradman et al., 2006
100	3.9	N=2040; NHANES 2003/4; geometric mean	Sjodin et al., 2008
	0.2; <0.1-2.4	Median, range; 10 of 12 U.S. blood donor samples colleted in ILL in 1988 quantified	Sjodin et al., 2001
	2.7; 0.3-280.6	Geometric mean; range of 93 urban angler samples in NY and NJ; 88% detected	Morland et al., 2005
	6.9	N=1; pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al., 2004
	5.9 (3.5-18)	Median from 200/2 (range) in trend study from pooled blood from around U.S.	Sjodin et al., 2004
	4.2; 1.9-110	Median; range from maternal cord blood (n=12); 2001; cord blood was identical	Mazdai et al., 2003

	5.7, 5.2, 4.7	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=11) from Tx; mean of 39 individuals - 28 MS, 10 NY	Schecter et al., 2005
	8-22; 30-39; 57-87	Range from 2 parents, 1 5 year-old, and 1 18-month old from case study in CA in 2004	Fischer et al., 2006
	1.8	N=100; median serum level in 100 mothers who were pregnant and near WTC on 9/11	Wolff et al., 2005
	5.7	N=8; mean from vegans (ND=0)	Schecter et al., 2006a
	3.7	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	1.8; 44.0	N=24; median and maximum from a cohort of pregnant Latina women in CA	Bradman et al., 2006
HexaBDE			
138	NA, 0.3, 0.2	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals - 29 MS, 10 NY	Schecter et al., 2005
	0.07	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	0.04	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
153	5.7	N=2040; NHANES 2003/4; geometric mean	Sjodin et al., 2008
	0.3; 0.1-2.0	Median, range; 12 of 12 U.S. blood donor samples collected in Ill in 1988 quantified	Sjodin et al., 2001
	3.2; 0.4-165.2	Geometric mean; range of 93 urban angler samplers in NY and NJ; 96% detected	Morland et al., 2005
	6.2	Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. 2004
	7.3 (1.8-17)	Median from 2000/2 (range) in trend study from pooled blood from around U.S.	Sjodin et al., 2004
	2.9; 1 - 83	Median; range from maternal/cord blood (n=12); 2001; cord blood was identical	Mazdai et al., 2003
	12.3, 11.7, 5.7	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals - 29 MS, 10 NY	Schecter et al., 2005
	19 - 42; 49 - 65; 75 - 141	Range from 2 parents, 1 5 year-old, and 1 18-month old from case study in CA in 2004	Fischer et al., 2006
	1.8	N = 100; median serum level in 100 mothers who were pregnant and near WTC on 9/11	Wolff et al., 2005
	14.6	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	8.9	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	1.5; 35	N=24; median and maximum from a cohort of 24 pregnant Latina women in CA	Bradman et al., 2006
154	0.6; 0.09-24.7	Geometric mean; range of 89 urban angler samples in NY and NJ; 25% detected	Morland et al., 2005
	1.2	N=1; pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al., 2004
	0.95 (0.5-1.8)	Median from 2000/2 (range) in trend study from pooled blood from around U.S.	Sjodin et al., 2004
	0.3 (ND-6.1)	Median, range from maternal/cord blood (n=12); 2001; cord blood was identical	Mazdai et al., 2003
	0.8, 0.8, 1.0	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals - 29 MS, 10 NY	Schecter et al., 2005
	0.6-6; 3-7; 4-17	Range from 2 parents, 1 5 year-old, and 1 18 month-old from case study in CA in 2004	Fischer et al., 2006
	0.6	N=8; mean from vegans (ND=0)	Schecter et al., 2006a
	0.4	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	0.3; 4.2	N=24; median and maximum from a cohort of	Bradman et al., 2006

		24 pregnant Latina women in CA	
HeptaBDE			
183	0.2; 0.1-1.3	Median, range; 12 of 12 U.S. blood donor samples collected in Ill in 1988 quantified	Sjodin et al., 2001
	0.5; 0.1-2.0	Geometric mean; range of 93 urban angler samplers in NY and NJ; 29% detected	Morlandet alet al., 2005
	ND; 0-2.7	Median; range from maternal/cord blood (n=12); 2001; cord blood was identical	Mazdai et al., 2003
	0.3, 0.4, 0.4	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals — 29 MS, 10 NY	Schecter et al., 2005
	0.04	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	0.05	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
NonaBDE			
203	ND(0.1); <0.1-0.2	Median, range; 5 of 12 U.S. blood donor samples collected in Ill in 1988 quantified	Sjodin et al., 2001
DecaBDE			
209	ND(1.0); <1.0-33.6	Median, range; 5 of 12 U.S. blood donor samples collected in Ill in 1988 quantified	Sjodin et al., 2001
	NA, 1.4, 2.7	3 2003 results: 1 pooled serum (n=100) from TX, 1 pooled whole blood (n=100) from TX; mean of 39 individuals — 29 MS, 10 NY	Schecter et al., 2005
	2 - 23; 9 - 143; 19 - 233	Range from 2 parents, 1 5 year-old, and 1 18-month old from case study in CA in 2004	Fischer et al., 2006
	ND	N=8; mean from vegans (ND = 0)	Schecter et al., 2006a
	3.7	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c

Table 5.2. Breast milk concentrations of PBDE congeners in the United States

Congener	Concentration, ng/g lwt	Comment	Citation
MonoBDE			
3	0.15	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
BiBDE			
7	0.01	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
8 + 11	0.02	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
12	0.01	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
13	0.01	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
15	1.46	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	1.5	WHO inter-lab calibration study with results from 17 of 24 countries; U.S. listed here	Kotz et al., 2005
TriBDE			
17	0.1	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	0.02 (0.01)	Mean (median), 47 women in Texas 2002	Schecter et al. 2003
	0.02	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	DL (0.01)	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
25	0.02	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
28	7.4	Mean of 3 pooled sample analyses from CO, CA, and NC	Focant et al., 2004
	8.6	WHO inter-lab calibration study with results from 17 of 24 countries; U.S. listed here	Kotz et al., 2005
	2.4 (1.2)	Mean (median), 47 women in Texas 2002	Schecter et al., 2003
	1.1	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	1.80, 0.47	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	0.93	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
28 + 33	6.2	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
28/32	3.8	Mean of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
32	0.2	Mean of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
35	0.02	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
37	0.06	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
TetraBDE			
47	193.0	Mean of 3 pooled sample analyses from CO, CA, and NC	Focant et al., 2004

	84.9	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	50; 26	Mean; median of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
	233.9	WHO inter-lab calibration study with results from 17 of 24 countries; U.S. listed here	Kotz et al., 2005
	35	Partial data from longitudinal study in Pennsylvania; 3 women	Sjodin et al., 2005
	40.8; 18.4	Mean, median, 47 women in Texas 2002	Schecter et al., 2003
	17.1	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	40.7, 7.7	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	13.9	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
49	0.6	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
66	1.1	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	0.3	Mean of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
	0.65 (0.14)	Mean (median), 47 women in Texas 2002	Schecter et al., 2003
	0.07	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	1.07, <0.84	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	0.12	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
71	0.05	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	0.5, 0.2	Mean, median of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
75	0.07	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
77	0.01 (NA)	Mean (median), 47 women in Texas 2002	Schecter et al. (2003)
	0.02, <0.84	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
PentaBDE			
85	5.5	Mean of 3 pooled sample analyses from CO, CA, and NC	Focant et al., 2004
	2.3	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	1.1, 0.6	Mean, median of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
	1.15, 0.41	Mean, median, 47 women in Texas 2002	Schecter et al. (2003)
	0.4	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	1.02, <0.46	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	0.26	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007

99	55.0	Mean of 3 pooled sample analyses from CO, CA, and NC	Focant et al., 2004
	20.9	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	10, 5.4	Mean, median of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
	58.7	WHO inter-lab calibration study with results from 17 of 24 countries; U.S. listed here	Kotz et al., 2005
	8	Partial data from longitudinal study in Pennsylvania; 3 women	Sjodin et al., 2005
	14.0, 5.7	Mean, median, 47 women in Texas 2002	Schecter et al. (2003)
	3.5	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	11.8, 1.5	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	2.42	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
100	34.4	Mean of 3 pooled sample analyses from CO, CA, and NC	Focant et al., 2004
	18.4	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	12, 5.2	Mean, median of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
	39.2	WHO inter-lab calibration study with results from 17 of 24 countries; U.S. listed here	Kotz et al., 2005
	8.2, 2.9	Mean, median, 47 women in Texas 2002	Schecter et al., 2003
	4.4	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	6.9, <0.46	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	2.40	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
118	0.2, <0.46	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
119	0.06	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
126	0.04	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
HexaBDE			
138	0.60, 0.09	Mean (median), 47 women in Texas 2002	Schecter et al. (2003)
	0.04	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	0.16, <0.03	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	0.03	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
138+166	0.26	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
140	0.21	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
153	16.4	Mean of 3 pooled sample analyses from CO, CA, and NC	Focant et al., 2004

	19.8	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	16, 4.8	Mean, median, of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
	17.6	WHO inter-lab calibration study with results from 17 of 24 countries; U.S. listed here	Kotz et al., 2005
	5.3, 2.0	Mean, median, 47 women in Texas 2002	Schecter et al., 2003
	7.0	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	5.2, 1.1	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	3.05	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
154	3.6	Mean of 3 pooled sample analyses from CO, CA, and NC	Focant et al., 2004
	1.5	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	0.8, 0.4	Mean, median, of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
	3.0	WHO inter-lab calibration study with results from 17 of 24 countries; U.S. listed here	Kotz et al., 2005
	0.76, 0.22	Mean, median, 47 women in Texas 2002	Schecter et al., 2003
	0.30	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	0.63, 0.05	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	0.17	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
155	0.3	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
HeptaBDE			
183	0.15	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	0.3, 0.2	Mean, median, of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
	0.13, 0.07	Mean, median, 47 women in Texas 2002	Schecter et al., 2003
	0.09	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	6.2, 4.1	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	0.065	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007
190	0.01	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
NonaBDE			
203	< 7 (ND)	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
206	0.01	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
207	0.06	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
208	0.01	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004

DecaBDE			
209	0.24	Mean from 20 primaparae women from around U.S..	Lunder & Sharp, 2004
	0.8, 0.4	Mean, median, of 40 samples from primeparae women from Pacific Northwest	NEW, 2004
	0.92, ND	Mean, median, 47 women in Texas 2002	Schecter et al., 2003
	0.42	N=11; mean from blood/milk study (ND=0)	Schecter et al., 2006c
	<204 (ND)	N=38; mean, median from 23 towns in MA	Johnson-Restrepo et al. 2007
	DL (0.25)	N=46; median from 46 women in the Boston, MA area	Wu et al., 2007

Table 5.3. Representative body burden levels of PBDEs in Americans

Description	Blood ng/g lwt	Mother's milk ng/g lwt
BDE 17	NA	NA
BDE 28	1.2	1.7
BDE 47	20.5	26.0
BDE 66	NA	0.1
BDE 85	NA	0.6
BDE 99	5.0	5.4
BDE 100	3.9	5.2
BDE 138	NA	NA
BDE 153	5.7	4.8
BDE 154	NA	0.4
BDE 183	NA	0.3
BDE 197	NA	NA
BDE 206	NA	NA
BDE 209	NA	0.4
TOTAL	36.3	44.9

Note: blood data from Sjodin et al., 2008; mother's milk data from NEW, 2004.
NA = not available.

Table 5.4. Estimates of general population intake, or exposure, dose of total PBDEs provided in the literature.

Pathway; units	Exposure dose; target individual	Reference; comment
United States and Canada		
Dust ingestion, ng/d	120 – 6000 for children; 3.3 for adults	Stapleton et al. (2005); children's estimate pertinent to ages 1-4, assuming 0.02 – 0.2 g/day ingestion rate; adult estimate assumes 0.00056 g/day.
Dust ingestion, ng/d	Child median – 1380 Adult median - 154	Stapleton et al. (2008); measurements on hands on 33 individuals; empirical model to estimate dust ingestion
Dust ingestion, ng/d	400 for adults	Sjodin et al. (2004); based on median of 4200 ng/g found in Atlanta vacuum dust samples, and 100 mg/day adult ingestion
Dust ingestion, ng/d	147; 228 for adults 580; 910 for toddlers	Harrad et al. (2008); dust conc from TX; median & average dust conc; 50, 200 mg/d dust ing for adults, toddlers
Diet, Dust, Inhalation, ng/d	155 for adults; 264 for toddlers (4 mo – 2 yrs); 227 for fish eater; 2190 for occupational	Jones-Otazo et al. (2005); 66% of adult exposure due to dust; 90% of toddler due to dust; air/dust modeling + food concentrations; Toronto Canada
Diet, ng/kg-d	0.9-1.5 for males/females above 12 yr; 2.6 for 2-5 yr; 306 for infant	Schecter et al. (2006); based on market basket survey and age-based intake rates; infant was for breast feeding
Diet, ng/kg-d	0.3 – 0.8 for adults	Huwe et al. (2005); lower estimate based on "lean meats" and higher on meats of high fat content.
Diet, ng/kg-d	<1 for children/adults for fish; 0.04 – 20 for children/adults for beef/chicken	Luksembourg et al. (2005); used market basket survey data on fish, beef, chicken from Northern CA.
Total dose, (i.e., all pathways), ng/kg-d	8.5, 16.0, and 53.6 for women	McDonald (2005); mean, median and 95% estimate for total dose based on backward PK modeling and body burden data for women.
Europe and Asia		
Inhalation and diet, ng/d	20; 9 (inhalation) and 107; 90 (food) for adults	Harrad et al. (2004); UK, mean and median provided for inhalation and food
Dust ingestion and inhalation, ng/d	0.9 – 22 (dust ing) and 2 (inh) for adults; 12 – 43 (dust ing) and 0.4 (inh) for toddlers	Harrad et al. (2006); UK; average results based on dust in 8 homes and air in 32 indoor locations; mean concentrations & range of contact rates
Dust ingestion, ng/d	143; 2205 for adults 572; 9020 for toddlers	Harrad et al. (2008); UK; median & average dust conc; 50, 200 mg/d ing for

		adults, toddlers; Canadian results also
Inhalation	0.2, 0.0005 – 2.9	Mandalakis et al. (2008); median and range for inhalation in cars only.
Diet, ng/d	97.3 for adults	Bocio et al. (2005); Spain
Diet, ng/d	72 and 63 for adults	Schuhmacher et al. (2007); industrial and urban areas of Spain
Dust ingestion & inhalation, ng/kg-d	2.0 and 0.2 for children, 0.2 and 0.4 for adults	Gavao et al. (2005); Kuwait
Diet, ng/d	62.5, 48.6, and 149.0 for adults	Knutsen et al. (2005); Norway
Diet and breast milk, ng/d	51 for adults; 110 for infants	Darnerud et al. (2001); Sweden; infant dose assumed 4.2 ng/g lwt in breast milk
Diet, ng/kg-d	0.79, adult	Bakker et al. (2006); Netherlands; food consumption survey combined with composite measurements of 47, 99, 100, 153
Diet, ng/kg-d	5.9, of which BDE 209 is 4.5, adult	FSA (2006); UK, based on total diet survey food samples + consumption data
Diet, ng/d	35, adult	Voorspoelset al. (2006); Belgium, based on market basket samples + consumption data, includes BDE 209
Fish, ng/d Nursing, ng/d Inhalation, ng/d	1.7 – 12.9, child to adult 48.2, infant 2.7 – 9.2, child to adult	Menget alet al. (2007); China; median values for fish intake/inhalation for different ages, 0-1 for nursing infant

Table 5.5. Exposure pathways and factors for the PBDE intake dose estimate

Exposure Factors; Units	Comment; description	Adult	Ages 1-5	Ages 6-11	Ages 12-19
body weight, kg	Used for converting ng/day to ng/kg body weight/day	70	15	30	58
soil Ingestion, mg/d	Central tendency values	50	100	50	50
soil dermal contact, mg/d	Surface area that contacts the skin (5700 cm^2/d for adults) * amount soil adhering to skin (0.07 mg/cm^2) * fraction absorbed thru skin (0.03); area corresponds to head, hands, forearms, lower legs	12	2.2	3.2	11
inhalation, m^3/d	Unpublished estimates from recent studies at 16.1 m^3/day for adults	13.3	7.5	12	14
fraction indoor	Children > 12 years and adults assume 21 hr/d; 19 hr/d for children	0.875	0.792	0.792	0.875
water ingestion, l/d	Estimates from EPA (1997) still considered current	1.4	0.69	0.79	0.97
milk ingestion, g/d	Data from USDA (1995)	175	348	357	308
dairy ingestion, g/d	Data from USDA (1995)	55	103	88	77
egg ingestion, g/d	Data from EPA (1997), in units of g/kg bw/d, assumes bw above	16.8	11.25	12.3	13.9
beef ingestion, g/d	Data from EPA (1997), in units of g/kg bw/d, assumes bw above	49.7	21	33	48.1
pork ingestion, g/d	Data from EPA (1997), in units of g/kg bw/d, assumes bw above	15.4	7.2	10.5	15.7
poultry ingestion, g/d	Data from EPA (1997), in units of g/kg bw/d, assumes bw above	35	16.5	26.1	33.6
other meat, g/d	Data from EPA (1997), in units of g/kg bw/d, assumes bw above	24.5	16.5	20.7	24.4
freshwater/marine fin fish, g/d	Data developed in EPA (2003), based on EPA (2000)	11.6	3	3.8	4.5
freshwater/marine shellfish, g/d	Data developed in EPA (2003), based on EPA (2000)	3.8	1	1.2	1.5

Note:

All exposure factors and approaches developed in EPA's Dioxin Reassessment (EPA, 2003), which relied on EPA's *Exposure Factors Handbook* (EPA, 1997) and U.S.DA (1995).

Table 5.6. Congener-specific and total adult intake estimates of PBDEs (exposures in units of ng/day)

Exposure Pathways	17	28	47	66	85	99	100	138	153	154	183	197	206	209	TOTAL	Fraction
soil ingestion	0.00	0.00	81.26	0.92	4.38	102.92	39.86	7.92	10.67	6.86	2.86	0.08	0.01	104.83	362.55	0.66
soil dermal contact	0.00	0.00	19.50	0.22	1.05	24.70	9.57	1.90	2.56	1.65	0.69	0.02	0.00	25.16	87.01	0.16
Inhalation	0.10	0.32	2.15	0.05	0.04	1.00	0.21	0.00	0.06	0.09	0.00	0.00	0.00	1.45	5.47	0.01
water ingestion	0.01	0.00	0.06	0.00	0.00	0.04	0.01	0.00	0.01	0.00	0.01	0.00	0.00	0.06	0.20	0.00
Milk ingestion	0.01	0.04	5.25	0.05	0.14	5.25	0.88	0.01	0.70	0.35	0.35	0.00	0.00	7.00	20.02	0.04
dairy ingestion	0.00	0.01	1.65	0.02	0.04	1.65	0.28	0.00	0.22	0.11	0.11	0.00	0.00	2.20	6.29	0.01
egg ingestion	0.00	0.00	0.34	0.00	0.03	0.67	0.10	0.00	0.07	0.05	0.00	0.00	0.00	0.17	1.44	0.00
beef ingestion	0.02	0.99	2.49	0.01	0.03	1.99	0.30	0.00	0.30	0.20	0.05	0.00	0.00	0.15	6.53	0.01
pork ingestion	0.00	0.00	1.23	0.00	0.08	1.85	0.23	0.02	0.31	0.15	0.14	0.00	0.00	0.31	4.31	0.01
poultry ingestion	0.00	0.01	2.10	0.01	0.00	4.20	1.05	0.07	0.70	0.04	0.07	0.00	0.00	4.20	12.45	0.02
other meats	0.01	0.16	1.55	0.00	0.05	2.29	0.42	0.03	0.38	0.12	0.10	0.00	0.00	1.17	6.26	0.01
fresh/marine finfish	0.12	0.35	6.96	0.23	0.05	1.97	1.51	0.01	0.23	0.58	0.02	0.00	0.00	1.04	13.07	0.02
fresh/marine shellfish	0.00	0.00	13.68	0.00	0.00	4.56	3.42	0.00	0.00	0.00	0.00	0.00	0.00	0.00	21.66	0.04
TOTAL	0.27	1.89	138.21	1.52	5.88	153.09	57.82	9.96	16.20	10.19	4.39	0.10	0.01	147.74	547.27	
Fraction	0.00	0.00	0.25	0.00	0.01	0.28	0.11	0.02	0.03	0.02	0.01	0.00	0.00	0.27		1.00

Table 5.7. Pharmacokinetic parameters and predicted concentrations of BDEs compared with measurements in blood and milk

| Exposure Doses, Parameters, Results	28	47	99	100	138	153	154	183	209	TOTAL
I. Doses										
soil ingestion, ng/d	0.00	81.25	102.92	39.86	7.92	10.67	6.86	2.86	104.83	357.17
soil dermal contact, ng/d	0.00	19.50	24.70	9.57	1.90	2.56	1.65	0.69	25.16	85.73
Inhalation, ng/d	0.32	2.15	1.00	0.21	0.00	0.06	0.09	0.00	1.45	5.28
Food & water ingestion, ng/d	1.57	35.30	24.47	8.18	0.14	2.91	1.6	0.85	16.3	92.24
II. Parameters										
Dust absorption fraction	0.33	0.69	0.44	0.78	0.67	0.73	0.19	0.48	0.04	---
Other absorption fraction	0.90	0.94	0.78	0.93	0.90	0.90	0.86	0.90	0.90	---
Elimination half-life, yrs	6.0	3.0	5.4	2.9	6.0	11.7	5.8	0.26	0.04	---
III. Results										
Predicted concentration, ng/g lwt	0.3	8.6	15.6	3.3	1.1	3.7	1.1	0.02	0.1	33.8
Observed blood, ng/g lwt	1.2	20.5	5.0	3.9	NA	5.7	NA	NA	NA	36.3
Observed milk, ng/g lwt	1.7	26.0	5.4	5.2	NA	4.8	0.4	0.2	0.4	44.1

Table 5.8. Pharmacokinetic parameters for modeling the body burden impacts to infants via breast feeding, and then to children from food and household exposures

| Time after birth	PBDE half-life, yr	Body lipid fraction	Body weight, kg	Total PBDE dose, ng/day
0	0.40	0.14	3.3	1411
1 mon	0.50	0.16	4.3	1411
2 mon	0.60	0.18	4.6	1411
3 mon	0.70	0.20	6	1411
4 mon	0.75	0.22	6.7	1411
5 mon	0.80	0.23	7.4	1411
6 mon	0.85	0.25	7.9	1411
7 mon	0.90	0.25	8.4	1411
8 mon	0.95	0.24	8.8	1411
9 mon	1.00	0.24	9.2	1411
10 mon	1.05	0.23	9.4	1411
11 mon	1.10	0.23	9.8	1411
12 mon	1.15	0.23	11.3	1411
1 yr, 3 mon	1.30	0.22	11.7	757
1 yr, 6 mon	1.50	0.21	12.5	757
1 yr, 9 mon	1.70	0.20	12.9	757
2 yr	2.00	0.20	13.3	757
3 yr	2.50	0.18	15.6	757
4 yr	3.00	0.16	17.6	757
5 yr	3.50	0.15	19.7	757
6 – 11 yr*	4.00 – 6.00	0.15 – 0.13	24 – 41	450
12 – 19 yr*	6.00	0.13 – 0.15	41 - 64	550

*Note: the time increments of calculation for pk modeling was one year after age 5; body weight and lipid fractions incrementally decreased/increased during that time within the ranges noted. Doses were constant at the values noted.
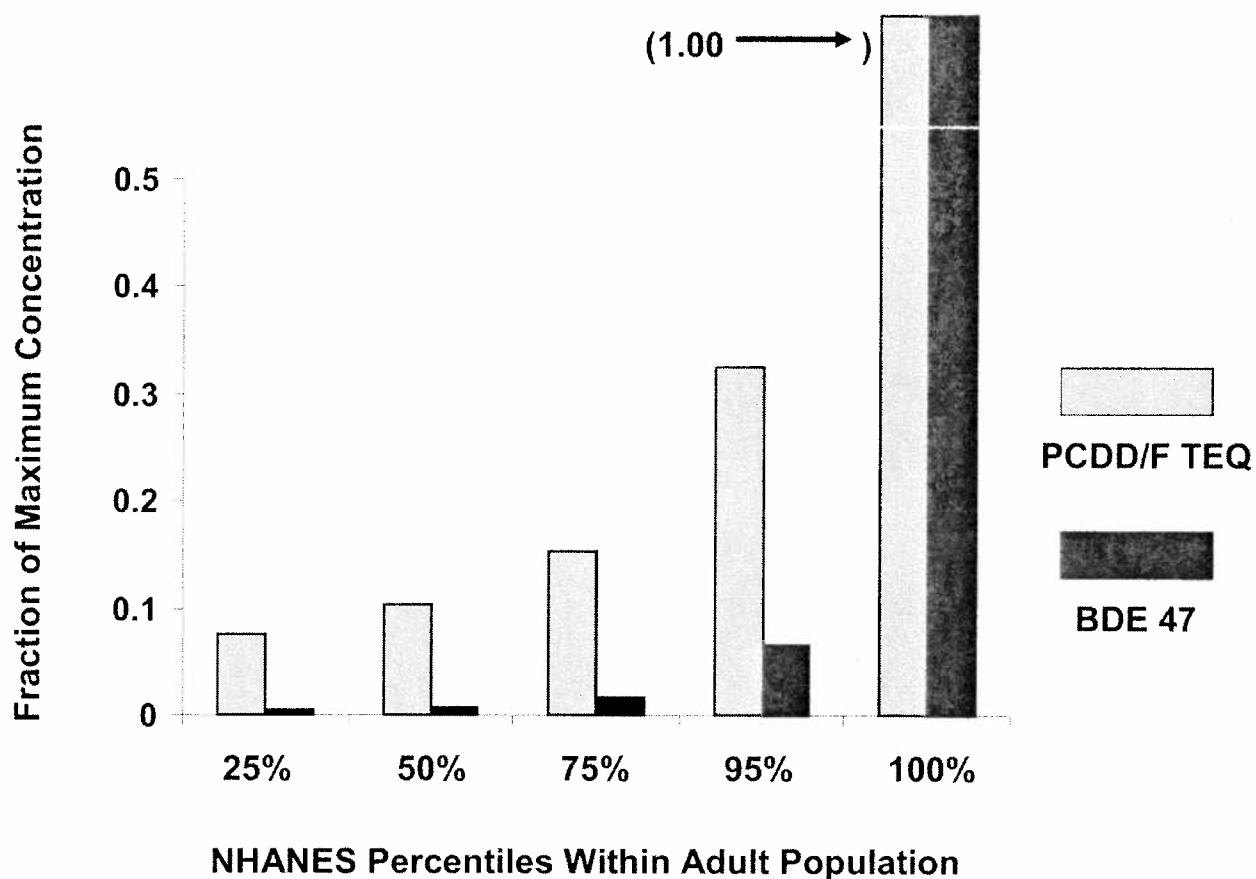
Figure 5.1. Approach for characterizing exposure to polybrominated diphenyl ethers in this report.
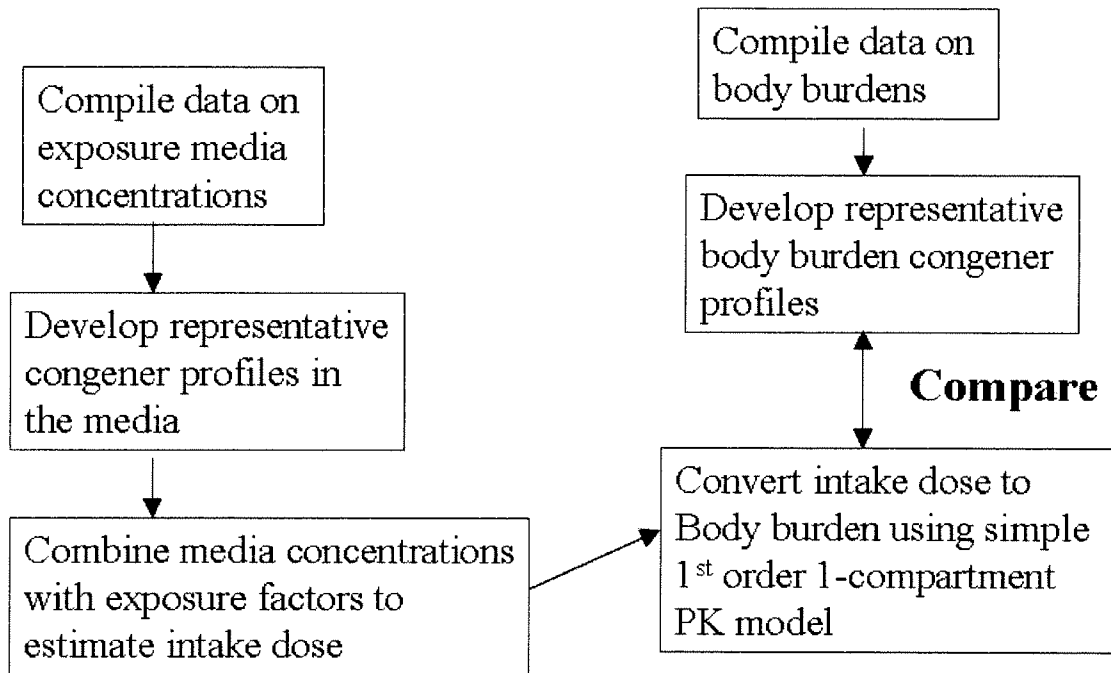
Figure 5.2. Modeled infant and childhood body burdens of PBDEs.
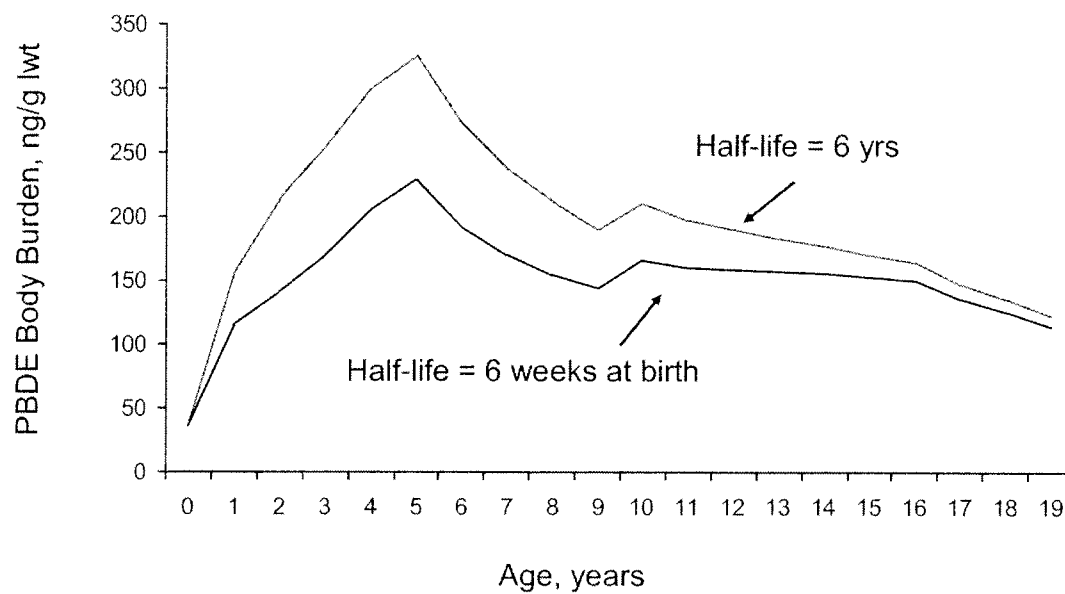
Figure 5.3. The fraction of the maximum concentrations of PCDD/F TEQ and BDE 47 concentrations found at various percentiles within NHANES surveys of these two contaminants in adults.

Note: NHANES data on PBDEs from Sjodin et al. (2008), and data on PCDD/F from Ferriby et al. (2006)