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An Exposure Assessment of Polybrominated Diphenyl Ethers

National Center for Environmental Assessment
Office of Research and Development
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ABSTRACT

Polybrominated diphenyl ethers, PBDEs, are a class of brominated flame retardants that, like other persistent organic pollutants (POPs), have been found in humans, wildlife, and biota worldwide. Unlike other POPs, however, the key routes of human exposure are thought to be from their use in household consumer products, and their presence in house dust, and not from dietary routes. The exposure of Americans to PBDEs was systematically evaluated in this study. The production and lifecycle of the formulated PBDE products were examined. Literature on their fate and presence in the environment was reviewed. Exposure media data on brominated diphenyl ether (BDE) congeners were combined with estimates of adult, childhood, and infant intake factors to estimate a total intake of PBDEs for these receptors. The exposure pathways evaluated included food and water ingestion, inhalation, and ingestion of and dermal contact with house dust. For the adult intakes, a body burden of PBDEs was simulated using a simple pharmacokinetic model. The predicted body burdens were com- pared with representative adult profiles of PBDEs in blood and milk. The adult intake dose of total PBDEs was estimated to be 7.1 ng/kg body weight-day. Comparable published values for total PBDE exposure average 3 to 16 ng/kg-day. The estimated adult intake dose of 7.1 ng/kg-day was predicted to result in a body burden of 31.0 ng/g lipid weight (lwt). This compared to body burdens of 36.3 ng/g lwt found in blood and 44.1 ng/g lwt found in mother's milk. Adult food intake contributes about 10% to total exposure and was estimated in this analysis to be about 0.6 ng/kg-day. Comparable published values for food intake were about 0.5 to 2.0 ng/kg-day. The largest source contributing to U.S. PBDE exposure was found to be house dust, estimated as 6.4 ng/kg-day, and contributing 90% of the overall estimated intakes. These adult intakes and body burdens were derived as central tendency estimates for the US general population. Intakes from housedust exposures (ingestion plus dermal contact) could range up to an order of magnitude higher than calculated for this central tendency considering the highest PBDE concentrations found in dust. The 95th percentile adult body burden of total PBDEs found in the 2003/2004 National Health and Nutritional Evaluation Survey (NHANES) was 291 ng/g lwt, about an order of magnitude higher than the central tendencies of 36.3 ng/g lwt in blood. Children's estimated intakes were higher at 47.2 ng/kg/day for ages 1–5, 13.0 ng/kg/day for 6–11, and 8.3 ng/kg/day for 12–19. Infant intakes due to ingestion of mother's milk were the highest at 141 ng/kg/day. Other exposures of interest examined in this report include fetal exposures, occupational exposures, and unusually high exposures in the general population.

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ABBREVIATIONS AND ACRONYMS

ABS	acrylonitrile butadiene styrene
BAF	bioaccumulation factor
BCF	bioconcentration factor
BDE	brominated diphenyl ether; used for congener designation as in BDE 47
BFR	brominated flame retardant
BMF	biomagnifications factor
BMP	biomagnifications potential
BW	body weight
CARB	California Air Resources Board
CDC	Centers for Disease Control and Prevention
CDD/Fs	chlorinated dibenzodioxins and dibenzofurans
CHDS	Child Health and Development Studies
CPSC	Consumer Products Safety Commission
CSFII	Continuing Survey of Intakes by Individuals
DE	diphenyl ether
decaBDE	since there are only 10 available spots for bromines, this is synonymous with the BDE congener with 10 bromines, which is BDE 209. decaBDE is also the name of the formulation dominated (>97%) by BDE 209
diBDE	the homologue group of BDE congeners with two bromines
DL	detection limit
DBDPE	decabromodiphenyl ethane
DBDPO	[^{14}C]decabromodiphenyl oxide
DSBG	Dead Sea Bromine Group
dwt	dry weight
EEE	electronics and electrical equipment
EF	emission factor
EP	epoxy resin
EPA	Environmental Protection Agency
EU	European Union
EWG	Environmental Working Group

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ABBREVIATIONS AND ACRONYMS (continued)

FPUF	flexible polyurethane foam
FSA	Food Standards Agency of the UK
GC/GC-IDTOFMS	gas chromatography and isotope dilution time-of-flight mass spectrometry
GC-IDHRMS	gas chromatography and isotope dilute high resolution mass spectrometry
GC/MS	Gas Chromatography / Mass Spectrometry
H	Henry's Constant
HBCD	hexabromocyclododecane
heptaBDE	the homologue group of BDE congeners with seven bromines
hexaBDE	the homologue group of BDE congeners with six bromines
Hi-Fi	high fidelity
HIPS	impact polystyrene
HRGC	high resolution gas chromatography
HRMS	high resolution mass spectrometry
IT	information technology
K_{oa}	octanol air partition coefficient
K_{ow}	octanol water partition coefficient
LOQ	limit of quantification
lwt	lipid weight
MSW, MSWLF	municipal solid waster, MSW landfill
MoBPXDD/Fs	monobromo-polychlorinated dibenzo-*p*-dioxins and dibenzofurans
monoBDE	the homologue group of BDE congeners with one bromine
NA	not analyzed, or not available (as in data not available)
NAS	National Academies of Science
ND	not detected
NEW	Northwest Environment Watch
NHANES	National Health and Nutritional Examination Survey
nonaBDE	the homologue group of BDE congeners with nine bromines
NSSS	National Sewage Sludge Survey

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ABBREVIATIONS AND ACRONYMS (continued)

octaBDE	the homologue group of BDE congeners with eight bromines; this also refers to a commercial formulation of PBDEs dominated by hepta- and octaBDEs
PBDD/Fs	polybrominated dibenzo-*p*-dioxins and dibenzofurans
PBDE	polybrominated diphenyl ether
PBT	persistent and bioaccumulative toxic; in the context of describing types of plastics to which PBDEs are added, it also could be an abbreviation for polybutylene terephthalate
PC	personal computer
PCB	polychlorinated biphenyl
PCDD/Fs	polychlorinated dibenzo-*p*-dioxins and dibenzofurans
PE	polyethylene
PERC	perchloroethylene
pentaBDE	the homologue group of BDE congeners with five bromines; this also refers to a commercial formulation of PBDEs dominated by penta BDE congeners
PK	pharmacokinetic
POP	persistent organic pollutants
PP	polypropylene
P_S	solid phase vapor pressure in Pa
PS-1	refers to the high volume air sampler
PUF	polyurethane foam
PUR-H	polyurethane hard foam
RCRA	Resource Conservation and Recovery Act
RD	reductive dehalogenase
RfD	reference dose
SML	sea-surface microlayer
SD	standard deviation
SRM	Standard Reference Material
SSW	subsurface seawater
STP	sewage treatment plant

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ABBREVIATIONS AND ACRONYMS (continued)

Σ_9BDEs	summation of the concentrations of 9 key BDE congeners; other numerical subscripts would indicate a different number of congeners
TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TCE	trichloroethylene
TEQ	toxic equivalent; used in the context of dioxin, where TEQ is the concentration of a mixture of dioxin-like compounds toxically equivalent to 2,3,7,8-TCDD
tetraBDE	the homologue group of BDE congeners with four bromine
TRI	Toxics Release Inventory
triBDE	the homologue group of BDE congeners with three bromines
TV	television
UC	University of California
UNEP	United Nations Environmental Program
UPE	unsaturated polyesters
U.S.	United States
UK	United Kingdom
UV	ultraviolet
VCCEP	Voluntary Children's Chemical Evaluation Program
VCR	video cassette recorder
V_p	vapor pressure
WHO	World Health Organization
WS	water solubility
WWF	World Wildlife Fund
wwt	wet weight
WWTP	waste water treatment plant
WTC	World Trade Center
XAD	As in XAD resin, a highly absorbent resin used in continuous sampling of organic materials, especially for monitoring of pollutants in gas streams.
XRF	X-ray fluorescence

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ABBREVIATIONS AND ACRONYMS (continued)

Units of Measurement

atm	atmospheres
C	centigrade
cm	centimeter
g	gram
h, hr	hour
kg	kilogram
km	kilometer
L	liter
m	meter (used mostly as m^3, as air volume, but also as a length measure)
mg	milligram
µg	microgram
µL	microliter
µm	micrometer (also micron)
µM	micro molar
mL	millileter
mm	millimeter
MMT	million metric tons
MT	metric tons
mol	mole, in chemistry
ng	nanogram
Pa	Pascals
pg	picogram
pmol	picomole
s	seconds
yr	year

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PREFACE

The U.S. Environmental Protection Agency (EPA) formed a working group composed 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, and updated that plan in December 2008. EPA reports regularly on progress in completing the activities identified in the project plan, with the most recent status report issued in March 2008 and a planned update for 2010. The Web site that describes this working group, including the project plan and status reports, 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 life cycle 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 flame retardant 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 209 possible PBDE compounds; each are termed *congener* and are assigned a specific brominated diphenyl ether (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 BDE 99 (35−50%), BDE 47 (25−37%), BDE 100 (6−10%), BDE 153 (3−5%), and BDE 154 (2−4%). The octa formulation is composed of BDE 183 (40%), BDE 197 (21%), BDE 203 (5−35%), BDE 196 (8%), BDE 208 (10%), BDE 207 (7%), BDE 153 (5−10%), and BDE 154 (1−5%). The deca formulation is dominated by BDE 209 (97.5%), with the remainder being BDE 206 (2.2%), BDE 207 (0.24%), and BDE 208 (0.06%).

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. The carcinogenic potential of some PBDEs have been studied. In a review of toxicological studies as part of a toxicological review for EPA's Integrated Risk Information System (2008), EPA has found that the data for decabromodiphenyl ether (the commercial formulation composed mostly of BDE 209) support a finding of "suggestive evidence of carcinogenic potential" according to EPA's *Guidelines for Carcinogen Risk Assessment*. For BDE congeners BDE 99, BDE 153, and

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BDE 47, EPA has found that the data support a finding of "inadequate information to assess carcinogenic potential."

Production, Use and Release:

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/year 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 U.S. 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. However, in December of 2009, the EPA announced the phase out of decaBDE, with production, importation, and sales of decaBDE for most uses in the United States to end by 12/31/2012, and all uses to end by 12/31/2013.

A limited life cycle 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 disposal of products containing PBDEs. Figure E-1 is a summary of a generalized annual life cycle of PBDE in the United States: production, use, disposal, recycling, and sewage treatment. The Toxics Release Inventory (TRI) reports a total release of 32.2 MT of decaBDE to the air, land, and water in 2007. 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 a drop in releases in 2003 to 36.3 MT, followed by an increase in 2004 to 44.8 MT, and then declines in 2005, 2006, and 2007.

Much of the manufactured PBDEs have been used to make plastic consumer products flame retardant. A mathematical model was used to estimate the amount of PBDEs that may volatilize from the treated product to the air (mostly indoor air) during product use in the United States. It was estimated that 133.5 kg/year of BDE 47; 24.1 kg/year of BDE 99; 8.5 kg/year of BDE 209; 7.8 kg/year of BDE 28; and 3 kg/year each of BDE 66, BDE 153, and BDE 183

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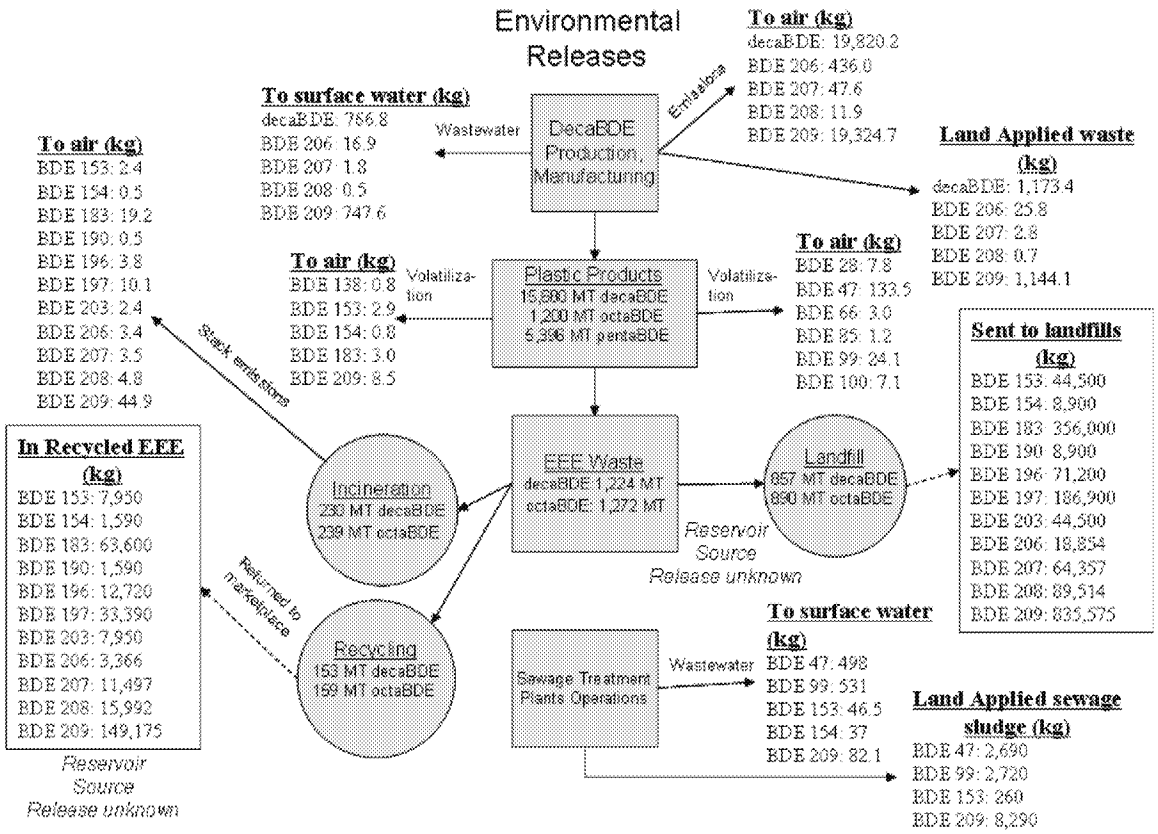

Figure E-1. Summary of a generalized annual life cycle of PBDE in the United States: production, use, disposal, recycling, and sewage treatment.

may volatilize to the air using this model. These estimates can, at best, be considered initial estimates that are unverifiable. However, they are consistent with chamber studies in that the greater the vapor pressure, and the lower the octanol air partition coefficient, the more likely the BDE congener will volatilize from the plastic product into the air. In this regard, BDE 47 has the greatest tendency to volatilize from plastic. Although uncertain, the estimates are judged to have provided an order of magnitude estimate of the volatile release of these flame retardants from plastic products in use. Another release mechanism noted is the flaking off, crumbling, and/or general physical removal of PBDEs from treated foam products. Although such a loss mechanism may be significant, there is no way to quantify this avenue of loss, or even qualitatively compare it to other loss mechanisms.

Releases can occur from the disposal of electronic and electrical equipment (EEE) waste. In the United States, an estimated 2.4 million metric tons (MMT) of EEE waste was generated in 2005. Of this amount, 0.3 MMT was recovered for recycling, 0.42 MMT was incinerated in

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municipal waste combustors, and 1.68 MMT was buried in municipal solid waste (MSW) landfills. Extrapolating to U.S. from limited European studies suggests that the EEE waste contains about 1,224 MT decaBDE and 1,272 MT of octaBDE. However, U.S. EEE waste has yet to be adequately characterized for PBDE content. Losses of PBDEs in recycled products were not estimated in the life cycle analysis. The landfilling of EEE waste was assumed not to result in environmental releases of PBDEs, because of the careful conditions (liners, treated leachate) required by law for MSW landfills in the United States. However, the life cycle analysis suggests that MSW landfills are significant environmental reservoirs for PBDEs. It is possible that 1,747 MT PBDEs were disposed of in U.S. landfills in 2005 alone. Based on measured concentrations of decaBDE in EEE products and the destruction efficiency from MSW incinerators with a high degree of pollution control, releases of BDE 209 totaled 44.9 kg/year.

Sewage treatment plant operations also result in environmental releases of PBDEs. The untreated sewage entering the treatment plant contains significant amounts of PBDEs. Both sewage sludge from the treatment plant processes as well as the treated sewage treatment plant effluent contain appreciable amounts of PBDEs. The annual land application of sewage sludge as a soil amendment practice results in 0.26, 2.69, 2.72, and 8.29 MT/year of BDE 153, BDE 47, BDE 153, and BDE 209, respectively, being applied to land in the United States. The life cycle analysis estimates that 37, 46.5, 82.1, 498, and 531 kg/year of BDE 154, BDE 153, BDE 209, BDE 47, and BDE 99, respectively, are discharged annually into surface waters in the United States from sewage treatment plant effluent.

Environmental Fate

PBDEs are ubiquitous environmental contaminants. Widespread dispersion of BDE congeners in the environment is governed by their respective physical and chemical properties. The atmosphere is the primary transport media, and soils and sediments are environmental sinks. Transport can occur over relatively long distances, >1,000 km. Evidence for this comes from the presence of PBDEs in the polar environments, and in the tissues of deep ocean-dwelling whales and other marine mammals who spend a significant portion of their lives far from anthropogenic sources. PBDEs are lipophilic and hydrophobic compounds and readily bioaccumulate into terrestrial and aquatic food webs. This tendency has resulted in extensive accumulation of PBDEs in a wide variety of birds, fish, insects, and aquatic and terrestrial mammals reflective of

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dietary exposures through the food chain. 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 fish usually follows 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, BDE 209 has ranged from <1 up to 88% of total PBDE body burden, depending on the dietary source of BDE 209. The PBDE body burden of birds is highly influenced by diet. For example, in a study of Peregrine Falcon eggs, the rank order by PBDE concentration in eggs (high to low) was: BDE 153 > BDE 99 > BDE 100 > BDE 154 > BDE 209 > BDE 183 > BDE 197 > BDE 47 > BDE 207. BDE 47 was a minor constituent in Peregrine Falcon eggs (on average only 4.4%). The Peregrine Falcon consumes a diet consisting of medium sized birds such as doves, waterfowl, songbirds, waders, and pigeons. The Cormorant nests in swampy areas or near large bodies of water such as rivers and lakes, and its diet consists mainly of fish. BDE 47 contributed approximately 42% of the total body burden of PBDEs in the Cormorant. In the Common Buzzard, the most prevalent congeners detected were BDE 153 (29% of total), BDE 99 (23%), and BDE 47 (22%). The Common Buzzard preys mainly on small mammals (mice, voles, rabbits, squirrels, rats, and 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. Terrestrial mammals also tend to accumulate brominated PBDEs in relation to their diet. For example, the red fox is a top terrestrial predator that mainly consumes voles, rabbits, squirrels, and mice as prey, and BDE 209 dominated the total body burden. Maritime fish-eating bears displayed a BDE congener pattern dominated by BDE 47 followed by BDE 209 > BDE 99 > BDE 100 > BDE 153. In the interior meat-eating bears, the tissues were dominated by BDE 209 followed by BDE 206 > BDE 47 > BDE 207 > BDE 208.

Once released into the air, PBDEs partition between the vapor and particle 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. Photolysis (degradation by sunlight) in air is an important atmospheric removal process for BDE 47 and BDE 99. In contrast, the photolysis of BDE 209 in air is a minor atmospheric removal process. Atmospheric wet and dry surface deposition of BDE 209 is the most important atmospheric removal pathway. This is because BDE 209 predominantly exists bound to particles in air. Evidence suggests that BDE 209 can be degraded by ultraviolet

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light (i.e., photolysis) to form lower-brominated BDEs, and this may be an important degradation pathway in 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 decaBDE has induced reductive debromination of decaBDE in vitro to yield octa- and heptaBDE after a contact time of 2 months. The octa- and heptaBDE did not further debrominate in the presence of the microbe—even after 1 year. The researcher of this study concluded that the microorganism was specific to the degradation of decaBDE and is incapable of debrominating lower-brominated PBDE compounds. In another study, BDE 209 was debrominated in vitro to yield BDE 206, BDE 207, and BDE 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.

Several studies provide evidence that in vivo metabolic debromination occurs in fish, birds, and mammals. 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. Debromination in fish has also been observed in the wild. In a study at the outfall to a sewage treatment plant, sunfish bioaccumulated BDE 209. It was observed that the sunfish tissues contained BDE congeners not present in the sewage treatment plant effluent or in the surrounding aquatic environment. These were two octa- (BDE 201 and BDE 202) and three hepta- (BDE 188, BDE 184, and BDE 179) congeners. The investigating scientists proposed that the presence of these congeners in the sunfish was supportive of in vivo synthesis from the metabolic debromination from BDE 209.

In another study, Sprague-Dawley rats were fed a commercial formulation of decaBDE. Evidence of metabolic debromination of BDE 209 to lower congeners was observed from an apparent 160% increase in the tissue concentration of BDE 197, BDE 201, and BDE 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.

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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 accounted for 4.2%, 21%, and 30% of serum PBDE levels in house cats consuming canned, mixed, and dry food, respectively. 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, 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 food-eaters. The authors regarded these data as possible evidence for the metabolic debromination of BDE 209 to form BDE 207.

Human Exposure and Biomonitoring

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 1970s. Environmental time-trends can be observed from lake sediment core studies and archived animal tissue samples. The sediment cores show 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 pentaBDE formulation has garnered the most concern because it appears to be the major contributor to current environmental, biotal, and human body levels. Even with both the penta and octa formulations having been withdrawn from the U.S. market, past use and possible debromination of higher-brominated congeners (e.g., BDE 209) by photolytic or biological mechanisms to form

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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 U.S. 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 EPA's *Exposure Factors Handbook*.

As lipophilic contaminants, PBDEs bioaccumulate in the lipids of organisms. This generalization is less true for BDE 209; it is not as lipophilic as other BDEs despite it being found and quantified in lipids of animals and humans. While the term "bioaccumulation" may also be questionable for BDE 209, there is little doubt that it occurs in human and animal tissues (see discussion above on concentrations in animal tissues). In humans, lipid concentrations are typically measured in blood and breast milk, although adipose tissue concentrations have also been measured. Studies measuring BDE 209 in both blood and mother's milk show lower concentrations in mother's milk, suggesting limited transport for this congener within the body. 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-2 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*

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critical, congeners as there is, for example, with dioxin-like compounds. Some studies focus on as few as 4 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 BDE 28, BDE 47, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 209. Tables E-1 and E-2 provide the results of this exposure exercise.

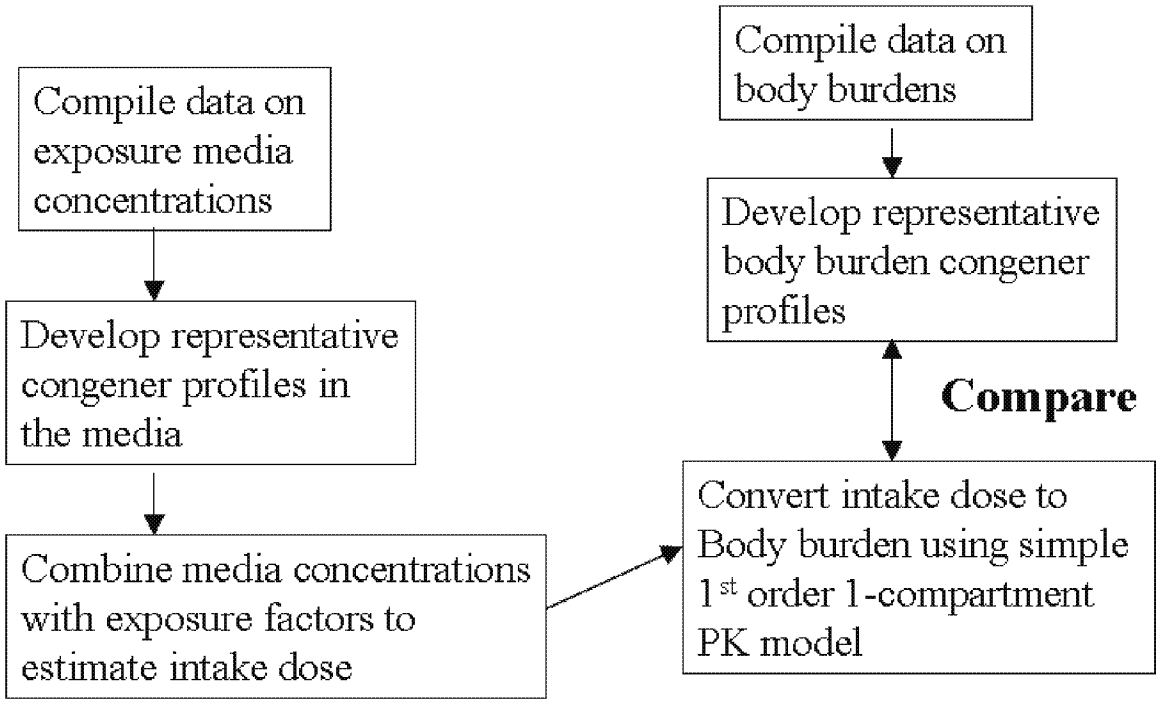

Figure E-2. Approach for characterizing exposure to polybrominated diphenyl ethers in this report.

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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	1,857	2,352	911	181	243	156	60	2,394	8,275
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	0.01	0.20	0.04	0.04	0.0007	0.006	0.015	0.001	0.008	0.32
Beef, ng/g wwt	0.0006	0.009	0.01	0.001	ND	0.003	0.0009	0.0004	0.003	0.028
Pork, ng/g wwt	0.0007	0.031	0.033	0.005	0.0009	0.007	0.003	0.02	0.016	0.13
Poultry, ng/g wwt	0.0003	0.005	0.007	0.001	ND	0.001	0.001	0.001	0.004	0.020
Dairy, ng/g wwt	0.001	0.04	0.032	0.005	ND	0.004	0.002	0.001	0.675	0.79
Eggs, ng/g wwt	ND	0.009	0.018	0.007	ND	0.007	0.004	ND	0.034	0.084

*Totals may include congeners other than those listed and are greater than the sum of the studied congeners; see Chapter 4 for more detail.

--- Means no data available; ND = data available but not detected; and wwt = wet weight.

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Table E-2. Predicted adult 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	0.22	18.98	7.98	4.39	0.03	0.70	0.46	0.66	10.75	44.17
II. Pharmacokinetic Results										
Predicted conc., ng/g lwt	0.1	8.6	12.5	3.9	1.3	3.9	0.6	<0.1	<0.1	31.0
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

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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 82 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 ng/g, 13,882 ng/g, and 4,213 ng/g, respectively also compare well to the 8,275 ng/g dwt estimate. Based upon all these studies, a range of average concentrations in dust may be about 4000 to 14,000 ng/g dwt. BDE 209 dominates the dust profile, and recent studies have found unusually high levels of BDE 209, including concentrations in the hundreds of thousands to over two million of ng/g dwt in the United Kingdom, and in the hundreds of thousands in one study in the United States. The indoor air profile came from this study of Boston homes in which the authors determined geometric mean concentrations for three locations (personal, bedroom, living room). The average of the three 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

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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 measurement of 31 samples, each a composite of 10 samples of the same food type taken from markets in Dallas, Texas for a total of 310 samples collected, Food types sampled include meat, dairy, fish, and eggs. Thirteen congeners, including BDE 209, were measured. The average total concentrations (on a wet weight, not lipid weight, basis) used from this study include the following: 0.32 ng/g in finfish, 0.028 ng/g in beef, 0.13 ng/g in pork, 0.020 ng/g in poultry, 0.79 ng/g in dairy, and 0.084 ng/g in eggs. A total concentration of 5.7 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 are 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 are from Europe. The study selected as the representative blood profile came from the National Health and Nutritional Examination Study (NHANES) from 2003/2004. The geometric mean total PBDE concentration of all adults from this study was 36 ng/g lwt; the 95th percentile was 291 ng/g lwt. BDE 209 was not quantified in individual samples of NHANES; hence, Table E-2 lists it as NA. However, in the literature publication on this data set, it was stated that BDE 209 concentrations in pooled samples from this set of NHANES samples had a concentration of 2 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 congeners are BDE 99 and BDE 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 5 ng/g lwt, with the exception of one case. In this case study, which entailed taking measurements from a family of four, 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.

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Unlike human blood data, there was 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, 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 included 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 years for congeners BDE 47, BDE 99, BDE 100, BDE 138, BDE 153, and BDE 154. The second reference derived significantly smaller half-lives for the higher-brominated congeners BDE 183 and BDE 209—half-lives of 0.26 year and 0.041 year, respectively. The absorption fraction for BDEs in dust ranged from 0.04 (BDE 209) to 0.78 (BDE 100). These were derived from a study of the bioavailability of BDEs in dust when fed to rats. The absorption fractions 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. The exposure pathways of dust ingestion and dust-dermal contact dominated total adult exposure, explaining 90% of the 492 ng/day total (equivalent to 7 ng/kg-day for the 70 kg adult). The congeners BDE 47, BDE 99, and BDE 209 dominated the exposure intake: each explained between 25 and 29% of total intakes, with other congeners explaining the remaining intakes. Using the estimated adult dose of 492 ng/day, a blood serum concentration of BDE was predicated. The predicted concentration of total BDEs of 31.0 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 six of nine congeners. However, predicted

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concentrations of BDE 47 and 209 were less than NHANES measurements and the predicted concentration of BDE 99 was more than NHANES measurements. The cause of the differences between predicted and measured values for these congeners was unknown, but could point to the inappropriate assignment of elimination half-life, or in the case of BDE 99, metabolic debromination.

While predictions of adult body burdens were 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. Consequently, the dose via food/water ingestion and inhalation might be considered reasonably certain, for purposes of this discussion. However, food/water ingestion and inhalation explained less than 20% of the body burden, based upon the estimate of total exposure derived using the pharmacokinetic model. The remainder of the estimated exposure likely came from house dust through the pathways of ingestion and dermal contact, or some other, unknown source. Assignment of dust contact rates in combination with concentrations found in house dust lead to exposures that resulted in reasonably accurate predictions of measured body burdens. The assertion that dust is a major source of PBDE exposure is supported by the circumstantial evidence that PBDE concentrations in U.S. house dust are generally ten times higher than concentrations reported in European studies and that European body burdens (ca 10 ng/g lwt) are as much as ten times lower than U.S. body burdens (30 – 100 ng/g lwt).

In addition to adult body burden measurements and intake dose estimates, exposures for other populations were also examined. These unique exposures included fetal exposures, infant exposures via breast milk, childhood exposures, occupational, and unusually high exposures at the high end of the general population. Key findings from these examinations are summarized below.

PBDE Body Burdens in U.S. Children: Body burden data, as well as intake and body burden impact modeling, suggest that infants and toddlers have higher exposures than older children or adults. In paired sampling studies, including mother and child, the child's body burden has exceeded by the mother's by about a factor of 4, exceeding 100 ng/g lwt total PBDEs

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in some cases. Higher levels in toddlers were attributed to ingestion of mother's milk and high exposures to house dust.

PBDE Fetal Body Burdens: Limited data from fetal tissue and numerous studies including measurements of BDE congeners from umbilical cord blood support the conclusion that the fetus is exposed to PBDEs through the mother. In some paired studies including both maternal and umbilical cord serum, there is a suggestion that the fetus may be less exposed than the mother as lower concentrations of BDE congeners in umbilical cord as compared to maternal serum were found. This trend of lower concentrations were seen mostly for higher-brominated congeners in two of these studies, where this general observation could be made, but in four other studies reviewed, similar, if not slightly higher, concentrations of all congeners were seen in umbilical cord serum.

Intake Rates for Infants and Children: Intake modeling for the breast milk pathway combining measured milk concentrations and infant ingestion of human milk led to an intake of 1,411 ng/day in this study. 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. It is lower than intake estimates for children derived in this study, which were 47.2 ng/kg-day for ages 1−5 (assuming 15 kg bw), 13.0 ng/kg-day for ages 6−11 (assuming 30 kg bw), and 8.3 ng/kg-day for ages 12−19 (assuming 58 kg bw). The estimated adult intake rate was 7.1 ng/kg-day.

Occupational Exposure: Limited occupational data support the observation that individuals in occupations that would lead to higher exposures to specific congeners have higher concentrations of PBDE congeners in their blood than the general population.

High-End Exposures in U.S. Population: Even in background adult populations, there are individuals experiencing very high exposures. As noted earlier, a range of body burdens of total PBDEs found in the literature was 30-100 ng/g lwt, and the 95th percentile found in NHANES was 291 ng/g lwt. Modeling exercises undertaken to examine this hypothesis suggest that even the highest dust concentrations might not be able to explain the highest body burdens found.

This report has quantified central tendency intake estimates and body burdens of PBDEs, and found that dust-related exposures best explain body burdens. However, this examination of unusually high exposures in the general population suggests the possibility that there are other exposures not identified in this assessment, or exposures that were identified but not quantified

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properly for all individuals of the population. These exposures not identified or improperly quantified are affecting a small percentage of individuals at the high end (upper 5%, perhaps) of the U.S. population. This might be the most important uncertainty identified in this report.

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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 209 possible PBDE compounds, each termed *congeners*. Each is assigned a specific brominated diphenyl ether (BDE) number (note: in this document, the abbreviation PBDE was used to denote the class of brominated flame retardants, while BDE was used in the context of PBDE congeners). For example, there are 42 tetrabromodiphenyl ether congeners (those with four 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 10 Br positions occupied. The penta and octa formulations were voluntarily withdrawn from the 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 1/1/2007.[1]

[1]See http://www.emfacts.com/weblog/index.php?p=547.

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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		BDE 118	2,3',4,4',5-BDE
BDE 3	4-BDE	BDE 119	2,3',4,4'6-BDE
II. DiBDE		BDE 126	3,3',4,4',5-BDE
BDE 7	2,4-BDE	BDE 138	2,2',3,4,4',5'-BDE
BDE 8	2,4'-BDE	BDE 140	2,2',3,4,4',6-BDE
BDE 11	3,3'-BDE	**VI. HexaBDE**	
BDE 12	2,6-BDE	BDE 153	2,2',4,4',5,5'-BDE
BDE 13	3,4'-BDE	BDE 154	2,2',4,4',5,6'-BDE
BDE 15	4,4'-BDE	BDE 155	2,2',4,4',6,6'-BDE
III. TriBDE		BDE 166	2,3,4,4',5,6-BDE
BDE 17	2,2',4-BDE	**VII. HeptaBDE**	
BDE 25	2,3',4-BDE	BDE 181	2,2',3,4,4',5,6-BDE
BDE 28	2,4,4'-BDE	BDE 183	2,2',3,4,4',5',6-BDE
BDE 30	2,4,6-BDE	BDE 190	2,3,3',4,4',5,6-BDE
BDE 32	2,4',6-BDE	**VIII. OctaBDE**	
BDE 33	2',3,4-BDE	BDE 196	2,2',3,3',4,4',5',6-BDE
BDE 35	3,3',4-BDE	BDE 197	2,2',3,3',4,4',6,6'-BDE
BDE 37	3,4,4'-BDE	BDE 203	2,2',3,4,4',5,5',6-BDE
IV. TetraBDE		**IX. NonaBDE**	
BDE 47	2,2',4,4'-BDE	BDE 206	2,2',3,3',4,4',5,5',6-BDE
BDE 49	2,2',4,5'-BDE	BDE 207	2,2',3,3',4,4',5,6,6'-BDE
BDE 66	2,3',4,4'-BDE	BDE 208	2,2',3,3',4,5,5',6,6'-BDE
BDE 71	2,3',4',6-BDE	**X. DecaBDE**	
BDE 75	2,4,4',6-BDE	BDE 209	2,2',3,3',4,4',5,5',6,6'-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		

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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 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 pentaBDE 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 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. The carcinogenic potential of some PBDEs have been studied. In a review of toxicological studies as part of a toxicological review for EPA's Integrated Risk Information System (U.S. EPA, 2006), EPA has found that the data for decabromodiphenyl ether (the commercial formulation composed mostly of BDE 209) support a finding of "suggestive evidence of carcinogenic potential" according to EPA's *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005). For BDE congeners BDE 99, BDE 153, and BDE 47, EPA has found that the data support a finding of "inadequate information to assess carcinogenic potential" (U.S. EPA, 2005).

EPA has formed a working group composed 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 PBDEs.[2] 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

[2]See http://www.epa.gov/oppt/pbde.

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Americans to this class of persistent organic pollutants. That document was updated in December of 2008 and a further update is planned for 2010.

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 fetal, infant, children, and adult exposures to PBDEs (see Chapter 5).

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2. PRODUCTION, USE, AND LIFE CYCLE OF POLYBROMINATED DIPHENYL ETHERS

2.1. INTRODUCTION

Polybrominated diphenyl ethers (PBDEs) 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 flame retardants in the environment has heightened concerns for adverse ecological and human health risks. PBDEs are a class of aromatic compounds intentionally manufactured to retard the combustibility of treated materials. When fire occurs, the PBDE formulations utilize vapor 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 (FPUFs) used in upholstery stuffing for furniture and car seats, electronic components, electrical components, and plastics used in the casings of televisions (TVs), personal computers (PCs), and other electronic equipment.

The purpose of this chapter is to describe the production and uses of PBDEs, and to present a basic life cycle 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 flame retardants in a number of plastic resins and finished products. Section 2.4 presents a generalized life cycle analysis for the United States that derives estimates of environmental releases of specific brominated diphenyl ether (BDE) congeners based on production, the uses of PBDE-treated products, and estimates the amounts that may be recycled or disposed of in landfills and incinerators once PBDE-treated products become functionally obsolete. Section 2.5 presents a summary and the key findings from this life cycle analysis. Because of significant limitations and gaps in existing data, the life cycle analysis presented in this chapter should be regarded as an approximate assessment. However, the life cycle 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.

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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 known as penta-, octa-, and decaBDE. Each commercial formulation is manufactured through the chemical reaction of bromine with diphenyl oxide, (also known as 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-brominated PBDE 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 10 homologue groups identified by the preface mono-, di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and decaDBE. 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.

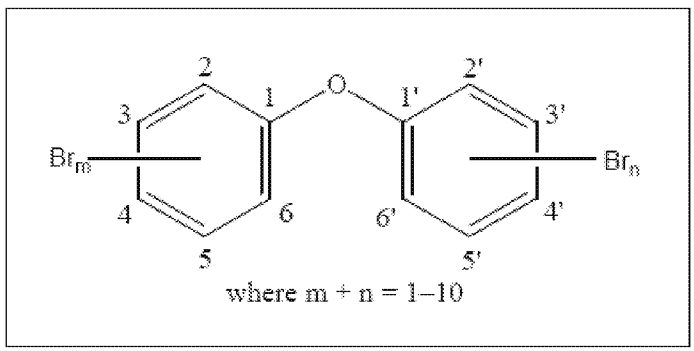

Figure 2-1. General structure of polybrominated diphenyl ethers.

Commercial formulations are mixtures of PBDE congeners with penta-, octa-, and decaBDE having a bromine content of about 70.8%, 79%, and 83%, respectively (EU, 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

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Table 2-1. Approximate BDE homologue- and congener-specific content of PBDE commercial formulations

Congener identity	Commercial formulations of PBDE fame 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 206			2.2
BDE 207		7	0.24
BDE 208		10	0.06
Total NonaBDE		8–14	–2.5
BDE 209		0–3	97.5

Sources: EU (2001, 2002, 2003); Kemmlein et al. (2005); Peele (2004); Palm et al. (2004); IPCS (1994); La Guardia et al. (2006).

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approximate BDE congener compositions of the three commercial formulations. The composition of the three commercial formulations change with time and manufacturer, and Table 2-1 shows general congener composition. 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 tetraBDE (24–38%) homologues, with hexaBDE 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 nonaBDE 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, 97.5% of the formulation is composed of BDE 209, with the remainder being BDE 206 (2.2%), BDE 207 (0.24%), and BDE 208 (0.06%). BDE 209 is the dominant congener, and is a signature of this formulation. The congener composition of the commercial decaBDE product has become progressively dominated by the BDE 209 congener over time. For example, in a 1986 toxicity evaluation, the National Toxicology Program noted that BDE 209 was approximately 96% (or greater) in the commercial decaBDE product (NTP, 1986).

Because of mergers and acquisitions, PBDE flame retardant formulations are produced worldwide by three chemical manufacturers: Albemarle, Chemtura, and ICL Industrial Products. In the United States, all PBDE flame 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-flame retardants. ICL Industrial Products acquired the DSBG in 2005. The major producer of PBDE products in the United States was the Great Lakes Chemical Corporation, which sold flame retardants under the Firemaster brand name. The Great Lakes Chemical Corporation merged with Crompton Chemical to form Chemtura. Chemtura produces flame retardant products composed 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 flame retardant. Firemaster-550 is technically not

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enriched with PBDE, but the exact active chemical ingredients remain proprietary. However, Stapleton et al. (2008) analyzed dust samples in homes in the Boston area and identified these brominated components of Firemaster-550: 2-ethylhexyl 2,3,4,5-tetrabromobenzoate (TBB) and (2-ethylhexyl)tetrabromophthalate (TBPH), the latter compound being a brominated analogue of di(2-ethylhexyl)phthalate (DEHP). Albemarle produces a decaBDE-formulated flame retardant under the brand name SAYTEX 102E Flame Retardant. 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 12/31/2004 (Birnbaum and Hubal, 2006). The EPA announced the phase-out of decaBDE in December of 2009 (U.S. EPA, 2009c). The three companies producing PBDEs (noted above) have committed to end production, importation, and sales of decaBDE for most uses in the United States by 12/31/2012, and to end all uses by the end of 2013.

Production figures for PBDE manufacture are not readily available. The PBDE industry usually reports only on market demand for PBDE commercial formulations, which likely translates into sales volume. The latest reporting year for market demand is 2001 (Hites, 2004). The market demand for decaBDE in 2001 within the Americas was approximately 24,500 metric tons. The Americas includes North, Central, and South America, combined. There are no further breakdowns available by country. 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 (analysis of data in Hites, 2004). Two-thirds of all decaBDE produced globally are consumed in North America and Asia (analysis of data in Hites, 2004).

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 U.S. manufacturer of penta- and octaBDE phased out production of these products at the end of 2004. Additionally, several states enacted individual legislation banning the use of penta-, octa- and decaBDE formulations as summarized below.

- As of 6/1/2006, California and Maine prohibited the manufacture, distribution, and processing of products containing penta- and octaBDE flame retardants (CalEPA, 2006; NCEL, 2005).

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- As of 1/1/2006, Hawaii, Illinois, and Michigan prohibited the manufacture, processing, and distribution of products containing more than 0.1% of the PBDE formulations penta- and octaBDE (Illinois EPA, 2006; NCEL, 2005).

- Minnesota similarly prohibited the manufacture and uses of penta- and octaBDE effective 1/1/2008 (NCEL, 2007).

- Washington State banned the uses of penta-, octa-, and of decaBDE in mattresses effective 1/1/2008 (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 1/1/2011.

- Beginning 10/1/2008, Maryland will prohibit the manufacture, processing, and distribution of products containing more than 0.1% of the PBDE formulations of penta- and octaBDE (NCEL, 2007).

- As of August 2004, New York prohibited the manufacture and processing, but not the sale or use, of penta- 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 penta- and octaBDE effective 2/6/2003, with Directive 2003/11/EC of the European Parliament and Counsel (European Parliament, 2003). At the time, the EU exempted decaBDE from the ban. However, on 4/1/2008, 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 7/1/2008. The ban applies to all EU member countries. Australia banned the use and import of pentaBDE effective 3/6/2007 (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 information technology (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 penta- and octaBDE, and the current uses of decaBDE.

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Prior to the end of production in 2004, approximately 95% of pentaBDE was used as an additive flame retardant in FPUF materials (EU, 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 metric tons (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 (EPs) used as protective coatings on 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 for certain plastics. 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; EU, 2003; IPCS, 1994). OctaBDE was typically added to ABS at a loading of 12−15% weight of the final product (EU, 2003). Materials that were treated with octaBDE included the housings of office equipment and business machines, and PC casings (EU, 2003).

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 (UPEs) are common plastics treated with decaBDE (Alaee et al., 2003; BSEF, 2006). A major use of decaBDE in the United States is as an additive flame retardant to HIPS. HIPS-based plastics are used in the manufacture of housings and back panels to televisions, in casings of audio and video equipment, mobile phones, remote controls, PCs, and PC monitors. The PE-based plastics are used in the insulation of wire and cables of electrical equipment. The PP-based plastics are used in communication cables, capacitor films, building

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cables, pipes, stadium seats, lamp sockets and holders, and kitchen hoods. The PBT-based 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 the back coating of upholstery textiles used in sofas, chairs, and office furniture. The decaBDE-treated nylon is used for 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 for the flame retardation of the back panels and the HIPS-based plastic casings of television sets. The Lowell Center for Sustainable Production at the University of Massachusetts estimated that approximately 17,150 MT of decaBDE was used in 28 million television sets sold in the United States in 2003 (Pure Strategies, Inc., 2005).

2.3.1. PBDE Content of Consumer Products

An assessment of the penta- and octaBDE content of various polymers was conducted by the German Environmental Protection Agency (Kemmlein et al., 2003). A total of 32 samples, 8 each of ABS copolymer ABS resin, HIPS, polyurethane hard foam (PUR-H), and epoxy resin were evaluated using 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; HIPS samples with 2.96% octaBDE; and PUR-H and EP with 2% pentaBDE. Table 2-2 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 2005 provides a quantitative basis for assigning a plausible PBDE content of final products made from flame 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 TVs, video camcorders, radios, high fidelity (Hi-Fi) stereo systems, portable compact disc 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 flame retardants in EEE (Morf et al., 2005).

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Table 2-2. Mean concentration (mg/g) 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

ABS a acrylonitrile-butadiene-styrene-copolymer; HIPS = high-impact polystyrene; PUR-H = polyurethane hard foam; EP = epoxy resin.

Source: Kemmlein 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 2-3.

Table 2-3. Mean concentrations of PBDEs (mg/kg) found in EEE 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 (±9)
TV housings (wood)	10 (±4)	10 (±4)	20 (±30)
TV/PC housings (plastic)	50 (±3)	7,500 (±600)	4,800 (±400)
TV housing rear covers	50 (±20)	7,700 (±3,600)	13,000 (±9,000)

Source: Morf et al. (2005).

In addition to the mean concentrations in EEE waste, Morf et al. (2005) determined the abundance of select PBDE congeners in the EEE materials. One sample of each of the following

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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 high resolution gas chromatography (HRGC)/high resolution mass spectrometry (HRMS). Table 2-4 summarizes the results of the analysis of these single EEE waste material samples. Because these were essentially grab samples, the results in Table 2-4 only serve as a general indication of the potential congener distribution in the EEE waste.

Table 2-4. BDE congener concentrations (mg/kg) present in EEE waste components sampled at a recycling facility in 2002

BDE congener	PC screen housings (mg/kg)	Television housings rear covers (mg/kg)	Fine particulates (dust) (mg/kg)	Fine-grained plastics (5−10 mm) (mg/kg)	Printed circuit boards (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	3,800	3,900	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). The concentrations of technical BDE mixtures were calculated by Morf et al. (2005) as follows: pentaBDE: Σ (BDE 47 + BDE 99 + BDE 100) × 1.26 octaBDE: BDE 183 × 2.87 decaBDE = BDE 209.

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The Consumer Products Safety Commission reported on the chemical flame retardant content of polyurethane foams (PUFs) used in furniture in the mid-1990s (Cobb, 2005). 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). Typically, PUF was made flame retardant with pentaBDE up until its discontinued use in 2004 (EU, 2002). An average of 3% (range 2.9–3.2%) PBDE was found in this particular PUF. The other samples contained melamine and/or TDCP as flame 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. GENERALIZED LIFE CYCLE OF PBDEs

This section presents a general analysis of the life cycle of PBDE as it relates to production, use, recycling, and disposal in the United States. In general, the life cycle of PBDE, from production to eventual disposal, includes activities that can cause 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 flame retardants, and to the final disposal and recycling of PBDE-treated consumer goods as products reach obsolescence. Figure 2-2 portrays the idealized life cycle of PBDE. Each element of the life cycle can potentially release PBDE to the open and circulating environment. If all data were available to address each facet of the life cycle, then the life cycle would be considered complete. However, with regard to all opportunities for environmental releases, the scarcity of information restricts the life cycle analysis presented in this chapter, limiting what can be said about potential releases during production, the consumer use of PBDE-treated products, and the disposal and recycling of PBDE-treated products. In addition, releases of PBDE from sewage treatment plants (STPs) in terms of wastewater discharges to surface waters and the land application of sewage sludge are addressed in the life cycle analysis.

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 as a flame retardant in the production of other materials for 2007 (U.S. EPA, 2009a; the latest reporting year). A total of 21.76 MT of decaBDE were released to the air, land, and water in 2007. Great Lakes Chemical (El Dorado, Arkansas), the

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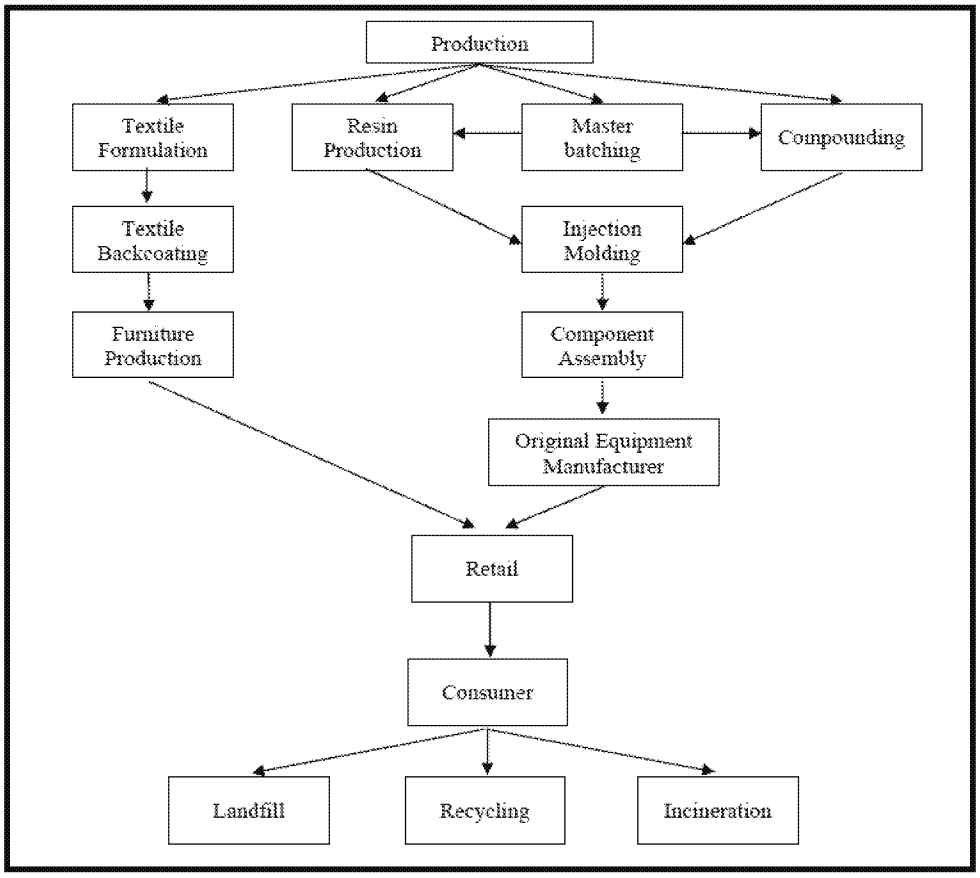

Figure 2-2. Idealized life cycle of polybrominated diphenyl ethers if all information is available to complete the analysis.

major U.S. producer of decaBDE, reported a total of 16.90 MT released to the environment. Figure 2-3 displays a summary of fugitive, stack, surface water, and land application and treatment releases for 2007 (top chart on figure). DecaBDE production and manufacturing waste was also transferred to chemical waste landfills, bringing the total TRI reported data to 32.2 MT in 2007. However, the disposal of the decaBDE 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 the Resource Conservation and Recovery Act (RCRA)-permitted landfills are designed to prevent off-site migration and contamination of groundwater sources. Releases of decaBDE to the air from fugitive and stack emissions accounted for the preponderance of environmental release, ~91% of total releases, from the chemical manufacture of decaBDE and its use in the production of other materials. The

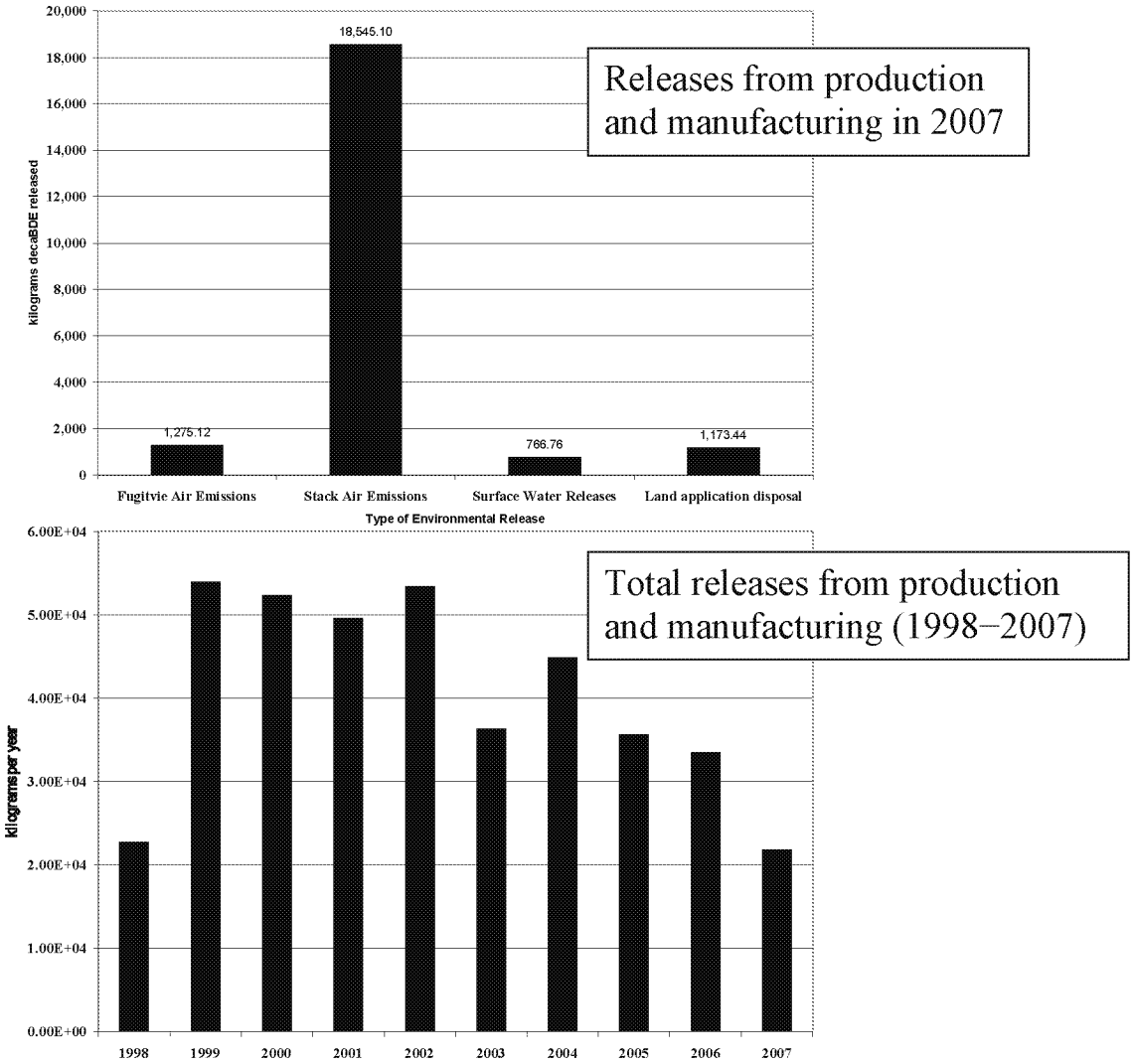

Figure 2-3. TRI data showing environmental releases (kg) of decaBDE from production and manufacturing facilities in 2007, and total environmental releases[a] from 1998 to 2007 (U.S. EPA, 2009a).

Source: U.S. EPA (2009a)

[a]Total environmental releases (kg/year) = fugitive air releases + stack releases + surface water releases + land application disposal and treatment releases.

discharges to surface water and the off-site land treatment of the waste accounted for 3.5% and 5.5%, respectively. The bottom chart in Figure 2-3 displays total environmental releases of decaBDE from production facilities from 1998 to 2007. From the chart, it appears that total environmental releases peaked in 1999 (at 53.9 MT) and remained essentially the same through

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2002. There was a drop in environmental releases in 2003 (to 36.3 MT), followed by an increase to 44.8 MT in 2004. The decaBDE releases in 2007 have decreased to 1998 levels (22.7 MT in 1998 and 21.7 MT in 2007). It is unknown 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 U.S. production in 2007. Table 2-5 displays the assumed BDE congener distribution in the environmental releases associated with production of decaBDE in 2007. TRI data do not represent all releases of decaBDE from all sources and use activities, although substantial releases associated with industrial use are captured by the program. Therefore, the emissions of decaBDE to air, water, and land are likely underestimated.

Table 2-5. Estimated environmental releases (kg) of BDE congeners from U.S. decaBDE production and manufacturing facilities in 2007[a]

Congener	Stack emissions	Fugitive emissions	Land application	Surface water
BDE 206	407.99	28.05	25.82	16.87
BDE 207	44.51	3.06	2.82	1.84
BDE 208	11.13	0.77	0.70	0.46
BDE 209	18,081.48	1,243.24	1,144.11	747.59
Total decaBDE	18,545.10	1,275.12	1,173.44	766.76

[a]Calculations based on BDE congener distribution in decaBDE from Table 2-1. Releases of decaBDE are from TRI data as reported in U.S. EPA (2009a).

2.4.2. Estimates of PBDE Releases to Indoor Air Based on Chamber Testing

PBDEs are basically flame 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 one of the mechanisms of release from the product into the surrounding indoor air.

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Two studies were found that attempted to measure the 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, IT devices, upholstered furniture, upholstery polyurethane foams, mattresses, and circuit boards. All materials had been treated with PBDE as additive flame 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 PBDEs 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. Workstations A and B were tested in the air chamber for 93 and 150 days, respectively. In PC Workstation A, BDE 47, BDE 100, BDE 99, and BDE 85 were detected in the air at concentrations of less than 0.3 ng/m^3. BDE 47, BDE 100, and BDE 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-hour): BDE 17 = 0.6; BDE 28 = 1.9; BDE 47 = 14.2; 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. BDE 28 (maximum concentration 0.5 ng/m^3), BDE 47

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(maximum concentration 8 ng/m^3), BDE 66 (maximum concentration 0.24 ng/m^3), BDE 100 (maximum concentration 0.27 ng/m^3), and BDE 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 BDE 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 BDE 47 and BDE 99 was 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).

A second laboratory study evaluated the potential for volatilization of PBDEs from TVs in Japan (Hirai et al., 2006). Three waste TV sets, obtained from a recycling facility, were opened, and the components were cleaned of dust and rinsed with hexane. In the experimental design, two TVs were turned on, and the third was left off. Separate experiments were conducted for each TV, with TV #1 and TV #2 kept on for a duration of 48 and 144 hours, respectively, and TV #3 kept in the off position. The TVs were placed in a 2.0 m (w) × 1.1 m (h) × 0.91 m (d) test chamber. A 1 m^3/hour air flow was introduced into the chamber, and the chamber was kept at a constant 28°C during the test period. Air samples were taken at the outlet of the chamber during the duration of the experiments. After the testing, samples were taken of the TV casing of TV #3. The air samples were Soxhlet extracted and then analyzed by high resolution gas chromatography coupled with high resolution mass spectrometry. Hirai et al. (2006) detected total PBDEs were as follows: 9,000 ng (TV #1 kept on for 48 hours); 3,900 ng (TV #2 kept on for 144 hours), and 610 ng (TV #3—not turned on at all). Approximately 76,000 mg/kg total PBDEs were detected in the TV casing. Emission factors were calculated for TVs #1 and #2 by dividing the total emission by PBDE content in TV casings and the length of test periods. From this study, emission factors (weight-to-weight emissions per year) of 4.8×10^{-6} and 7.1×10^{-7} were estimated for TVs #1 and #2, respectively. It should be noted that almost all of the PBDEs detected were nona- and decaBDE.

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 were used. There were three

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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, and (3) Only the PC built in 2003 was used by 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 was in rooms in office buildings equipped with numerous desktop PCs (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).

PUF from upholstered furniture treated with decaBDE showed no emission of PBDE congeners after 168-day residence time in a test chamber (Kemmlein et al., 2003). The flexible polyurethane 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.

2.4.3. Estimates of PBDE Releases to Air from Plastic Consumer Products

Attempts have been made to estimate the annual amount of PBDE that may be volatilized to the air during the general service life of a treated product. This is complicated by the fact that PBDEs are additive flame retardants rather than bonded with the plastic matrix. Two separate methods have been proposed for calculating the amounts of PBDEs volatilized from the product: an approach that utilizes the vapor pressures of the PBDEs, and an approach that calculates volatilization rates from the octanol-to-air partitioning coefficient. The vapor pressure approach was used by the EU to calculate the theoretical amount of deca-, octa-, and pentaBDE formulation that could have volatilized into the atmosphere over the entire European continent during the usage and product life of flame retardant plastic products (EU, 2001, 2002, 2003). Equation 2-1 was used by the EU and gives an approximate estimate of the percentage of PBDE that may volatilize over the product life:

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$$Percentage\ loss\ due\ to\ volatilization = \left(1.1 \times 10^{6}\right) \times V_p \times N\left(\%\right) \qquad (2\text{-}1)$$

where

V_p = vapor pressure of the PBDE flame retardant, mmHg at 21°C

N = service life of the flame retarded product in years (assumed to be 10 years by the EU).

Equation 2-1 was initially derived to approximate the volatilization rate of chemical plasticizers added to plastic films during their product use (EU, 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/year) from the treated products.

Assuming a vapor pressure of 3.47×10^{-8} mmHg (at 21°C) and a product life of 10 years, a volatilization loss rate of decaBDE from the product was calculated by the EU (using eq 2-1) to be 0.38% over 10 years (or 0.038% per year). Approximately 6,710 MT/year 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; EU, 2002). Using a vapor pressure of 4.9×10^{-8} mmHg for octaBDE, the EU estimated a volatilization rate of 0.54% over 10 years (or 0.054% per year) (EU, 2002). Assuming a 1994 consumption figure of 2,550 MT/year, the EU estimated that approximately 1.38 MT/year of octaBDE volatilized from product usage throughout Europe (EU, 2002). PentaBDE was assumed to have a vapor pressure of 3.5×10^{-7} mmHg, and this yielded an estimated annual loss rate of 3.9% over a 10-year product life (or 0.39% per year) (EU, 2001). The EU used a value of 1,100 MT/year pentaBDE in polyurethane foam products to calculate an annual release rate of 4.3 MT/year pentaBDE for Europe as a whole (EU, 2001). It should be noted that the product service life of 10 years was a subjective assumption by the EU, but, 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).

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Prevedouoro et al. (2004) estimated the volatilization flux of BDE 47 from products treated with pentaBDE and consumed in the United Kingdom. Using eq 2-1 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 to be 450–750 kg in 2000. By assuming a consumption rate for each year, Prevedouoro et al. (2004) then estimated the annual mass flux from 1975–2000. 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.

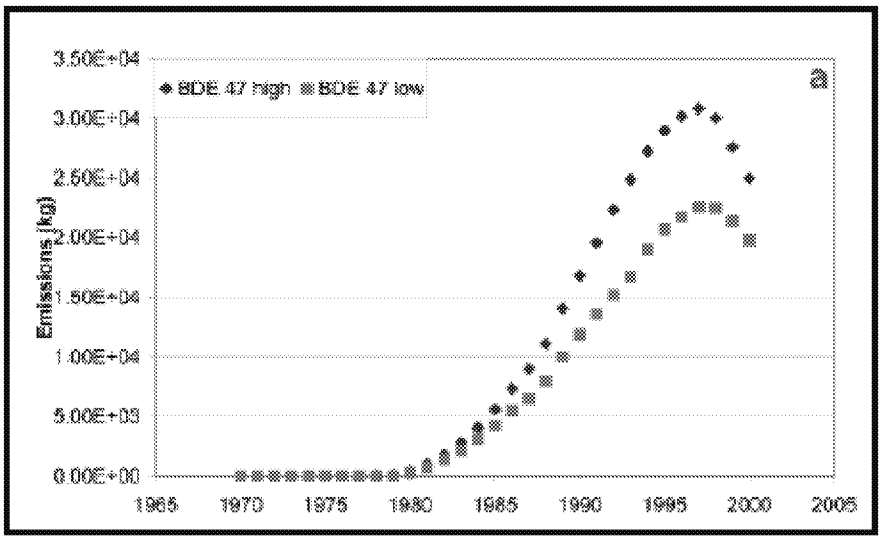

Figure 2-4. Volatilization of BDE 47 from PBDE-treated consumer products in the U.K.

Source: Prevedouoro et al. (2004). Assumes an average product life of 10 years. High and low estimates correspond to range of predicted consumption rates for the year 2000 (74 and 65 MT, respectively).

A second approach, termed the K_{oa} approach, was used by Prevedouoro et al. (2004) to estimate the amount of PBDE that may volatilize from the plastic matrix. The justification for use of this approach was that the logarithm (base 10) of the octanol air partition coefficient (log K_{oa}) of the PBDE may best describe the volatilization behavior (see Chapter 3, Section 3.2.5). Knowing the log K_{oa} of the BDE congener, Prevedouoro et al. (2004) used an equation (see eq 2-2) developed by Breivik et al. (2002) to estimate air release emission factors for PBDE

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releases from treated plastics; Breivik et al. (2002) derived eq 2 from regressing results from chamber experiments involving the volatilization of polychlorinated biphenyls (PCBs) from commercial sealants containing PCBs.

$$\log EF = -0.839 \times \log K_{OA}\,(20°C) + 4.83 \qquad (2\text{-}2)$$

where

log EF = emission factor, mass PBDE emitted/mass PBDE used per year (unitless)

Assuming a log K_{oa} of 10.82 (at 20°C) for BDE 47, Prevedouoro et al. (2004) calculated an emission factor of 5.64×10^{-5}. Multiplying this emission factor by the consumption rate of BDE 47 in 2000 (65 MT) gave an estimate of 3.5×10^{-3} MT (or 3.5 kg) BDE 47 emitted to the air from plastic-treated materials. The log K_{oa} approach produced an emission estimate that was 145 times lower than the vapor pressure approach (see eq 2-2) (Prevedouoro et al., 2004). Similar to eq 2-1, this K_{oa} approach must be considered uncertain and preliminary. PBDEs are added to the plastic (additive flame retardants) and not covalently bonded with the plastic, whereas the tested sealant material used in the development of eq 2-2 likely contained PCBs that were bonded to the sealant material.

These methods will be used to estimate the possible total volatilization of PBDE from treated plastic products in the United States. The following assumptions are used in the calculation:

1. The market demand of the penta-, octa-, and decaBDE formulations for 2001 (the latest year for which statistics are available) is used as a surrogate for inferring the amount used as flame retardant for plastic products in the United States.
2. The product life is 10 years.
3. Two separate methods are used to calculate possible amounts of decaBDE volatilized from plastic: eq 2-1; the vapor pressure (V_p) method advocated by the EU, and eq 2-2 based on the octanol air partition coefficient (log K_{oa}).

Estimates of releases to air using the EU method. In 2001, the market demand for decaBDE was approximately 24,500 MT in the Americas (Hites, 2004). The Americas includes all countries in North, Central, and South America. Roughly 80% of decaBDE is used for flame retardant hard plastics (Pure Strategies, Inc., 2005), with the remaining 20% used in textiles.

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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. 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. As discussed above, eq 2-1 produces an estimated volatilization loss rate of 0.038%/year—or 0.38% over 10 years. Applying these loss rates would result in approximately 5.99 MT decaBDE/year volatilized from these new products made from the flame retardant plastics. Assuming no additional products were put into the market during that period, this means that approximately 59.9 MT of decaBDE would have volatilized from plastic products over a 10-year time frame in the United States.

The market demand for octaBDE was 1,500 MT in the Americas (Hites, 2004), or 1,200 MT in the United States (the United States represents 80% of the total demand in North, South, and Central America). Using a vapor pressure of 4.9×10^{-8} mmHg for octaBDE (EU, 2002) results in an annual estimated volatilization rate of octaBDE of 0.54% over 10 years (or 0.054% per year). Approximately 0.648 MT/year octaBDE may volatize from plastic products (0.054% × 1,200 MT).

Total market demand for pentaBDE in the Americas was estimated at 7,100 MT (Hites, 2004), of which 95% (6,745 MT) was used as an additive flame retardant in FPUF materials (EU, 2001). Assuming that market demand in the United States accounted for 80% of total market demand in the Americas gives an estimate of 5,396 MT pentaBDE used in FPUF in the United States. Using eq 2-1 and a vapor pressure of 3.5×10^{-7} mmHg (EU, 2001) gives an estimated volatilization rate of 3.9% over a 10-year product life (or 0.39% per year). Multiplying the annual loss rate (0.39%/year) by the annual pentaBDE used in the United States (5,396 MT) produces an estimated annual volatilization of pentaBDE from FPUF of 21 MT/year.

Table 2-6 summarizes these calculations using the vapor pressure approach (see eq 2-1). Estimates of volatilization flux of the individual BDE congeners contained within the commercial formulations were made based on their respective vapor pressures. This analysis represents a picture of possible annual emissions from the volatilization of PBDE commercial formulations and BDE congeners 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 (EU, 2003). In

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Table 2-6. Calculated PBDE emission factors and estimated PBDE emissions from plastic products in the United States for 2001, derived using two approaches: vapor pressure approach; log K_{oa} approach

Chemical	2001[a,b] market demand (kg)	BDE[c] content (kg)	Vapor Pressure (V_p) Approach (see eq 2-1)			Log K_{oa} Approach (see eq 2-2)				Total BDE Congener Emissions Summed Across Formulations[j]		
			V_p[d] at 20°C (mmHg)	V_p-based emission factor (%/yr)e	V_p-based emissions (kg/yr)[f]	Log[g] K_{oa} at 20°C	K_{oa}-based Emission factor (kg/kg)[h]	Log K_{oa}-based emissions (kg/yr)[i]	Ratio (V_p/ K_{oa})	BDE congener	Total V_p method (kg)	Total K_{oa} method (kg)
DecaBDE	1.57×10^7		3.47×10^{-8}	0.038	5.99×10^3	13.21	5.58×10^{-7}	8.75×10^0	684	BDE 28	8.08×10^2	7.81×10^0
BDE 206	2.20%	3.45×10^5	NA	NA	NA	NA	NA	NA	NA	BDE 47	6.14×10^3	1.33×10^2
BDE 207	0.24%	3.76×10^4	NA	NA	NA	NA	NA	NA	NA	BDE 66	NA	3.05×10^0
BDE 208	0.06%	9.41×10^3	NA	NA	NA	NA	NA	NA	NA	BDE 85	9.53×10^0	1.20×10^0
BDE 209	97.50%	1.53×10^7	3.47×10^{-8}	0.038	5.84×10^3	13.21	5.58×10^{-7}	8.53×10^0	684	BDE 99	6.00×10^2	2.41×10^1
OctaBDE	1.20×10^6		4.90×10^{-8}	0.054	6.47×10^2	12.78	1.28×10^{-6}	1.54×10^0	421	BDE 100	1.47×10^2	7.10×10^0
BDE 153	5%	6.00×10^4	7.46×10^{-9}	0.008	4.92×10^0	11.82	8.19×10^{-6}	4.91×10^{-1}	10	BDE 138	NA	8.37×10^{-1}
BDE 154	1%	1.20×10^4	1.98×10^{-9}	0.002	2.61×10^{-1}	11.92	6.75×10^{-6}	8.10×10^{-2}	3	BDE 153	8.24×10^1	2.86×10^0
BDE 183	40%	4.80×10^5	NA	NA	NA	11.96	6.25×10^{-6}	3.00×10^0	NA	BDE 154	2.61×10^0	8.09×10^{-8}
BDE 190	1%	1.20×10^4	4.32×10^{-11}	0.00005	5.70×10^{-3}	NA	NA	NA	NA	BDE 183	NA	3.00×10^0
BDE 196	8%	9.60×10^4	NA	NA	NA	NA	NA	NA	NA	BDE 190	5.70×10^{-3}	NA
BDE 197	21%	2.52×10^5	NA	NA	NA	NA	NA	NA	NA	BDE 196	NA	NA
BDE 203	5%	6.00×10^4	NA	NA	NA	NA	NA	NA	NA	BDE 197	NA	NA
BDE 207	7%	8.40×10^4	NA	NA	NA	NA	NA	NA	NA	BDE 203	NA	NA
BDE 208	10%	1.20×10^5	NA	NA	NA	NA	NA	NA	NA	BDE 207	NA	NA

Table 2-6. Calculated PBDE emission factors and estimated PBDE emissions from plastic products in the United States for 2001, derived using two approaches: vapor pressure approach; log Koa approach (continued)

Chemical	2001[a,b] market demand (kg)	BDE[c] content (kg)	Vapor Pressure (V_p) Approach (see eq 2-1)			Log K_{oa} Approach (see eq 2-2)				Total BDE Congener Emissions Summed Across Formulations[j]		
			V_p[d] at 20°C (mmHg)	V_p-based emission factor (%/yr)e	V_p-based emissions (kg/yr)[f]	Log[g] K_{oa} at 20°C	K_{oa}-based emission factor (kg/kg)[h]	Log K_{oa}-based emissions (kg/yr)[i]	Ratio (V_p/ K_{oa})	BDE congener	Total V_p method (kg)	Total K_{oa} method (kg)
PentaBDE	5.40×10^6		3.50×10^{-7}	0.385	2.08×10^4	11.31	2.19×10^{-5}	1.18×10^2	176	BDE 208	NA	NA
BDE 28	0.2%	1.08×10^4	6.80×10^{-6}	7.483	8.08×10^2	9.50	7.24×10^{-4}	7.81×10^0	103	BDE 209	5.84×10^3	8.53×10^0
BDE 47	25%	1.35×10^6	4.14×10^{-7}	0.455	6.14×10^3	10.53	9.89×10^{-5}	1.33×10^2	46			
BDE 66	1%	5.40×10^4	NA	NA	NA	10.82	5.65×10^{-5}	3.05×10^0	NA			
BDE 85	2%	1.08×10^5	8.03×10^{-9}	0.009	9.53×10^0	11.66	1.11×10^{-5}	1.20×10^0	8			
BDE 99	35%	1.89×10^6	2.89×10^{-8}	0.032	6.00×10^2	11.59	1.28×10^{-5}	2.41×10^1	25			
BDE 100	6%	3.24×10^5	4.13×10^{-8}	0.045	1.47×10^2	11.31	2.19×10^{-5}	7.10×10^0	21			
BDE 138	0.5%	2.70×10^4	NA	NA	NA	11.13	3.10×10^{-5}	8.37×10^{-1}	NA			
BDE 153	3%	1.62×10^5	4.35×10^{-8}	0.048	7.75×10^1	11.82	8.19×10^{-6}	1.32×10^0	58			
BDE 154	2%	1.08×10^5	1.98×10^{-9}	0.002	2.35×10^0	11.92	6.75×10^{-6}	7.28×10^{-1}	3			

[a]Market demand (kg) for the United States assuming 80% of total market demand for the Americas.
[b]Percent (%) is the percent distribution of individual BDE congeners in the PBDE commercial formulation and is from Table 2-1.
[c]Calculated kg of BDE congener in the U.S. market demand for each PBDE commercial formulation.
[d]Taken from Table 3-4 in Chapter 3: Environmental Fate of Polybrominated Diphenyl Ethers. V_p in Pascals (Pa) converted to mmHg using 1 Pa = 0.0075 mm Hg.
[e]Emission factor is percent released to air per year from total kg used in treatment of plastic products and is calculated using eq 2-1.
[f]Derived by multiplying the V_p-based emission factor by the market demand (kg) for the PBDE commercial formulation (2nd column) or by individual BDE congeners contained in commercial formulation (3rd column) (kg).
[g]Taken from Table 3-5 in Chapter 3: Environmental Fate of Polybrominated Diphenyl Ethers.
[h]Emission factor is unitless based on weight/weight (kg released to air from total kg used in treatment of plastic products) and is calculated using eq 2-2.

Table 2-6. Calculated PBDE emission factors and estimated PBDE emissions from plastic products in the United States for 2001, derived using two approaches: vapor pressure approach; log Koa approach (continued)

[i]Derived by multiplying the K_{oa}-based emission factor by the market demand (kg) for the PBDE commercial formulation (2nd column) or by individual BDE congeners contained in commercial formulation (3rd column) (kg).
[j]Totals (kg) are based on summing the amounts calculated for each congener contained in each PBDE commercial formulation, i.e., penta-, octa-, and decaBDE.

NA = Data not available.

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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 eq 2-1 (EU, 2001, 2002, 2003). In order to compensate for this potential underestimation due to considering only 1 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 penta- 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 estimated 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.

Estimates of releases to air using the log K_{oa} method. From Table 2-1, it is evident that decaBDE is 97.5% (by weight) BDE 209. Therefore, approximately 1.53×10^7 kg of BDE 209 was used to treat products in the United States in 2001. The log K_{oa} for BDE 209 has been calculated by Webster et al. (2006) as 13.21 (at 20°C). Then using eq 2-2,

$$\log EF = -0.839 \times 13.21 + 4.83$$

$$\log EF = -6.25319$$

$$EF = 5.58E-07$$

Multiplication of this emission factor (5.58×10^{-7}) by 1.53×10^7 kg BDE 209 contained in decaBDE yields at estimated volatilization of 8.53 kg per year from plastic-treated products in the United States. Equation 2-1 predicted a volatilization rate of 5.99 MT/year (5.99×10^3 kg). This same procedure was repeated for a number of BDE congeners present in the PBDE formulations, and the results are shown in Table 2-6.

Table 2-6 compares the BDE emissions derived from both the EU and the log K_{oa} approaches (shown as the ratio of V_p/K_{oa}). In every case, the EU approach gave a result that was considerably greater than that produced by the K_{oa} approach. In general, BDE emissions

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calculated with the V_p approach are greater than the ones derived using the K_{oa} approach, by upwards of 4 orders of magnitude. For example, the EU approach (see eq 2-1) produced a volatilized amount of BDE 209 that is 684 times greater than with the K_{oa} approach of eq 2-2. Prevedouoro et al. (2004) thought that the K_{oa} approach gave a result that was more consistent with atmospheric measurements of PBDEs. Harrad et al. (2003) maintains that a ratio of BDE 47 to BDE 99 in air of about 3–3.5 is indicative of volatilization losses from products. The K_{oa} approach produced a ratio of 5.5, whereas the EU (V_p) approach resulted in a BDE 47:BDE 99 ratio of about 10. Hirai et al. (2006) derived decaBDE emission factors from measured emissions of brominated flame retardants from television sets. The televisions were placed in a chamber, and the air sampled for 48 or 144 hours or a wipe sample was collected. Hirai et al. (2006) calculated emission factors for BDE 209 of 4.8×10^{-6}/tv/year (based on 48-hour operation) or 7.1×10^{-7}/tv/year (144 hour operation). The calculated BDE 209 emission factor from plastics derived using the K_{oa} approach in Table 2-6 is 5.58×10^{-7}, and this falls within the range of emission factors developed by Hirai et al. (2006) in a chamber study. In consideration of these comparisons, the estimates derived from the K_{oa} approach (see eq 2-2) are relied on in the life cycle analysis in this report to give plausible estimates of the amounts of BDE congeners volatilized from plastic products. Table 2-6 shows that the volatilization rates from plastic products (with the K_{oa} approach) are 8.75 kg/year, 1.54 kg/year, and 118.30 kg/year for deca-, octa-, and pentaBDE, respectively. From the perspective of volatilization from products, pentaBDE accounts for 92% of emissions of total PBDE commercial formulations, followed by deca- (7%) and octaBDE (1%).

It should be noted that Hale et al. (2002) proposed the theory that the bulk degradation and crumbling of pentaBDE-treated polyurethane foam due to the aging of materials may give rise to indoor air concentrations of BDE 47, BDE 100, BDE 99, BDE 153, and BDE 154. While this is a possible mechanism for transferring BDE congener concentrations to indoor dust, the authors did not present a quantitative method for estimating the amounts of BDE congeners that may result in indoor air from the bulk degradation of PUF. An algorithm for estimating the release of PBDEs from degradation and crumbling of foam could not otherwise be found in the literature, and for that reason, estimates are not provided in this report for this important release mechanism.

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

Once PBDE-treated electronic and electrical equipment (EEE) 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 composed of products that have reached their end-life. The final results of the analysis described here are shown in Table 2-7.

Table 2-7. Estimated BDE congener emissions (kg/year) to air in the United States from incineration of EEE waste and estimated BDE congeners in EEE waste that is landfilled and recycled

		Incineration air emissions (kg/yr)	Landfilled (kg/yr)	Recycled (kg/yr)
DecaBDE	Distribution	46	857,000	153,000
BDE 206	0.022	1.012	18,854	3,366.0
BDE 207	0.0024	0.1104	2,057	367.2
BDE 208	0.0006	0.0276	514	91.8
BDE 209	0.975	44.85	835,575	149,175
OctaBDE	Distribution	48	890,000	159,000
BDE 153	0.05	2.4	44,500	7,950
BDE 154	0.01	0.48	8,900	1,590
BDE 183	0.4	19.2	356,000	63,600
BDE 190	0.01	0.48	8,900	1,590
BDE 196	0.08	3.84	71,200	12,720
BDE 197	0.21	10.08	186,900	33,390
BDE 203	0.05	2.4	44,500	7,950
BDE 207	0.07	3.36	62,300	11,130
BDE 208	0.1	4.8	89,000	15,900

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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 (MSW) in the United States (U.S. EPA, 2006) In the United States, an estimated 2.4 million metric tons (MMT) of EEE waste were generated in 2005 (U.S. EPA, 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, video cassette recorder (VCRs), digital video disc (DVD) players, video cameras, stereo systems, telephones, cell phones, hand-held electronic devices, personal computers, laptop computers, printers, and fax and copy machines (U.S. EPA, 2006). Most, but not all, of these products contain PBDE as a flame 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 deca- 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., TV sets, videos, camcorders, radios, Hi-Fi stereo systems, and portable compact disk (CD players), and small size E&E [*sic*] equipment (e.g., plugs and mobile phones)." They justified this selection of waste as the EEE that contained the bulk of brominated flame retardant use, 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 Tables 2-3 and 2-4 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 averages of these total product concentrations are given as 510 and 530 mg/kg for deca- and octaBDE, respectively. For purposes of calculating deca- and octaBDE content in EEE waste in the United States, the total product concentrations of deca- 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 U.S. EPA, 2006). Although imperfect, 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 (U.S. EPA, 2006) to permit a rough estimate of the amounts of deca- and octaBDE that may in

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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 × 2.4 MMT 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 (3×10^5 MT recycled EEE waste × 510 and 530 mg/kg for deca- and octaBDE, respectively). The amount of BDE congeners in the EEE waste in the United States each year is estimated assuming the low end of the range of distributions of BDE congeners present in the PBDE commercial formulation (see Table 2-1). The amounts of BDE congeners that may be present in the deca- and octaBDE-treated EEE waste that is recycled in the United States in 2005 are shown in Table 2-7.

Not all of the EEE waste is recycled in the United States. The remaining 2.1 MMT of EEE material in MSW is disposed of either 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 (U.S. EPA, 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 deca- 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.

BDE congeners sent to landfills: The amount of BDE congeners sent to landfills in the United States each year is estimated assuming the low end of the range of distributions of BDE congeners present in the PBDE commercial formulation (see Table 2-1). The amounts of BDE congeners may be present in the deca- and octaBDE-treated EEE waste that is landfilled in the United States in 2005 are shown in Table 2-7.

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 to support any estimation of this as an environmental release. For example, Osako et al. (2004) evaluated the untreated and treated leachate at seven landfills in

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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), and 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. (2009) 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 was as follows: Landfill 1 (ND: 2,670), Landfill 2 (ND: 6,638), Landfill 3 (ND: 7,230), Landfill 4 (ND: 41–4,009), and Landfill 5 (ND: 90–9,793). Individual congeners across all five landfills ranged in the following concentrations (pg/L): BDE 28 (ND: 100–3,333), BDE 47 (ND: 1,469–9,793), BDE 66 (ND: 4,020), BDE 71 (ND: 1,667–9,459), BDE 75 (ND: 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), and BDE 183 (ND: 263).

Kim et al. (2006) investigated the leaching potential of PBDEs from plastics under simulated landfill conditions. In the lab tests, four samples representing high-impact TV housing plastics were tested for leaching potential. The plastic samples were as follows: Sample 1 consisted of HIPS containing no decaBDE; Sample 2 was HIPS TV housing with decaBDE; Sample 3, RC-TV, was a mixture of roughly-cut actual TV housings; and Sample 4, WC-TV, was well-crushed TV housings (containing decaBDE). The leaching potential was determined using distilled water, 20% methanol solution, and dissolved humic solution of 1,000 mg/L (based on organic carbon) as leachants. The leaching test conditions were a liquid-to-solid ratio of 100:1, and a contact period of 5 days, with twice-daily agitation in a temperature-controlled room of 30°C without pH or ionic strength control. Concentrations in simulated leachate samples were obtained using HRGC coupled with HRMS, with the preparation of samples described by Kim et al. (2006). Results showed that PBDE concentrations were highest with the well-crushed HIPS TV housing, with total PBDE leachate concentrations of 220, 23,000, and 1,200 ng/L, using the leachants of distilled water, methanol, and dissolved humic acid, respectively. In all conditions, the nonaBDE homologue dominated all other homologue groups detected in the leachate samples. DecaBDE was detected in all leachate samples from the laboratory tests in the

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following ranges and test conditions: distilled water: 0.21–77 ng/L; 20% methanol: 8.9–7,600 ng/L; dissolved humic solution: 10–210 ng/L.

Odusanya et al. (2009), Osako et al. (2004), and Kim et al. (2006) give an indication of the ranges in concentration of BDE congeners that may be present in untreated landfill leachate. However, these studies suggest that PBDE congeners are not expected to be detected in treated landfill leachate. In the United States, by federal regulation, MSW landfills are required to collect and treat landfill leachate, and to continuously monitor groundwater for an indication of leachate migration from the landfill (Code of Federal Regulations, 2008). BDE concentrations in treated landfill leachate are likely to be small. Due to the lack of data, no attempt was made here to estimate the amount of PBDE potentially released into the U.S. 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 are 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, respectively, 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, respectively, subject to stack emissions. Through the use of strict regulations, the United States has 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, respectively, 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]). Table 2-7 displays the amounts of BDE congeners that may be emitted each year from incineration of EEE waste in the United States (assuming that the BDE

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congener distribution in Table 2-1 is not altered either by the heats of combustion or by the air pollution control device).

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 intramolecular 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.

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

There are approximately 16,519 STPs operating within the United States (U.S. EPA, 2008). These STPs have a combined daily sewage flow rate of approximately 1.3×10^{11} L/day (NAS, 2002). Approximately 93% of total daily sewage flow undergoes secondary and/or tertiary treatment before the wastewater is discharged into surface water (U.S. EPA, 2008). Applying the factor of 93% to the total daily sewage flow from all STPs combined gives an estimated flow rate of 1.2×10^{11} L/day 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 6.3×10^{6} MT dwt (metric tons, dry weight) of sewage sludge are used or disposed of annually in the United States, of which 3.8×10^{6} MT dwt (60%) is land applied or commercially distributed as fertilizer (NAS, 2002; U.S. EPA,

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1999). The remaining sludge is disposed of in the following manner: 1.1×10^6 MT dwt are landfilled (17%), 1.4×10^6 MT dwt are incinerated (22%), and 6.3×10^4 MT dwt (1%) have miscellaneous uses such as daily landfill cover (NAS, 2002; U.S. EPA, 1999).

EPA recently conducted an updated national sewage sludge survey (NSSS) of PBDEs and other contaminants present in sewage sludges in the United States (U.S. EPA, 2009b). This information was used to estimate the amount of PBDE contamination in the sewage sludge that is applied to land as a soil amendment and fertilizer. The NSSS was a statistically based survey in which sewage sludges were randomly selected from 74 STPs in 35 states to represent the United States as a whole. Samples were collected between August 2006 and March 2007. The mean concentrations (µg/kg dwt) of PBDE congeners in sewage sludge were BDE 47 = 709.17, BDE 99 = 716.36, BDE 153 = 68.33, and BDE 209 = 2,181.23. On a national basis, the dominant congener was BDE 209.

A study by North (2004) was chosen to represent the PBDE content of STP effluent discharges to surface waters in the United States. This is because it was a careful mass balance study of the distribution of PBDEs in sewage sludge and the effluent at a large tertiary STP in Palo Alto, California. 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. In the wastewater effluent discharged into surface water after secondary and tertiary treatment, the mean concentrations 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% of the total concentration); however, BDE 47 and BDE 99 accounted for about 36% and 39% of the total concentration 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

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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, with a mean concentration of 82 μg/m^3. North (2004) calculated the mass loading of total BDD/BDF on the basis of assuming half the detection limit for the nondetected homologues (hepta–octaBDD and BDF). This estimate was 2.8×10^{-7} kg/year based on total BDD/BDF in the stack emissions (assuming 1/2 detection limit for nondetects).

The following mass loading calculations of BDE congeners from STPs to the U.S. 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 United States (mean concentrations from USEPA 2009b):**

General equation:

$$\text{Mass loading PBDE to land } (MT/yr) = \text{mean concentration PBDE in sewage sludge} \times \text{mass sludge applied to land}/yr \quad (2\text{-}3)$$

(1) $$BDE\ 47\ loading = 709.17 \frac{ug\ BDE47}{kg\ sludge} \times \frac{1\times10^{-9}\ kg}{ug} \times \frac{3.8\times10^{+6}\ MT\ sludge}{yr}$$
$$BDE\ 47\ loading = 2.69\ MT/yr\ to\ land$$

(2) $$BDE\ 99\ loading = 716.36 \frac{ug\ BDE\ 99}{kg\ sludge} \times \frac{1\times10^{-9}\ kg}{ug} \times \frac{3.8\times10^{+6}\ MT\ sludge}{yr}$$
$$BDE\ 99\ loading = 2.72\ MT/yr\ to\ land$$

(3) $$BDE\ 153\ loading = 68.33 \frac{ug\ BDE\ 99}{kg\ sludge} \times \frac{1\times10^{-9}\ kg}{ug} \times \frac{3.8\times10^{+6}\ MT\ sludge}{yr}$$
$$BDE\ 153\ loading = 0.26\ MT/yr\ to\ land$$

(4) $$BDE\ 154\ loading = not\ tested\ in\ USEPA(2009)$$

(5) $$BDE\ 209\ loading = 2{,}181.23 \frac{ug\ BDE\ 209}{kg\ sludge} \times \frac{1\times10^{-9}\ kg}{ug} \times \frac{3.8\times10^{+6}\ MT\ sludge}{yr}$$
$$BDE\ 209\ loading = 8.29\ MT/yr\ to\ land$$

(6) $$\Sigma BDE\ loading = BDE\ 47 + BDE\ 99 + BDE\ 153 + BDE\ 209$$
$$\Sigma BDE\ loading = 13.96\ MT/yr\ to\ land$$

- **Estimated annual loading of BDE congeners to surface waters from STP effluent in the United States (Mean concentrations from North, 2004):**

General equation:

$$\text{Mass loading PBDE to water} = \text{mean concentration PBDE in STP effluent} \times \text{total STP effluent/yr} \quad (2\text{-}4)$$

(1)

$$BDE\ 47\ loading = 10.5\ \frac{ng\ BDE47}{L\ effluent} \times \frac{1\times10^{-9}\ g}{ng} \times \frac{kg}{1\times10^{+3}\ g} \times \frac{1.3\times10^{+11}\ L\ total\ effluent}{d} \times \frac{365\ d}{yr}$$

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

(2)

$$BDE\ 99\ loading = 11.2\ \frac{ng\ BDE\ 99}{L\ effluent} \times \frac{1\times10^{-9}\ g}{ng} \times \frac{kg}{1\times10^{+3}\ g} \times \frac{1.3\times10^{+11}\ L\ total\ effluent}{d} \times \frac{365\ d}{yr}$$

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

(3)

$$BDE\ 153\ loading = 0.98\ \frac{ng\ BDE153}{L\ effluent} \times \frac{1\times10^{-9}\ g}{ng} \times \frac{kg}{1\times10^{+3}\ g} \times \frac{1.3\times10^{+11}\ L\ total\ effluent}{d} \times \frac{365\ d}{yr}$$

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

(4)

$$BDE\ 154\ loading = 0.78\ \frac{ng\ BDE154}{L\ effluent} \times \frac{1\times10^{-9}\ g}{ng} \times \frac{kg}{1\times10^{+3}\ g} \times \frac{1.3\times10^{+11}\ L\ total\ effluent}{d} \times \frac{365\ d}{yr}$$

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

(5)

$$BDE\ 209\ loading = 1.73\ \frac{ng\ BDE209}{L\ effluent} \times \frac{1\times10^{-9}\ g}{ng} \times \frac{kg}{1\times10^{+3}\ g} \times \frac{1.3\times10^{+11}\ L\ total\ effluent}{d} \times \frac{365\ d}{yr}$$

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

(6)

$$\sum BDE_{n=28}\ loading = 29.02\ \frac{ng\ \sum BDE}{L\ effluent} \times \frac{1\times10^{-9}\ g}{ng} \times \frac{kg}{1\times10^{+3}\ g} \times \frac{1.3\times10^{+11}\ L\ total\ effluent}{d} \times \frac{365\ d}{yr}$$

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

2.5. SUMMARY OF THE GENERALIZED LIFE CYCLE ANALYSIS OF PBDEs IN THE UNITED STATES

This section presents a summary of a simplified and generalized life cycle analysis of PBDEs used in plastics, and EEE in the United States. This life cycle analysis is focused on the

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production, use, and disposal of plastics and EEE treated with deca- and octaBDE. Included in the life cycle is an analysis of the environmental loadings of PBDE congeners that may be occurring from sewage treatment plant operations in the United States. It is noted that all quantities generated in this example are highly uncertain. For this analysis, data and procedures described and summarized in previous sections are used. To the extent practical, this analysis is congener-specific. Figure 2-5 presents a summary of a generalized annual life cycle of PBDE in the United States to include production, use, disposal, recycling, and sewage treatment. All estimates of PBDE content of materials, EEE waste, and congener-specific emissions and releases are discussed and documented in Section 2.4 (above).

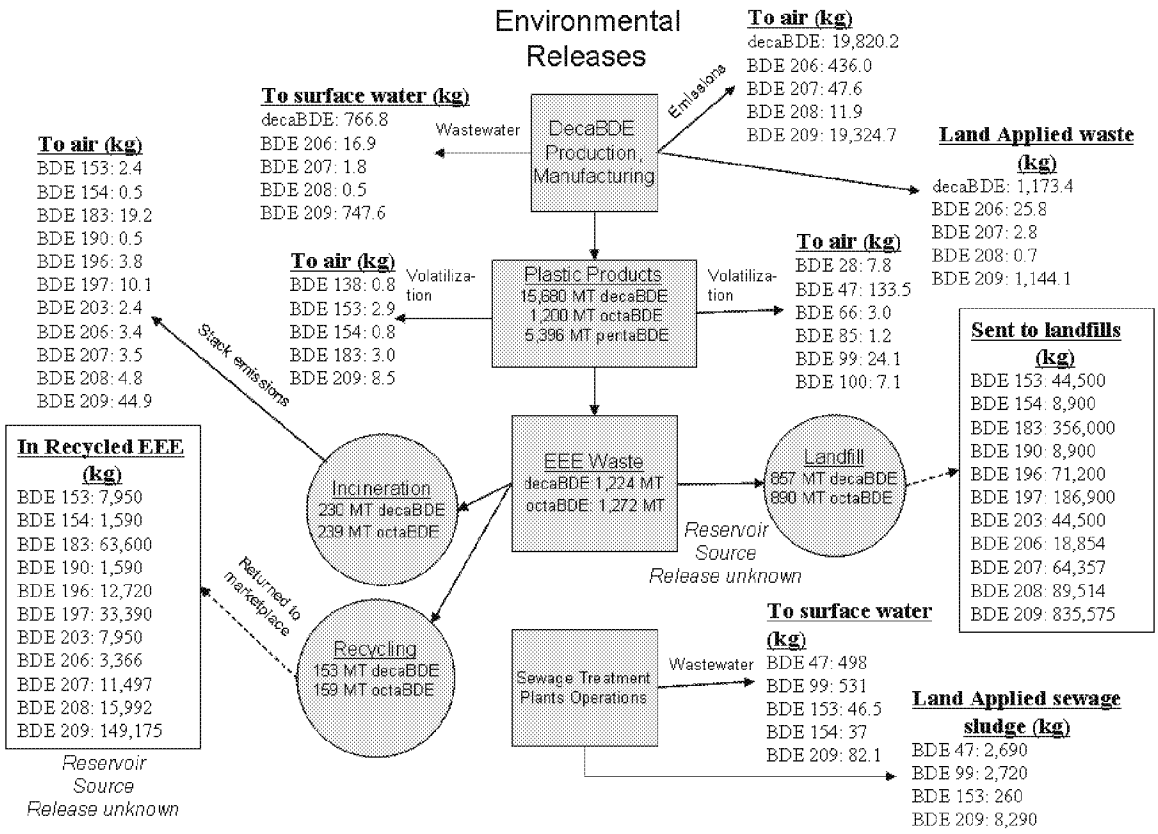

Figure 2-5. Summary of a generalized annual life cycle of PBDE in the United States: production, use, disposal, recycling, and sewage treatment.

The following observations are made (from Figure 2-5) within the context and limitations of this life cycle analysis:

1. BDE 209 is the dominant congener in every aspect of the life cycle analysis, with the exception of wastewater discharges from sewage treatment plant operations, in which case, BDE 99 is the most abundant congener;

2. Major reservoirs for BDE 209 include municipal solid waste landfills (835,575 kg/year landfilled) and recycled EEE waste (149,175 kg/year). The annual amount of BDE 209 that may be released into the environment from these reservoirs is unknown and is a major area of uncertainty;

3. The production and manufacturing of decaBDE causes the highest amounts of BDE 209 releases to air (19,325 kg/year) when compared to other sources analyzed, followed by the incineration of EEE waste (45 kg/year), and volatilization from plastic products (8.5 kg/year);

4. Approximately 9,434 kg/year of BDE 209 are applied to land through the application of industrial (1,144 kg/year) and sewage (8,290 kg/year) sludges;

5. Production/manufacturing facilities and sewage treatment plants discharge approximately 748 and 82 kg/year BDE 209, respectively, to surface water;

6. With regard to BDE 47, information was sufficient to estimate air releases from products (134 kg/year), wastewater discharges to surface water from sewage treatment plants (498 kg/year), and the land application of sewage sludge (2,690 kg/year);

7. Estimation of the BDE annual volatilization rates from plastic products gave the following rank order by amounts volatilized (high to low): BDE 47 > BDE 99 > BDE 209 > BDE 28 > BDE 100 > BDE 66/183 > BDE 153 > BDE 85 > BDE 138/154;

8. With regard to land-applied sewage sludge, the rank order of BDE congeners by kg/year (high to low) is: BDE 209 > BDE 99 > BDE 47 > BDE 153;

9. With regard to wastewater discharges from sewage treatment plant operations, the rank order of BDE congeners by kg/year (high to low) is BDE 99 > BDE 47 > BDE 209 > BDE 153 > BDE 154.

Not all potential sources of PBDE releases to the environment were addressed in the generalized life cycle analysis. For example, Choi et al. (2008) found PBDE emissions emanating from a steel mill in South Korea using an array of air samplers near the complex. An average concentration of 25.2 pg of total PBDEs/m^3 of air was detected (range: 9.0–61.6 pg/m^3) near the facility, and air dispersion modeling related these results to mill operations. Although indicative of steel mills as a possible source of PBDE air emissions, this study did not give a sufficient basis for estimating emissions from U.S. steel mills. Rather than directly measuring

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emission rates of PBDEs from the mill, the study relied on the indirect method of environmental monitoring and inferring impacts from these results with air dispersion analysis. Cetin and Odabasi (2008) also observed higher air concentrations of PBDE compounds in ambient air samples taken in urban areas of Turkey where steel mill operations were located. However, this study also did not directly measure PBDE emissions from a steel plant, and only provided indirect evidence that steel mills do emit PBDEs to the atmosphere.

Other studies have implicated dust in the cabin spaces of automobiles as another source of PBDE releases to the environment. Lagalante et al. (2009) sampled dust from the interiors of 60 automobiles that were available for resale at U.S. dealerships. Lagalante et al. (2009) detected BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209 in the dust. BDE 209 was the dominant congener detected, with a mean concentration of 48.1 μg/g. This study was in agreement with a previous study of dust obtained from 20 used automobiles in the United Kingdom (Harrad et al., 2008). However, Harrad et al. (2008) found a higher mean concentration of BDE 209 of 410 μg/g. Although these studies have implications for human exposures to PBDEs for time spent within the automobile, this information was judged to be insufficient in estimating mass flows of PBDEs to the environment and incorporation into the life cycle analysis.

Finally, the potential for PBDEs to enter the indoor (or car) environment through the flaking and crumbling of treated foam products cannot be characterized quantitatively, or even qualitatively. It is theorized to be a major source to the indoor environment, but procedures to estimate this kind of release from foams is not available.

Major uncertainties are associated with this generalized life cycle analysis. These include the following:

- No accurate or current production figures are readily available on the amount of PBDE produced annually in the United States. Hence, this analysis presumed that the amounts corresponding to market demand are equal to the amounts produced. Moreover, it was assumed that 80% of the total market demand of North, Central, and South America (combined) is the market demand of the United States. Although this is a reasonable assumption when U.S. consumption patterns are considered, there is no published information to validate this assumption.

- The environmental releases associated with production and manufacturing were derived from TRI data reported for the year 2007. These data are self-reported by industry and

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are often based on engineering calculations rather than on actual measurements. The accuracy of such numbers is largely unknown.

- The estimates of air emissions of PBDEs due to volatilization from plastic products are very uncertain. Rates of volatilization were estimated by using an equation (see eq 2-2) that relies on the use of the chemical's octanol air partition coefficient to approximate the volatilization process. Although Prevedouoro et al. (2004) have determined that estimates from this method appear to be reasonable, additional research on volatilization losses is necessary to reduce uncertainty.

- Due to limitations in existing information, this analysis relied on the use of information collected over different years, i.e., not all information corresponded to the same year. For example, market demand figures were from 2001 (the latest available information); estimates of environmental releases from production and manufacturing facilities were for 2007; estimates of EEE waste generation amounts and amounts recycled, landfilled, and incinerated were for 2005, and releases from sewage treatment plant operations were from 2007. Combining these years into a single annual estimate, although uncertain, likely means that the life cycle is fairly representative of annual releases between 2001 and 2007.

- Although production of octa- and pentaBDE ceased by the end of 2004, estimates of PBDE volatilization from plastic materials assumed the presence of deca-, octa-, and pentaBDE in the products. The analysis presumed that plastic products were made flame retardant in 2001 (corresponding to the market demand of PBDEs), and this predated the time when octa- and pentaBDE were no longer used. Because the analysis assumed a functional life of 10 years for the plastic products, it is logical to presume that the products would contain octa- and pentaBDE.

- The analysis assumed that once EEE materials were placed into landfills, there would be no further environmental releases. There is the potential that PBDEs may be released when incorporated into landfill leachate and the leachate leaves the landfill site to contaminate groundwater or surface water. However, strict Federal regulations are intended to restrict both the formation of landfill leachate and migration of leachate from the site. No information is currently available that directly demonstrates that PBDE contaminated leachate has migrated from landfills in the United States and subsequently contaminated surface waters or groundwater. No information is readily available on the amounts of leachate currently generated by U.S. landfills.

2.6. SUMMARY

This chapter has presented information on the production, uses, and life cycle of PBDE commercial formulations and their respective BDE congeners. 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,

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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 2003 (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 4/1/2008. 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 flame retardant to rigid plastics used in casings to TVs, PCs, liquid crystal display (LCD) screens, and other 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 manage 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.

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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 polybrominated diphenyl ether (PBDEs). This chapter includes 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 brominated diphenyl ether (BDE) congeners to bioaccumulate and biomagnify in 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 (V_p).

3.2. PHYSICAL/CHEMICAL PROPERTIES

The following section 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 pentaBDE commercial formulations as flame 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 aqueous solubility is to add an excess amount of a 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

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(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 some specific PBDE congeners. PBDEs have low water solubility. Organic contaminants with low solubility do not readily dissolve in surface water (relatively hydrophobic) and 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 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 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

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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[a]
DecaBDE (Commercial)	<0.001	EU (2002)	Low
OctaBDE (Commercial)	0.005 0.002	EU (2003) ATSDR (2004)	Low
PentaBDE (Commercial)	0.013	EU (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

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Table 3-1. Estimated water solubility values for PBDEs (continued)

Brominated diphenyl ether	Aqueous solubility (mg/L @ 25° C)	Reference	Solubility score[a]
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
BDE 209[b]	<0.001	EU (2002)	Low

[a]Solubility score is as follows: low = relatively insoluble; medium = somewhat soluble; high = very soluble.
[b]No estimates or measurements of BDE 209 could be found in the literature. Because the decaBDE commercial formulation is 97% BDE-209, the value for decaBDE is assumed to represent BDE 209.

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

Polybrominated diphenyl ether	Log K_{ow} coefficient	Reference
DecaBDE (Commercial)	6.27 6.27	ATSDR (2004) EU (2003)
OctaBDE (Commercial)	6.29 6.29	ATSDR (2004) EU (2003)
PentaBDE (Commercial)	6.64−6.97 6.57	ATSDR, (2004) EU (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 6.77	Palma et al. (2002) ATSDR (2004) Kuramochi et al. (2004) Tittlemier et al. (2002) Hardy (2004)
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)

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Table 3-2. Estimated octanol water partition coefficients (log Kow) values for PBDEs (continued)

Polybrominated diphenyl ether	Log K_{ow} coefficient	Reference
BDE 99	6.53−7.66 7.32 7.21 7.13 7.66	Palma et al. (2002) ATSDR (2004) Kuramochi et al. (2004) Tittlemier et al. (2002) Hardy (2004)
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)
BDE 209[a]	6.27 6.27	ATSDR (2004) EU (2003)

[a]No estimates or measurements of BDE 209 could be found in the literature. Because the decaBDE commercial formulation is 97% BDE-209, the value for decaBDE is assumed to represent BDE 209.

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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 vapor 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 summarizes estimated H for PBDEs.

3.2.4. Vapor Pressure (V_p)

Volatilization of a chemical and its presence in air is driven by the V_p of the chemical. V_p also controls the phase distribution of a chemical in air (e.g., the proportion that exists in the vapor and particle phases). The V_p 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. V_p is usually expressed in units of Pascals (Pa) or millimeter of Hg (mmHg). In general, volatile organic compounds have a solid phase vapor pressure ≥10 Pa at an atmospheric temperature of 25°C (Olsen and Nielsen, 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 V_ps of PBDE compounds best represents their tendency to partition between the vapor and particle 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 eq 3-1 (Paasivirta et al., 1999).

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

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

Brominated diphenyl ether	(H) (atm-m^3/mol @ 25°C)	Reference
DecaBDE (Commercial)	1.20×10^{-8} 3.95×10^{-7}	ATSDR (2004) Cetin et al. (2005)
OctaBDE (Commercial)	7.5×10^{-8}–2.6×10^{-7}	ATSDR (2004)
PentaBDE (Commercial)	3.5×10^{-6}–1.2×10^{-5}	ATSDR (2004)
BDE 15	2.07×10^{-4} 2.10×10^{-4}	ATSDR (2004) Tittlemier et al. (2002)
BDE 28	5.03×10^{-5} 7.80×10^{-5} 5.10×10^{-5}	ATSDR (2004) Cetin et al. (2005) Tittlemier et al. (2002)
BDE 47	1.48×10^{-5} 8.39×10^{-6}	ATSDR (2004) Cetin et al. (2005)
BDE 66	4.93×10^{-6}	ATSDR (2004)
BDE 77	1.18×10^{-5}	ATSDR (2004)
BDE 85	1.09×10^{-6}	ATSDR (2004)
BDE 99	2.27×10^{-6} 5.92×10^{-6}	ATSDR (2004) Cetin et al. (2005)
BDE 100	6.81×10^{-7} 2.37×10^{-6}	ATSDR (2004) Cetin et al. (2005)
BDE 153	6.61×10^{-7} 4.34×10^{-6}	ATSDR (2004) Cetin et al. (2005)
BDE 154	2.37×10^{-6} 7.90×10^{-7}	ATSDR (2004) Cetin et al. (2005)
BDE 183	7.3×10^{-8}	ATSDR (2004)
BDE 209	3.95×10^{-7}	Cetin et al. (2005)

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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 V_p, log K_{oa} is temperature dependent. For every 10°C decrease in atmospheric temperature, there is a corresponding two- to threefold 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 of the log K_{oa} is that *n*-octanol is a good surrogate for absorption of all organic carbon. The greater the Log K_{oa} (e.g., ≈10) is, the stronger 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.

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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[a]	P_L	P_L Reference
DecaBDE (Commercial)	573.15	EU (2002)	9.28×10^{-9}	4.63×10^{-6}	EU (2002)
OctaBDE (Commercial)	473.15	EU (2003)	1.26×10^{-7}	6.59×10^{-6}	EU (2003)
PentaBDE (Commercial)	475.15	EU (2002)	8.60×10^{-7}	4.69×10^{-5}	EU (2001)
BDE 1	NA			0.163	Wong et al. (2001)
BDE 2	NA			0.128	Wong et al. (2001)
BDE 3	NA			2.59×10^{-1}	Tittlemier et al. (2002)
BDE 7	NA			1.68×10^{-2}	Wong et al. (2001)
BDE 8	NA			1.37×10^{-02}	Wong et al. (2001)
BDE 10	NA			2.77×10^{-2}	Wong et al. (2001)
BDE 12	NA			1.19×10^{-2}	Wong et al. (2001)
BDE 15	329.15	Marsh et al. (1999)	8.59×10^{-3} 4.88×10^{-3}	1.73×10^{-2} 9.84×10^{-3} 2.00×10^{-2}	Tittlemier et al. (2002) Wong et al. (2001) Wania et al. (2002)
BDE 28	337.15	Marsh et al. (1999)	9.07×10^{-4} 6.51×10^{-4}	2.19×10^{-3} 1.57×10^{-3}	Tittlemier et al. (2002) Wania et al. (2002)
BDE 30	358.15	Marsh et al. (1999)	1.18×10^{-3}	4.56×10^{-3}	Wong et al. (2001)
BDE 32	350.40	Palma et al. (2002)	6.91×10^{-4}	2.25×10^{-3}	Wong et al. (2001)

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

Brominated diphenyl ether	T_m (K)	T_m Reference	P_S[a]	P_L	P_L Reference
BDE 33	NA		1.49×10^{-3}	1.49×10^{-3}	Wong et al. (2001)
BDE 35	413.01	Palma et al. (2002)	1.04×10^{-4}	1.39×10^{-3}	Wong et al. (2001)
BDE 37	321.65	Palma et al. (2002)	6.00×10^{-4}	1.02×10^{-3}	Wong et al. (2001)
BDE 47	353.65	Palma et al. (2002)	7.42×10^{-5} 5.52×10^{-5}	2.50×10^{-4} 1.86×10^{-4} 2.15×10^{-4}	Wong et al. (2001) Tittlemier et al. (2002) Wania et al. (2002)
BDE 66	NA			2.38×10^{-4} 1.22×10^{-4}	Wong et al. (2001) Tittlemier et al. (2002)
BDE 69	NA			4.00×10^{-4}	Wong et al. (2001)
BDE 75	408.15	Marsh et al. (1999)	4.10×10^{-5}	4.92×10^{-4}	Wong et al. (2001)
BDE 77	368.15	Marsh et al. (1999)	3.21×10^{-5} 1.40×10^{-5}	1.56×10^{-4} 6.79×10^{-5}	Wong et al. (2001) Tittlemier et al. (2002)
BDE 82	NA		4.80×10^{-5}	6.47×10^{-5}	Wong et al. (2001)
BDE 85	396.45	Palma et al. (2002)	1.07×10^{-6}	9.86×10^{-6}	Tittlemier et al. (2002)
BDE 99	365.45	Palma et al. (2002)	1.49×10^{-5} 3.85×10^{-6} 7.94×10^{-6}	6.82×10^{-5} 1.76×10^{-5} 3.63×10^{-5}	Wong et al. (2001) Tittlemier et al. (2002) Wania et al. (2002)

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

Brominated diphenyl ether	T_m (K)	T_m Reference	P_S[a]	P_L	P_L Reference
BDE 100	371.15	Marsh et al. (1999)	5.50×10^{-6} 7.07×10^{-6}	2.86×10^{-5} 3.68×10^{-5}	Tittlemier et al. (2002) Wania et al. (2002)
BDE 115	NA			3.02×10^{-5}	Wong et al. (2001)
BDE 138	NA			1.58×10^{-6}	Tittlemier et al. (2002)
BDE 153	456.15	Palma et al. (2002)	1.63×10^{-7} 5.80×10^{-6}	5.80×10^{-6} 2.09×10^{-6} 8.87×10^{-6}	Wong et al. (2001) Tittlemier et al. (2002) Wania et al. (2002)
BDE 154	416.15	Marsh et al. (1999)	2.64×10^{-7}	3.80×10^{-6}	Tittlemier et al. (2002)
BDE 183	NA			4.68×10^{-7}	Tittlemier et al. (2002)
BDE 190	470.4	Palma et al. (2002)	1.85×10^{-8} 5.76×10^{-9}	9.05×10^{-7} 2.82×10^{-7}	Wong et al. (2001) Tittlemier et al. (2002)
BDE 209[b]	573.15	EU (2002)	9.28×10^{-9}	4.63×10^{-6}	EU (2002)

[a]P_S was calculated from P_L using eq 3-1 and assuming the melting point temperature (T_m) in the table. NA = not available in the literature.
[b]No estimates or measurements of BDE 209 could be found in the literature. Because the decaBDE commercial formulation is 97% BDE 209, the value for decaBDE is assumed to represent BDE 209.

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)
BDE 209	13.21	Webster et al. (2006)

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3.2.6. Vapor Particle Partitioning in Air

Semivolatile organic compounds are present in the ambient air partition between the vapor phase and the particle phase. The physics of this behavior is controlled by the V_p 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 transport 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 phase of PBDEs from the air. The vapor 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 V_p using the Junge-Pankow model as indicated in eq 3-2 (Su et al., 2006).

$$\Phi = (c\Theta)/(P_L + (c\Theta)) \tag{3-2}$$

where

Φ = fraction of the compound adsorbed to aerosol particles

P_L = saturation subcooled liquid phase V_p, 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 and vapor phases of the PBDE commercial formulations and BDE congeners calculated from eq 3-2 and assuming the subcooled liquid V_p 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 1.5×10^{-6} cm^2/cm^3 typical of aerosols in background urban air (Whitby, 1978). The predicted particle phase of the PBDE increases with decreasing V_p and increasing number of bromine atoms on the molecule.

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

Brominated diphenyl ether congener	Brominated diphenyl homologue	Vapor phase, %	Particle phase, %
BDE 1	monoBDE	100	0
BDE 2	monoBDE	100	0
BDE 3	monoBDE	100	0
BDE 7	diBDE	100	0
BDE 8	diBDE	100	0
BDE 10	diBDE	100	0
BDE 12	diBDE	100	0
BDE 15	diBDE	100	0
BDE 28	triBDE	99	1
BDE 30	triBDE	99	1
BDE 32	triBDE	99	1
BDE 33	triBDE	98	2
BDE 35	triBDE	98	2
BDE 37	triBDE	98	2
BDE 47	tetraBDE	90	10
BDE 66	tetraBDE	87	13
BDE 69	tetraBDE	94	6
BDE 75	tetraBDE	95	5
BDE 77	tetraBDE	81	19
BDE 82	pentaBDE	71	29
BDE 85	pentaBDE	28	72
BDE 99	pentaBDE	61	39
BDE 100	pentaBDE	56	44
BDE 115	pentaBDE	54	46
BDE 138	hexaBDE	6	94
BDE 153	hexaBDE	7	93
BDE 154	hexaBDE	13	87
BDE 183	heptaBDE	2	98
BDE 190	heptaBDE	1	99
BDE 209	decaBDE	1	99

By these calculations, and at the air temperature of 25°C, greater than 98% to 100% of the mono-, di-, triBDE congeners may be present in the vapor phase in ambient air. As the degree of bromination increases, the BDE congeners increasingly partition to the particle phase.

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For example, the various tetra- and pentaBDE congeners begin to distribute more to atmospheric particles than the lesser brominated compounds. The hexa- and hepta-congeners may be present in the particle phase from 87 to 99% at an ambient air temperature of 25°C. Approximately 99% of BDE 209 is 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 50–70% of pentaBDE 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 vapor 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 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 fish tissue divided by the concentration in the water. The log BAF is

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expressed in units of liters per kilogram of tissue and can be normalized to apply to wet weight (wwt), dry weight (dwt), 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 have estimated BAFs for PBDE congeners. Table 3-7 summarizes BAF and BMF 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 criterion 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 pentaBDE congeners exceed these criteria, and, therefore, have a 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.

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

Brominated diphenyl ether	Species	Factor	Reference
Bioaccumulation factors (BAFs)			
BDE 28	Lake trout	7.6	Tomy et al. (2004)
BDE 47	Lake trout Blue mussels	7.3 6.1	Streets et al. (2006) 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 Blue mussels	6.7 6.1	Streets et al. (2006) Gustafsson et al. (1999)
BDE 10	Lake trout	7.5	Streets et al. (2006)
Biomagnification factors (BMFs)			
BDE 15	Porpoise	1.6–2.4	Ramu et al. (2006)
BDE 28	Lake trout Porpoise	7.6 1.6–2.4	Tomy et al. (2004) Ramu et al. (2006)
BDE 47	Lake trout Coho salmon Porpoise	2.1 3.2 1.5	Tomy et al. (2004) Stapleton and Baker (2003) Ramu et al. (2006)
BDE 66	Lake trout	7.8	Tomy et al. (2004)
BDE 99	Lake trout Porpoise	6.6 1.8–2.4	Tomy et al. (2004) Ramu et al. (2006)
BDE 100	Lake trout Porpoise	6.5 1.7–2.4	Tomy et al. (2004) Ramu et al. (2006)
BDE 138	Lake trout	3.2–8.7	Tomy et al. (2004)
BDE 153	Lake trout Coho salmon Porpoise	9.4 4.0 1.6–2.2	Tomy et al. (2004) Stapleton and Baker (2003) Ramu et al. (2006)
BDE 154	Lake trout Porpoise	13.3 1.4–2.2	Tomy et al. (2004) Ramu et al. (2006)
BDE 183	Lake trout Porpoise	3.9 0.8–2.2	Tomy et al. (2004) 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)

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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, chlorobenzenes, polychlorinated biphenyls (PCBs), and polychlorinated dibenzo-*p*-dioxins (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 decaBDE has induced reductive debromination of decaBDE in vitro to yield octaBDE and heptaBDE after a contact time of 2 months (He et al., 2006). The octa- and heptaBDE did not further debrominate in the presence of the microbe—even after 1 year. Thus, the microorganism was specific to the degradation of decaBDE and is incapable of debrominating lower-brominated PBDE compounds. The organism normally has an affinity for TCE, but when decaBDE was dissolved in TCE and exposed to *S. multivaornas*, reductive debromination did not occur. However, TCE was completely dechlorinated to *cis*-DCE.

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Dehalococcoides ethenogenes 195 bacterium was experimentally tried in an attempt to debrominate octaBDE (He et al., 2006). *D. ethenogenes* of the 195 strain contains reductive dehalogenase 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 octaBDE dissolved in TCE for 12 months. One hundred thirty nmoles octaBDE composed of BDE 153, BDE 183, BDE 196, BDE 203, BDE 207, and BDE 208 added to the culture yielded 11.5 nmoles of combined BDE 47, BDE 49, BDE 99, and BDE 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 pentaBDE congeners. The experiment showed that bromines in the 2 (*ortho*) and 4 (*para*) positions on the PBDE molecule (e.g., BDE 99 and BDE 47) were the most resistant to microbial reductive debromination.

In another study, Gerecke et al. (2005) debrominated BDE 209 in vitro to yield BDE 206, BDE 207, and BDE 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 1×10^{-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 octaBDE 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

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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 be concluded unambiguously that it was a product of microbial degradation of BDE 209.

Tokarz et al. (2008) observed reductive debromination of BDE 209 from laboratory experiments simulating anaerobic conditions of sediment. Precleaned pond sediments were mixed with BDE 209, BDE 99, and BDE 47 to yield a final concentration of 5.0 μg/g of the BDE congeners in sediment. The sediment was transferred to 125-mL serum bottles, and dextrose and methanol were added to promote the rapid onset of anaerobic and methanogenic conditions. The sediments were placed into contact with anaerobic microorganisms (species not specified) and allowed to incubate. Extracts were prepared and analyzed with gas chromatography coupled with an electron capture detector and gas chromatography coupled with a mass spectrometer. In a second experiment, termed a 'biomimetic' experiment, 0.03 millimoles BDE 209, BDE 99, or BDE 47 were mixed with 5.0 millimoles titanium citrate and 0.2 millimoles Vitamin B12 in 0.33-moles buffer solution. The final total volume was 31 mL. The authors noted that titanium citrate is an electron-donating reducing agent. Reductive debromination was enhanced in the biomimetic experiment; BDE 209 completely debrominated within 24 hours, forming lower-brominated congeners BDE 47, BDE 66, BDE 99, and BDE 119. In the anaerobic sediment microcosm experiments, BDE 209 more slowly debrominated through reductive debromination. The authors calculated that the degradation half-life of BDE 209 in the live microcosms ranged from 6 to 50 years, with an average of just over 14 years.

Rayne et al. (2003a) demonstrated the in vitro anaerobic microbial reductive debromination of BDE 15. A nondifferentiated 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 nonsubstituted 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

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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 liver, and by one of three possible pathways: the deiodinase thyroid hormone-regulating enzymes (Stapleton et al., 2006a; Tomy et al., 2004), endogenous microbial activity in the gut (Stapleton et al., 2004), or the enzymes of the microsomal mono-oxygenase system (Stapleton et al., 2004). It is not known whether all fish have the metabolic capacity to biotransform PBDEs, nor is it known whether the rate of debromination of 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, b; Tomy et al., 2004; Kierkegaard et al., 1999).

The possibility that metabolic debromination of decaBDE may be occurring in fish was initially observed in the late 1990s 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. 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 13 BDE

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congeners spiked onto fish food. The BDE congeners included BDE 28, BDE 47, BDE 66, BDE 77, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, BDE 190, and BDE 209. The duration of dietary exposure to the spiked food was for 56 days, followed by 112 days 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 debromination, 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 debromination 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 were 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% ±0.8% of the BDE 99 dietary concentrations. 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 BDE 99 and BDE 183 could be mediated by either intestinal microflora, and/or by endogenous enzyme systems such as the hepatic mono-oxygenase enzymes.

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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 wwt 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 wwt 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 2 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 spiked food mixture. High resolution GC/MS confirmed the identification of the following BDE congeners in the fish tissue: BDE 188, BDE 201, BDE 202, BDE 207, and BDE 208. While BDE 207 and BDE 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 BDE 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 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

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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. La Guardia et al. (2007) provide evidence that debromination in fish and crustaceans occurs in the natural environment outside of laboratory conditions. La Guardia et al. (2007) compared the PBDE congener profiles in the sediments near the outfall of a sewage treatment plant with the PBDE profiles found in the tissues of sunfish (*Lepomis gibbosus*), creek chub (*Semotilus atromaculatus*), and crayfish (*Cambarus puncticambarus*). Similar BDE congener profiles were observed in the sewage treatment plant sludge, the surficial sediments at the outfall of the wastewater discharge, and downstream from the sewage treatment plant. Tri through decaBDE congeners were detected in the samples, with BDE 209 dominating the sediments. The resident sunfish tissues contained BDE 209 and also congeners not present in the sewage sludge and sediments. These were two octa (BDE 201, BDE 202) and three hepta (BDE 188, BDE 184, and BDE 179) congeners. La Guardia et al. (2007) proposed that the presence of these congeners in the Sunfish was suggestive of in vivo synthesis from the metabolic debromination from BDE 209.

3.4.2.2. *Evidence for Debromination in Rats*

In vivo debromination of PBDEs has been observed in a rat feeding study. Huwe and Smith (2007) found evidence of the metabolic debromination of BDE 209 in male Sprague-Dawley rats fed a commercial formulation of decaBDE (98.5% BDE 209). Other congeners detected in the formulation included nonaBDEs, octaBDEs, and a trace of BDE 183. DecaBDE was mixed in corn oil to a concentration of 18.9 μg 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 μg/g-day 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 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

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BDE 209 to lower congeners was observed from an apparent 160% increase in the tissue concentration of BDE 197, BDE 201, and BDE 207 as compared to levels in the feed. Huwe and Smith (2007) postulated that the possible formation of BDE 197 and BDE 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 PBDEs in chickens (Pirard and Pauw, 2007) and starlings (Van den Steen et al., 2007). Pirard and Pauw (2007) fed seven Sexaline hens a diet containing 3.4 mg/kg pentaBDE 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., BDE 47, BDE 100, and BDE 99), were found in chicken excreta compared to hexa- and heptaBDEs. 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 could be debrominated to form BDE 99 and BDE 100.

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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 frame. 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 twofold 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 BDE 208, BDE 207, BDE 206, BDE 197, BDE 196, and BDE 183 in liver and muscle was evidence of metabolic debromination of decaBDE 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, BDE 99, BDE 207, and BDE 209 were most frequently detected in blood serum of 23 exposed cats and were related to their dietary intake. The cats consuming only canned wet cat food (mostly composed of fish) (n = 4) had relatively little BDE 207 or BDE 209 present in serum. BDE 47 and BDE 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

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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. *Photodegradation 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 ultraviolet (UV)-based processes; e.g., the absorption of photons present in wavelengths found in sunlight, i.e., UV radiation. Photodegradation of PBDE

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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 photodegradation 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 hours; BDE 47 = 2.53 hours; BDE 99 = 0.32 hours; BDE 100 = 6.46 hours; BDE 153 = 0.29 hours; and BDE 183 = 0.26 hours. 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 congeners BDE 1 and BDE 3
- BDE 47 (tetraBDE) photodegraded to form two tribrominated species (BDE 17 and BDE 28), which in turn, further debrominated to form dibrominated BDE congeners
- BDE 4, BDE 8, and BDE 15 debrominated to form monoBDE congeners BDE 1 and BDE 3
- BDE 99 (pentaBDE) photodegraded to form the tetraBDE congeners BDE 66, BDE 49, and BDE 47 (tetraBDE), which subsequently underwent further photolysis
- BDE 100 (pentaBDE) photodegraded to form BDE 75 and BDE 47 (both tetraBDEs)
- BDE 153 (hexaBDE) photodegraded to form BDE 99, BDE 101, and BDE 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 flame retardants (BFRs) decaBDE and

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decabromodiphenyl ethane (DBDPE) in treated plastics. Four different flame retarded plastic samples were evaluated, including pure high impact polystyrene (HIPS) not treated with BFR, HIPS treated with decaBDE (0.15% by weight, HIPS treated with DBDPE (about 0.10% by weight), 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 a liquid nitrogen chamber. The pulverized plastic samples were initially passed through a 300 μm sieve and then a 10 μm sieve. The plastic powder collected between two sieves (106–300 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 (HRGC) coupled with high resolution mass spectrometry (HRMS) in the selective ion monitoring mode. Photolysis of BDE 209 in the decaBDE-treated HIPS samples was observed after 1 week of exposure to sunlight. The BDE 209 concentration in the plastic decreased, and hepta-, octa-, and nonaBDE congeners 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- through 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 in Maryland. The first material was typical indoor house dust known as Standard Reference Material (SRM) 2585. During chemical

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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 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), which 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-hour cumulative exposure to sunlight was achieved. Samples were transferred to a laboratory where they were extracted and analyzed for PBDE using high resolution gas chromatography 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 outdoor temperature was 26.9°C and ranged from 18.7 to 32°C. Photodegradation of BDE 209 was observed in both SRM dust samples after 100-hour exposure to natural sunlight. The first-order BDE 209 photodegradation rates were calculated as 2.3×10^{-3} and 1.7×10^{-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-hour 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 was 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.

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Rayne et al. (2006) photodegraded BDE 153 in acetonitrile, distilled water, and seawater at ultraviolet radiation wavelengths (UV radiation) of 302 nm. BDE 153 dissolved in acetonitrile and irradiated for 5 minutes formed three primary photodegradation products: (1) pentaBDE isomers BDE 99, BDE 101, and BDE 118 (20% yield); (2) brominated dibenzofuran congener 1,2,4,7,8-PeBDF (30% yield); and (3) three nonspeciated tetrabrominated 2-hydroxybiphenyls (20% yield). Continued irradiation up to 1 hour caused the photodegradation of the pentaBDE congeners to form the tetraBDE isomers BDE 47, BDE 49, BDE 66, and BDE 77. The irradiation of BDE 153 in distilled and/or seawater produced the same tetraBDE 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. DecaBDE formulation (approximately 97% BDE 209) was dissolved in hexane to create three solutions of BDE 209, ranging in concentrations from 6.92×10^{-6} to 6.14×10^{-6} 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 7/2/2003 and 10/23/2003, 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) accumulated successively over the 60-minute exposure period. OctaBDEs were transformed to heptaBDEs that were 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 be 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 to 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 g 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 was 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 hours when exposed to artificial light, but degraded to about 21% and 57% over 96 hours 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-hour exposure. The half-life of BDE 209 varied by matrix and artificial or natural sunlight. The half-life of BDE 209 in toluene or silica exposed to artificial light was less than 0.25 hours. By comparison, BDE 209 in sand, sediment, and soil had estimated half-lives of 12 hours, 40–60 hours, and 150–200 hours, respectively, when exposed to artificial light. For exposure to natural sunlight, the half-lives of BDE 209 in sand and sediments were 37 hours and 80 hours, respectively. Söderström et al. (2004) reported

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that the photolytic 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 BDE 206, BDE 207, and BDE 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 pentaBDE mixture was composed of the BDE congeners BDE 47 (4.1 μg/mL), BDE 85 (0.1 μg/mL), BDE 99 (1.2 μg/mL), BDE 100 (4.1 μg/mL), BDE 153 (0.23 μg/mL), and BDE 154 (0.34 μg/mL). A 5 mL aliquot of the solution was adsorbed to 100 μm polydimethylsiloxane 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 had 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 BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 154. Based on sequential formation of these products, Sánchez-Prado et al. (2005) postulated that the principle photodegradation pathway was reductive debromination.

The Wilford et al. (2008) study provides indirect, but weak evidence that photodegradation of higher brominated BDE congeners may occur on atmospheric particles under natural sunlight conditions. Air particulates were sampled over a 1-month period (April 17−May 20) in 2004 at a semirural area of the United Kingdom. The area was a government meteorological workstation located in grasslands 4 km from the city of Lancaster. PBDEs in the particle phase were collected on glass fiber filters continuously, 24-hour/day. BDEs in the vapor phase were collected on polyurethane foam plugs (PUFs), but only over a 7-day period. The purpose of PUF sampling was to confirm the dominance of particle bound BDE 209 during the cool month of spring. Mean air sampling volumes were 380 and 360 m^3 for

particle phase only samples, and for particle and vapor phase samples, respectively. An additional 9-day quality control sample was taken to confirm that the intended internal and recovery standards (BDE 181 and BDE 190) were not present in the atmosphere. Samples were collected daily and stored in amber glass containers at −20°C until analysis. Samples were analyzed by gas chromatography coupled with an electron capture detector for the higher brominated compounds BDE 197, BDE 196, BDE 208, BDE 207, BDE 206, and BDE 209. BDE 32, BDE 17, BDE 28, BDE 35, BDE 37, BDE 75, BDE 49, BDE 71, BDE 47, BDE 66, BDE 77, BDE 100, BDE 119, BDE 99, BDE 85, BDE 154, BDE 153, BDE 138, BDE 166, BDE 181, BDE 183, and BDE 190 were analyzed using gas chromatography coupled with mass spectrometry in negative ion chemical ionization mode. From the particle phase of sampling, mean air concentration of BDE 209 was found in a range from ND to 100 pg m^{-3}. The mean of total PBDEs ranged from ND to 400 pg m^{-3}. The investigators had expected BDE 209 to dominate all higher brominated congeners present in the particle phase. However, in this study, the nonaBDE congeners ([BDE 207] > [BDE 208] ≥ [BDE 206]) were found to be at much higher concentrations than BDE 209. Wilford et al. (2008) suggested a possible explanation for nonaBDE congeners dominating over BDE 209. It is possible that debromination or degradation of BDE 209 (i.e., actual or relative enrichment of nonaBDEs) is occurring in the atmospheric environment (e.g., photodegradation).

Raff and Hites (2007) reported on the relative importance of photolysis in the removal of PBDEs from the atmosphere by comparing photolytic degradation with the atmospheric removal processes of wet and dry deposition. The platform for this comparison was the study of the temporal concentration profile of PBDEs in the sediments of Siskiwit Lake on an island in Lake Superior. Through the temporal study of Siskiwit Lake, the investigators concluded that the PBDEs detected in the lake sediments were entirely the result of atmospheric deposition. This conclusion was reinforced by the fact that there were no significant anthropogenic sources within Siskiwit Lake's drainage basin (Raff and Hites, 2007). The mean concentration of BDE 209 in the sediment was 2,600 pg/g. This amount accounted for approximately 95% of the total mass of PBDEs in the most recent lake sediment layer, which corresponds roughly to the time frame of 1995−2005. The remaining BDE congeners were BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154 at mean concentrations of 57, 69, 20, 9, and 9 pg/g, respectively. The investigators examined the importance of photolysis in explaining the BDE congener profile in the lake

sediments by comparing photolytic half-lives of BDE congeners to the first-order removal rate constants for oxidant reactions and dry and wet deposition. Photolytic degradation rates of 25 BDE congeners were estimated from in-house experiments in a conventional photo-reactor exposing PBDEs to UV radiation in wavelengths of 280–350 nm. Deposition fluxes of the BDE congeners were calculated from the sediment concentrations and the sedimentation rate of the lake. The phase distribution of BDE in ambient air, along with the measured size distribution characteristics of atmospheric particles over Lake Superior, were used in the computation of atmospheric deposition. Raff and Hites (2007) assumed a mean dry particle deposition velocity of 0.2 cm/s. Wet deposition was calculated from estimates of atmospheric washout ratios of BDE congeners and the frequency and intensity of annual rainfall events. Dry vapor deposition of BDE congeners was estimated from their respective Henry's law constants (refer to Raff and Hites [2007] for the calculation method). The atmospheric depletion rate constants of the vapor phase BDE congeners (from reaction with the atmospheric hydroxyl radical) were calculated from published experiments and application of structure-activity relation techniques, and assuming an average hydroxyl radical concentration of 9.7×10^5 molecules/cm^3. It was found that the dominance of BDE 209 in the lake sediments is explained by the atmospheric removal process of the wet deposition of the particle bound BDE 209. The first-order atmospheric removal rate constant (k) for the wet deposition of BDE 209 was calculated to be 13×10^{-6}/s, corresponding to an atmospheric lifetime of 0.89 days. By comparison, the atmospheric removal rate constants for dry deposition, photolysis, and depletion by the hydroxyl radical were 0.80×10^{-6}/s, 0.047×10^{-6}/s, and approximately 0, respectively, corresponding to atmospheric lifetimes of 14 days and >200 days for dry deposition and photolysis. Raff and Hites (2007) determined that photolysis is a minor atmospheric removal process for BDE 209, and almost all of the BDE 209 in the sediments of Siskiwit Lake is due to the atmospheric wet and dry deposition (dominated by wet deposition) of particle bound BDE 209. On the other hand, Raff and Hites (2007) determined that photolysis is the major atmospheric removal pathway for BDE 47 and BDE 99, having estimated atmospheric removal rate constants of 26×10^{-6}/s and 43×10^{-6}/s, corresponding to atmospheric lifetimes of 0.45 and 0.27 days, respectively.

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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 hydroxyl 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 hydroxyl radical could be located in the literature. However, atmospheric half-lives have been estimated from quantitative structure-activity relationships. Assuming an hydroxyl radical concentration of 5×10^5 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 1.27×10^{-12} cm^3/molecule/s using the same assumption of hydroxyl radical concentration (EU, 2001). The ATSDR concluded that atmospheric degradation from reaction with the hydroxyl 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 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 that 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 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 polybrominated dibenzo-*p*-dioxins (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

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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 is 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 decaBDEs 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). 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. PBDEs are discharged into surface waters from industrial activity and 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. Transport of BDEs occurs over relatively long distances, >1,000 km.

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Evidence for this comes from the presence of PBDEs in sediments and the tissues of deep ocean-dwelling whales and other marine mammals. In general, BDE congeners are relatively hydrophobic and lipophilic compounds that have low water solubility and low vapor pressures. BDE congeners bioaccumulate into terrestrial and aquatic food webs. The body burdens of BDE congeners in a wide variety of avian species, fish, insects, 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 sections discuss the fate of PBDEs in air, water, and soil.

3.6.1. Fate of PBDEs in Outdoor and Indoor Air

3.6.1.1. *Fate of PBDEs in outdoor 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 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 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 a 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 pentaBDE 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 pentaBDE; and 480−735 km for decaBDE (Wania and Dugani, 2003). In theory,

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- Vapor phase mono, di, and tri brominated compounds have the highest tendency for long-range transport in the atmosphere because they are unsusceptible to particle surface deposition.

- The tetra and penta brominated congeners more equally partition between vapor and particle 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, BDE 47, and BDE 99) in air over remote oceanic regions; BDE 209 has only been occasionally detected (Wurl et al., 2006a). It has been noted (see Section 3.4.3.1) 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, BDE 99, and BDE 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. 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 (Horstmann and McLachlan, 1998). These processes of vegetative atmospheric depletion of semivolatile compounds are also observed with coniferous forests, grasses, and green leafy crops (Horstmann 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

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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 air-water fugacity ratio ≈ 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, BDE 99, and BDE 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, BDE 47, and BDE 100 dominated total PBDEs in the open ocean with BDE 209 detected only at trace levels. In the freshwater of Lake Ontario, approximately 60% of the total PBDE was composed of BDE 47 and BDE 99, with congeners BDE 100, BDE 153, and BDE 154 each contributing approximately 5 to 8% of the total (Environment Canada, 2006). Section 4.2 provides further discussions on sediment concentrations of PBDEs.

3.6.1.2. *Fate of PBDEs in Indoor Air*

The previous discussion has focused on the fate of PBDEs in outdoor air. PBDEs are also detected in indoor air, especially sorbed to aerially suspended dust inside homes and office buildings. Human contact with PBDE contaminated dust in indoor microenvironments is the

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primary way in which humans become exposed (see Chapter 5). This brings into question the fate of PBDEs in indoor air microenvironments. As was discussed in Chapter 2, many products in the home and office have been made flame retardant with PBDEs, e.g., the back casings of television sets; the casings to personal computers (PC); facsimile machines, and printers, to name a few. Chapter 2 has shown that PBDEs may volatilize from these products and contaminate indoor air through product use. For example, laboratory experiments have observed volatilization of BDE 47, BDE 100, BDE 99, and BDE 85 from a computer workstation consisting of PC casings, mouse, keyboard, and printer (Kemmlein et al., 2003). Studies have also linked PBDE-treated TVs and furniture to indoor dust concentrations of PBDEs (Hirai et al., 2006; Allen et al., 2008). 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., 2008). Indoor air with the highest PBDE levels were in rooms in office buildings equipped with numerous desktop PCs and numerous office furniture items containing pentaBDE-treated polyurethane foam. The flexible polyurethane foam in furniture padding has been linked to indoor dust levels of pentaBDE congeners, and the TVs and PCs have been linked to decaBDE in dust (Hirai et al., 2006; Allen et al., 2008).

Volatilization of PBDEs and congeners from products into surrounding air does occur. Given the vapor pressures of PBDEs, much of the amount volatilized would be expected to partition between vapor and particle phases, with increasing sorption to dust particles in air with increased degree of bromination of the molecule. Partitioning is enhanced by the organic fraction of dust, and this creates a homogeneous distribution of PBDEs in indoor dust particles (Webster et al., 2009). A second possible mechanism of transferring PBDEs and BDE congeners from products to indoor dust is through the weathering and abrasion of PBDE-treated materials (Webster et al., 2009). The abrasion causes small polymers particles that become incorporated as house dust. However, evolution of small abrased particles containing PBDEs from this mechanism is inconsistent and likely results in a heterogeneous distribution (Webster et al., 2009).

Dust from indoor spaces is often collected by vacuuming. Such dust would inherently contain some amount of PBDE contamination. Once the vacuumed dust is disposed of, either it likely is transferred to a landfill or it is incinerated with the other household and commercial waste. Some PBDE dust may directly transfer from indoor spaces to the outdoor air by

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convection through open doors and windows. The indoor air levels of PBDE contaminated dust are expected to be highly variable from one building or house to another. This variability is caused by such factors as the age and numbers of PBDE-treated materials within the indoor spaces, the ventilation rate, the rate of fresh air exchange within the indoor spaces, and the active use of PBDE-treated products and materials (Hazrati and Harrad, 2006).

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. 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 BDE 47, BDE 99, BDE 100, and BDE 66 in concentrations ranging from 0.13 to 10 pg/L (for individual congeners). Three congeners were detected in the particle phase: BDE 47, BDE 99, and BDE 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 BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 156, BDE 183, and BDE 209 in the sea-surface microlayer (SML) and subsurface seawater (SSW) from locations off the coast of Hong Kong. The SML of the seawater was defined as a 100-μm thick boundary layer between the atmosphere and the ocean surface that is composed of naturally occurring organic matter and μm-sized suspended particles. The subsurface was defined as 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

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from 26.2 to 32.5 pg/L in the particle phase. BDE 209 was detected at trace levels in the dissolved phase at all sampling locations. Only the BDE congeners BDE 28 and BDE 47 were detected in the particle phase of the SML. Below the SML in the SSW, BDE 28 and BDE 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 biomagnification 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 higher-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 effect 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

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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 BDE 47, BDE 99, and BDE 209 from PBDE sources. Fugacity modeling suggested that approximately 68% of DE and 98% of BDE 47, BDE 99, and BDE 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 positive relationships 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 BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154 (Hassanin et al., 2004), but other soil data show that BDE 209 dominates total soil PBDE concentration (Cetin and Odabasi, 2007), especially in urban areas. However, Hassanin et al. (2004) did not include BDE 209 as an analyte in the chemical analysis of soil samples. It is possible that BDE 209 would have been shown to dominate in this study if BDE 209 had been included. 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 octaBDE formulations to decaBDE, as well as high sorption and persistence of BDE 209. Further discussions of PBDEs in surface soils are found in Section 4.3.

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3.6.4. PBDEs in Sewage Treatment Plant Influent, Effluent, and Sludge

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 subject 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 95×10^6 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 composed of BDE congeners BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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, BDE 47, BDE 99, and BDE 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 unknown whether BDE 209 in sewage sludge degrades photolytically or microbially to form lower-brominated congeners.

Recently, the U.S. Environmental Protection Agency (EPA) conducted an updated National Sewage Sludge Survey (NSSS) of PBDEs and other contaminants present in sewage sludges in the United States (U.S. EPA, 2009). This information is used to estimate the amount

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of PBDE contamination in the sewage sludge that is applied to land as a soil amendment and fertilizer. The NSSS was a statistically-based survey in which sewage sludges were randomly selected from 74 STPs in 35 states to represent the United States as a whole. Samples were collected between August 2006 and March 2007. The mean concentrations (µg/kg dwt) of PBDE congeners in sewage sludge were BDE 47 = 709.17; BDE 99 = 716.36; BDE 153 = 68.33; and BDE 209 = 2,181.23. On a national basis, the dominant congener was BDE 209.

Knoth et al. (2007) investigated the distribution of BDE 28, BDE 47, BDE 99, BDE 153, BDE 154, BDE 183, and BDE 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 BDE 28, BDE 47, BDE 99, BDE 153, BDE 154, BDE 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 BDE 17, BDE 28, BDE 47, BDE 66, BDE 71, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 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 nondetect 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)

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regressed the data and determined correlations with BDE congener pairs. A significant and positive correlation ($r = 0.814$, $p < 0.001$) was found between BDE 47 and BDE 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 BDE 28, BDE 47, BDE 71, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, and BDE 183 were evaluated in the study. BDE 209 was not included in the list of analytes. The congeners of commercial pentaBDE formulation (BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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 composed of BDE 47 and BDE 99. BDE 47 and BDE 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 BDE 47 and BDE 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 BDE 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/day STP were as follows. Of the 10,560 mg/day total PBDEs entering the STP, approximately 9,609.6 mg/day (91%) ends up in the STP sludges (primary settling and activated sludge), and 950.4 mg/day (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

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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)
	NA	709.17	NA	USA (74 STPs)	U.S. EPA (2009)
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)
	NA	716.36	NA	USA (74 STPs)	U.S. EPA (2009)

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

PBDE congener (Br substitution)	STP influent (ng/L)	STP sludge (µg/kg dwt)	STP final effluent (ng/L)	Location	Reference
BDE 100	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)
	NA	0.86*	NA	Sweden (22 STPs)	Oberg et al. (2002)
	NA	68.33	NA	USA (74 STPs)	U.S. EPA (2009)
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)
	NA	2,181.23	NA	USA (74 STPs)	U.S. EPA (2009)

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

PBDE congener (Br substitution)	STP influent (ng/L)	STP sludge (µg/kg dwt)	STP final effluent (ng/L)	Location	Reference
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)

*Median concentration as opposed to mean.

NA = Not analyzed; ND = not detected.

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. 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 water body 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 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 and 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

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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.

BDE 47, BDE 66, BDE 99, BDE 100, BDE 153, and BDE 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 weight (lwt) in white-beaked 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-Restrepo et al., 2005; 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 of 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 that BDE 209 makes to PBDE in fish tissue.

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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 deepwater 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. Potter et al. (2009) reported on the distribution of PBDE congeners in 23 Peregrine Falcon (*Falco peregrinus*) eggs collected from 1993 to 2002. The eggs were from 13 nests at 11 locations in the region of the Chesapeake Bay. Once collected, the eggs were kept frozen until analyzed. Results of analysis showed that the median total concentration of BDEs (sum of BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, BDE 196, BDE 197, BDE 206, BDE 207, BDE 208, and BDE 209) was 201 ng/g wwt. BDE 209 was detected in all egg samples at a medium concentration of 6.35 ng/g wwt. The authors noted that because of the short biological half-life of BDE 209 in birds (about 13 days), the presence of BDE 209 in egg tissues may indicate relatively recent exposures to the maternal bird (Potter et al., 2009). BDE 209 averaged 5.9% of the sum of the BDE congeners. In this study, the rank order by BDE concentration in Peregrine Falcon eggs (low to high) was BDE 153 > BDE 99 > BDE 100 > BDE 154 > BDE 209 > BDE 183 > BDE 197 > BDE 47 > BDE 207. BDE 47 was a minor constituent in Peregrine Falcon eggs (on average only 4.4%). BDE 153 was the dominant congener present in Peregrine Falcon eggs, with a medium concentration of 26.1 ng/g wwt

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(representing a mean contribution to total PBDEs of 28%). This was similar to BDE congener profile observed in a study of Peregrine Falcon eggs by Chen et al. (2008). The small sample size did not permit a time-trend analysis of PBDE concentrations in eggs. However, BDE 209 concentrations were found to be higher in Peregrine Falcon eggs obtained from urban areas as compared to rural areas. For example, the authors indicated that the mean BDE 209 percent contribution to total PBDEs detected in Peregrine Falcon eggs collected from a rural low human population area was 0%, but the mean percent concentration to total PBDEs collected from a densely populated urban was 18.6% (Potter et al.,2009). This observation was consistent with a Peregrine Falcon egg study by Chen et al. (2008).

Chen et al. (2008) conducted a similar study of Peregrine Falcon eggs. A total of 114 Peregrine Falcon eggs were collected for purposes of investigating the distribution of BDE congeners in egg tissues. Samples were collected from nests in Connecticut, Massachusetts, Maine, New Hampshire, Rhode Island, and Vermont between the years 1996 and 2006. Once collected, the eggs were transferred to solvent-rinsed glass jars and frozen until chemical analysis. Seventeen BDE congeners were analyzed in egg samples, including BDE 47, BDE 99, BDE 100, BDE 119, BDE 138, BDE 153, BDE 154, BDE 183, BDE 197, BDE 196, BDE 201, BDE 202, BDE 203, BDE 206, BDE 207, BDE 208, and BDE 209. Results showed that the total PBDEs (sum of 17 congeners) concentrations ranged from 74.5 to 6610 ng/g wwt, with a median of 440 ng/g wwt. PBDE congener profiles in Peregrine Falcon eggs (defined as percent contribution to total of 17 measured BDE congeners) were dominated by BDE 153, followed by BDE 99, BDE 183, BDE 209, BDE 197, BDE 207, BDE 154, BDE 100, and BDE 196; with lesser contributions from BDE 47, BDE 208, BDE 203, BDE 201, BDE 206, BDE 202, BDE 138, and BDE 119. The third most prevalent congener present in egg tissue was BDE 183. The authors observed that the elevated BDE-183 concentrations (a hallmark of the octaBDE commercial formulation) in Peregrine Falcon eggs may indicate the high biomagnification potential of BDE 183 in terrestrial food chains (Chen et al., 2008). BDE 47 was a minor contributor (<2% on average) in Peregrine Falcon eggs. Time-trend analysis suggested that total PBDE (sum of 17 congeners) contaminant levels in the Peregrine Falcon eggs did not change significantly over a 10-year period from 1996 to 2006 (Chen et al., 2008). The influences of rural versus urban environments on PBDE concentrations in eggs were investigated with the data. There was no statistically significant difference ($p = 0.075$) in the total PBDE

concentrations present in eggs from urban areas (median, 570; range, 150−1910 ng/g ww) and eggs from rural areas (median, 380; range, 75−3,570 ng/g ww). However, BDE 209 was significantly higher in Peregrine Falcon eggs collected in urban areas as compared with eggs collected in rural areas ($p < 0.005$).

Naert et al. (2007) studied the body burden distribution of BDEs in brain and adipose tissue samples of Common Buzzards (*Buteo buteo*), Eurasian Sparrow Hawks (*Accipiter nisus*), Cormorants (*Phalacrocorax carbo sinensis*), and Common 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 BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183) in brain ranged from below the detection limit in Common Blackbirds to 14 ng/g wwt in sparrow hawks. The median concentrations of total PBDEs in adipose tissue ranged from below the detection limit in Common 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 versus the Common Buzzard (34.55 ng/g wwt), Common 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 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 PBDEs in the Cormorant. In the Common Buzzard, the most prevalent congeners detected were BDE 153 (29% of total), BDE 99 (23%), and BDE 47 (22%). The Common Buzzard preys mainly on small mammals (mice, voles, rabbits, squirrels, rats, and moles). The Common 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 Common Buzzards: BDE 153 > BDE 47 > BDE 99 > BDE 183 > BDE 100 > BDE 154; for Sparrow Hawks: BDE 99 > BDE 47 > BDE 153 > BDE 100 > BDE 183 > BDE 154; for owls BDE 153 > BDE 99 > BDE 47 > BDE 183 > BDE 100 > BDE 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 samples from sparrow hawks, BDE 209 was detected at a mean concentration of 17 ng/g lwt in liver, and 26 ng/g lwt in serum. In buzzards, BDE 209 was detected only at a frequency of 3 out of 29 (10%) in liver (mean = 79 ng/g lwt). However, in the serum of Common Buzzards, the frequency of detection was a surprising 16 out of 20 samples (80%). Chen et al. (2007a) also confirmed the presence of BDE 209 in liver of three Common 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 was 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 biomagnification 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 biomagnification 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 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 > BDE 99 > BDE 47 > BDE 183 > BDE 154 > BDE 209 > BDE 207. This differed from the BDE profile in the tissues of Japanese Sparrow Hawks—BDE 99 > BDE 153 > BDE 47 > BDE 209 > BDE 207 > BDE 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 BDE 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*); and 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 2,880 ng/g lwt, respectively, in Forster's Tern eggs were 1,820, 4,380, 5,460 and 3,600 ng/g lwt, respectively, and in California Least Tern eggs 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 (*Vulpes vulpes*) 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 lwt 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 (*Ursus arcos horribilis*) (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 BDE 209 > BDE 99 > BDE 100 > BDE 153. In the interior bears, the tissues were dominated by BDE 209, followed by BDE 206 > BDE 47 > BDE 207 > BDE 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).

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The polar bear (*Ursus maritimus*) diet is chiefly composed of ringed seal. Wolkers et al. (2004) studied the polar cod-ringed 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 greater than those in ringed seal, which in turn, were greater than those in polar cod were. From the 22 brominated compounds measured, only BDE 47, BDE 85, BDE 99, BDE 100, and BDE 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 polar cod. In particular, BDE 47 and BDE 99 were approximately one order of magnitude higher in seals than in polar 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, which was 1.5 and 3 times the body burden of ringed seals, respectively.

3.7.2.3. *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

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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 midge 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.

Sellström et al. (2005) reported on the bioaccumulation of PBDEs from contaminated sewage sludge-amended soils. Earthworms were collected from five sites in Sweden. Three sites were government-operated experimental agricultural research workstations where the land application of sewage sludge was carefully controlled. The other two sites were private farms. One farm accepted the land application of sewage sludge; the other did not. Sewage sludge had been applied to the land at these sites as soil amendment from 1978–1997. The sludge applied to private farmland-received sewage sludge from a local STP. The STP also accepted and treated wastewater from a local textile industry that used PBDEs in their processes. Triplicate soil samples were taken from each site. Earthworms were collected from the same areas as soil samples. Soil and earthworm samples were sent to a laboratory for chemical analysis using gas chromatography coupled with mass spectrometry run in the chemical ionization mode. In soil samples, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 154 were analyzed, and when reported, the total PBDE includes these congeners. In earthworm samples, BDE 35, BDE 47, BDE 49, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, BDE 196, BDE 197, BDE 198, BDE 203, BDE 206, BDE 207, and BDE 209 were analyzed, and these comprised total PBDEs when results were reported. The researchers examined the relationship between the application rates of sewage sludge and the resulting levels of PBDEs in amended soils. In general, they found that the higher the amounts of PBDE-contaminated sludge applied to land, the more the BDE concentrations increase in soils, including BDE 196, BDE 197, BDE 206, BDE 207, and BDE 209 (Sellström et al., 2005). BDE 209 concentrations in soils from the research workstations and a private farm ranged from 0.015 to 0.75 ng/g dwt. Earthworms ingest soils as they move through soil. When in contact with the

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PBDE-contaminated soils, the earthworms accumulated all the BDE congeners analyzed. Total PBDEs in earthworms ranged in concentration of all sludge-amended sites from 33 to 38,000 ng/g lwt. The highest whole worm concentration of total PBDEs was from the farm where sewage sludge contaminated with PBDEs from a textile industry was used as soil amendment. BDE 209 concentrations in earthworms ranged in mean concentration of from not detected to 5,200 ng/g lwt. Other congeners showed the following ranges in mean concentrations, in ng/g lwt: BDE 47: 1.4−10,000; BDE 85: not detected (ND): 290; BDE 99: 14−13,000; BDE 153: 1.1−1,200; BDE 154: ND: 870; BDE 183: ND: 0.71; BDE 206: ND: 830; and BDE 207: ND: 150.

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. However, several of the sediment core studies showed concentrations in cores dated prior to the 1970s. Their presence at these layers was likely due to sediment bioturbation, imprecise dating, or a similar explanation, since PBDEs were not in the environment at these early dates. After the 1970s, the presence of PBDEs in the environment increased throughout the remainder of 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), section the cores by taking slices, and date the slices using radiotracers. Once a section is dated, then a measurement of the contaminant in that segment provides an indication of relative environmental levels during that time frame. Further, knowing the deposition rate of sediments

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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, 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. Analytical results are separately reported as the sum of nine PBDE (Σ_9BDEs) congeners (BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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 PCBs to 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. This is not a reflection of when use of PBDEs began, because they were manufactured in the 1970s. Rather, it is likely the result of sediment bioturbation once deposition occurred, or cross contamination during the coring and slicing process. There is a striking increase in the deposition of total PBDE to the lake sediments from 1970s 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 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, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183 combined.

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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.

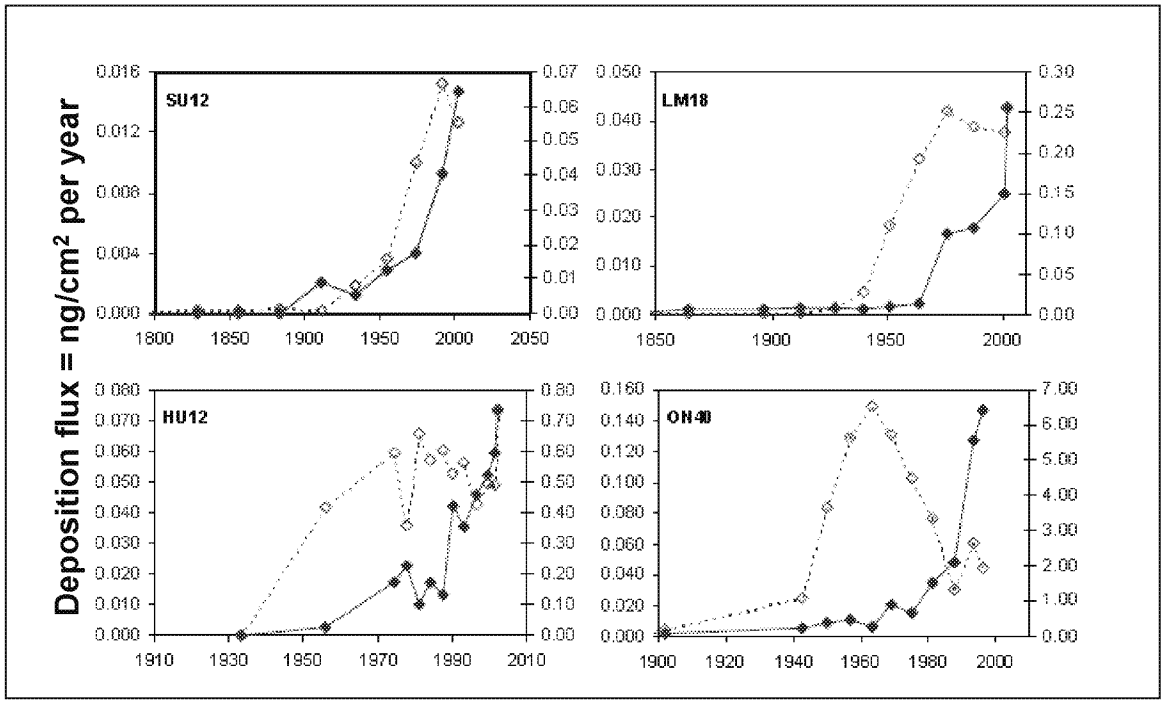

Figure 3-1. Time trends of deposition flux of PBDEs (blue solid diamonds) and PCBs (red open diamonds) to the sediments in each of the Great Lakes. (Left x-axis shows PBDE levels, and right x-axis shows PCB levels).

Source: Li et al. (2006).

Notes: SU12 = 12 samples from Lake Superior; LM18 = 18 samples from Lake Michigan; HU12 = 12 samples from Lake Huron; ON 48 = 48 samples from Lake Ontario. The x-axis shows the year dated by Pb-210 in the sediment core.

In the sediment surface corresponding to 2004, Qiu et al. (2007) detected $\Sigma_{3\text{-}7}$PBDE congeners (i.e., tri thru hepta BDE congeners BDE 28, BDE 47, BDE 49, BDE 99, BDE 100, BDE 116, BDE 153, BDE 154, BDE 181, and BDE 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 five 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. $\Sigma_{3\text{-}7}$PBDE congeners were initially detected around 1955, gradually increased in concentration between 1960 and 1990, and sharply increased in concentration from 1990 through 2000. These early detections before 1960, as noted earlier, do not reflect use at that time, but rather perhaps sedimentation or cross contamination during coring and slicing.

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 a detection limit of 1 ng/g dwt) to rise to 38 ng/g dwt 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 frames 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 versus 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 three 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 1980s and early 1990s. BDE 209, in particular, displayed an exponential increase in concentration in the sediments from the time frame 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 deposition flux of total congeners, not including BDE 209, to the Peal River delta in 2005, to be 2.1 and 29.7 metric tons (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-year. 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 that 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 1940s through 2001. Of the 10 BDEs, only BDE 28, BDE 47, BDE 99, BDE 100, and BDE 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, BDE 28, and BDE 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 (Evenset 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: Zegers at al. (2003) showed rising concentrations from about the 1970s to the present, with dominance of BDE 209, particularly in the later years. From

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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 threefold 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 BDE 99 and BDE 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 cores dated 1965 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, BDE 47 and BDE 99 reached high concentrations—about 20 ng/g dwt. Concentrations of 12 other congeners were mostly nondetects (at detection limits of about 1–3 ng/g dwt).

Many of the aforementioned sediment core studies suggest the presence of PBDEs in lake sediments prior to 1976 when PBDEs were intentionally manufactured (IPCS, 1994). This suggests that the core slices may not be precisely dated (relative dates rather than actual dates), or that mixing (bioturbation) from more recent sediment layers to lower layers may have occurred. However, when considered in their entirety, the sediment core studies do indicate the anthropogenic contribution of PBDE emissions to the environment.

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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 has 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, unlike sediment core studies, these show a predominance of BDE 47 and a virtual absence of BDE 209.

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 frame 1988 through 1999, and samples were collected during these years and frozen for future analysis. The total PBDE concentration (including BDE 28, BDE 47, BDE 71, BDE 77, BDE 99, BDE 100, BDE 153, BDE 154, BDE 155, and BDE 183) ranged from about 20 to almost 1,000 ng/g wwt in samples from 54 whales. The data suggest 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 (BDE 3, BDE 15, BDE 28, BDE 47, BDE 99, BDE 153, BDE 154, BDE 183, and BDE 209). The sum of the PBDE congeners ranged in concentration from a low of 0.33 ng/g lwt in the year 1972 up to 100 ng/g lwt in the year 1994. 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 during the time frame from 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 EPA. Only BDE congeners BDE 47, BDE 99, BDE 100, and BDE 153 were

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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 greater than 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 BDE 183 and BDE 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 six 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 fish 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:

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1. In general, PBDE congeners are lipophilic and persistent organic compounds having a propensity for bioaccumulation and biomagnification in the aquatic and terrestrial food webs. BDE 209 is a large molecule having reduced tendency for bioaccumulation. However, BDE 209 has been measured in tissues of terrestrial birds and mammals, and aquatic fish and mammals. Therefore, BDE 209 is contaminating the aquatic and terrestrial food chains.

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 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 phases in the atmosphere in accordance with their respective vapor pressures. The BDE congeners with 1−4 bromine atoms primarily exist in the vapor phase; BDE congeners with 5−6 bromine atoms more generally partition between the vapor phase and the particle 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 in soils by UV light (i.e., photolysis). Photo-degradation in soils can debrominate the higher BDE congeners to form lower-brominated BDE congeners and may be a significant degradation pathway in the environment. Photolysis of BDE 47 and BDE 99 in air is a major atmospheric removal process for these congeners. In contrast, the photolysis of BDE 209 in air is a minor atmospheric removal process. Atmospheric wet and dry deposition of BDE 209 is the most significant atmospheric removal pathway. This is likely due to the fact that BDE 209 predominantly exists bound to particles in air.

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

9. There exists good evidence that microbial anaerobic degradation naturally occurs in sediments and soils. However, the degradation rate has yet to be determined.

10. Soils and sediments are environmental sinks for PBDEs.

11. 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.

12. The environmental fate of BDE congeners is dependent on their respective chemical and physical properties.

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13. A principal source of PBDE in indoor microenvironments is household and commercial dust. PBDEs volatilize from PBDE-treated products to indoor dust, and or transfer to dust from the abrasion of products.

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4. ENVIRONMENTAL AND EXPOSURE MEDIA CONCENTRATIONS

4.1. INTRODUCTION

This chapter summarizes the concentrations of individual congeners and total polybrominated diphenyl ethers (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 brominated diphenyl ether (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 (BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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%). BDE 183 is often considered a marker for the octa formulation because it is a primary congener found in this formulation, while it is not found in the penta or deca formulations. Also, it is persistent in the environment and often found at low levels when measured. There is suggestive evidence that photolytic debromination of BDE 209 results in the formation of BDE 183 (Stapleton and Dodder, 2008), so its presence could also reflect past use of the deca formulation. As the deca formulation is the primary formulation of PBDEs marketed and used worldwide, BDE 209 is now more regularly measured, but that was not always the case. Surveys in the 1990s into the 2000s mostly did not include BDE 209, and even current surveys often do not include this critical congener. Analytical difficulties, evidence that it is rapidly eliminated from biota, and citation of previous literature focusing on the prevalence of the penta formulation congeners, are the primary reasons given for not measuring or reporting BDE 209. While nearly all studies include at least the five key congeners of the penta formulation, some studies report on the concentrations of over 20 congeners. Unlike dioxin-like compounds where 7 dioxins and 10 furans have been identified as toxic congeners of concern, there is no list of key toxic BDE congeners. Subsequently, there is not uniformity on which congeners should be measured, and when citing a "total" concentration (sum of all congeners) measured in a study, one must be careful to identify which congeners constitute that total. Efforts were made in the discussions below to indicate which congeners were measured in

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a study, and when referencing "total" for each study, it is only the total of the congeners measured. The original references should be obtained if the discussions below are not clear.

The sections on air, house 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 are little or no U.S. 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 CONCENTRATIONS

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, and clams) in the SF Estuary for 22 BDE congeners, including BDE 209 (Oros et al., 2005). Thirty three water samples were taken, with total PBDEs ranging from 3−513 pg/L, and a mean concentration of 146.2 pg/L. The region of the bay that had the highest concentrations, the Lower South Bay, receives 26% of the Estuary's total publicly owned treatment works wastewater effluents and only 10% of the Estuary's freshwater inflow. The most abundant congeners were BDE 47, BDE 99, and BDE 209. When BDE 209 was not reported as "Q" (outside QA limits, detected but not reported), it was present and quantified, and of a comparable magnitude as BDE 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. BDE 17, BDE 100, and BDE 28/33 (congeners notated as "28/33" translate to congeners that have coeluted 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.

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In a second study, Johnson et al. (2006), sampled for PBDEs in water and fish of Washington state rivers and lakes. Results from 15 samples taken in seven rivers and three lakes in 2005 and 2006 were available. Congeners measured included BDE 47, BDE 49, BDE 66, BDE 71, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, BDE 184, BDE 190, and BDE 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 BDE 47, BDE 99, and BDE 100.

Finally, Streets et al. (2006) measured PBDEs in the dissolved and particle phases in Lake Michigan and found BDE 47, BDE 99, BDE 100, and BDE 66 in the dissolved phase in concentrations ranging from 0.13 to 10 pg/L (for individual congeners). The quantified particle phase concentrations for BDE 47, BDE 99, and BDE 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 513 pg/L, dominated by BDE 47, BDE 99, and BDE 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 two 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 BDE 47, BDE 66, BDE 99, and BDE 100 ranged up to10 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

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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 BDE 47, BDE 99, BDE 183, BDE 204, and BDE 205. It is noteworthy that BDE 209 was not found with a detection limit (DL) of 1.5 ng/g. This contrasts with all other sediment studies where BDE 209 dominated the profile. It is also the only study found that provided concentrations of BDE 204 and BDE 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 and a mean of 12 ng/g dwt. BDE 99 was detected 77% of the time with a range and mean of 0.3 to 71 and 5 ng/g dwt, respectively. BDE 183, BDE 204, and BDE 205 were detected once, twice, and once, respectively.

Hun Yun et al. (2008) sampled PBDEs and polychlorinated biphenyls (PCBs) in sediment and flood plain soils of the Saginaw River Watershed in Michigan. They took 53 samples representing 0–12 cm in October–November 2004. They sampled for 10 congeners: BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, and BDE 209. The total concentration (sum of these congeners) ranged from 0.04–49.9 ng/g dwt, with an average of 3.75 ng/g dwt. BDE 209 dominated the concentration, explaining 3.19 ng/g dwt of this average.

Song et al. (2005a, b; 2004) collected 22 sediment cores in 2001 and 2002 and horizontally sectioned them into a total of 247 samples. Analytical results were separately reported as the sum of nine PBDE (Σ_9BDEs) congeners (BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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.

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

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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 accounting 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/year 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 in Section 4.6.2). The sediment samples were collected in 2002 as part of an earlier 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 dwt—the proportions were similar. BDE 209 explained about 50% of the concentration, while BDE 99 and BDE 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 BDE concentrations, including BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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 (PUF) manufacturing facility in the U.S. mid-Atlantic Region, which had ceased production in 1997; sampling occurred in 2001. An interior duct of the facility was tested, as was soil adjacent to the facility, sediment in a stream leaving the facility, and sediment and bluegill in a pond about 250 m from the facility. Total concentrations of PBDEs (including BDE 47, BDE 99, and BDE 100; BDE 153, and BDE 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 nondetects. 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,

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suggesting that the foam production facility did not result in increases to sediment concentrations compared 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 largest 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 BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154 ranged from nondetect 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%), BDE 153 (<5%), and BDE 154 (<5%).

Much higher concentrations have been identified in sediments directly downstream of a known source or in heavily industrialized areas. La Guardia et al. (2007) reported on sediment concentrations downstream of a wastewater treatment plant of a plastics manufacturer in North Carolina. In eight sample points downstream of the outfall, four each in 2002 and 2005, they found total PBDEs (sum of 19 congeners, including BDE 47, BDE 99, BDE 100, BDE 153, BDE 183, BDE 203, BDE 206, BDE 207, BDE 209, and others) to range from about 300 to over 3,000 parts per million (ppm; μg/g), with BDE 209 comprising over 95% of the total. This is over 4 orders of magnitude higher than reported above for background settings. They also took two samples upstream, in 2002 and 2005, and found much lower but still quite elevated concentrations of total PBDEs, at 38 and 97 ppm.

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 nondetect to 60.9 ng/g dwt, with an overall mean (± standard deviation) and median of 4.7 ±12.6 and 0.3 ng/g dwt, respectively. BDE 209 dominated the congener distribution in 86% of the sediment samples.

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Christensen and Platz (2001) sampled sediment from Danish marine coastal areas, freshwater lakes, and a river in 2000. The congeners measured included BDE 47, BDE 99, BDE 100, BDE 153, and BDE 209, and the total concentrations ranged from 0.06–24.7 and 0.07–10.6 ng/g dwt 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 dwt and 2.05 ng/g dwt, respectively. The rank order of BDE congeners by concentration in sediment was BDE 209 > BDE 99 > BDE 47 > BDE 100 > BDE 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: BDE 28, BDE 33, BDE 47, BDE 66, BDE 77, BDE 100, BDE 99, BDE 118, BDE 154, BDE 153, BDE 183, and BDE 209. Total PBDE levels ranged from 2.7 to 134 ng/g dwt in coastal marine sediments, with the highest levels of 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 BDE 209 > BDE 47 > BDE 99 > BDE 100 > BDE 153.

Water body sediment studies in North America included one in the San Francisco Estuary, along the Mississippi and Delaware Rivers, one in a Virginia watershed, one in a Michigan watershed, one in a pond near a closed polyurethane foam manufacturing facility in North Carolina, and several surficial sediment and core studies describing trends in the Great Lakes (some of these 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 dwt), and BDE 47 dominated the profile. In a few studies measuring BDE congeners in industrial settings and near outfalls of wastewater treatment plants of facilities producing PBDEs, substantially higher concentrations in

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the hundreds of μg/g (ppm) range are found, with over 90% of the total concentrations being BDE 209.

4.3. SURFACE SOIL CONCENTRATIONS

Only two systematic studies could be found that looked at PBDEs in surface soils in the United States (Offenberg et al., 2006; Hun Yun et al., 2008) in predominantly suburban and/or background settings. Offenberg et al. (2006) took a total of 33 surface soil samples 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 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 three samples at concentrations, ranging from 121 to 562 ng/g dwt, so that the survey-wide average was 37.4 ng/g dwt. Hun Yun et al. (2008) took 26 samples in floodplain soils of the Saginaw River Watershed in Michigan in October–November 2004. They measured for 10 congeners (BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, and BDE 209) to a depth of 2.5 cm. Total concentrations (sum of 10 congeners) ranged from 0.02–55.1 ng/g dwt, with an average of 5.6 ng/g dwt. BDE 209 dominated the profile, explaining 5.4 ng/g dwt of this average.

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

Two European studies 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) was taken from grassland and woodland areas in the United Kingdom (UK) and Norway. These samples were collected in 1998, and 20 BDEs were measured, although BDE 209 was not measured. The congeners most routinely detected (85–100% of the time) in the samples were BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183. Major trends

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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, minimum/maximum 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; and 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, BDE 99 averaging 40%, BDE 100 averaging 6%, BDE 153 averaging 8.7%, and BDE 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, dust, and other soil studies, its lack of measurement in this broad ranging surface soil sampling study is unfortunate.

A second study looked at soil samples in a rural-urban transect across the West Midlands of the United Kingdom, which includes the major urban center of Birmingham. The purpose of the study was to ascertain whether there was a relationship between concentrations and characterization of the location of sample (urban, suburban, or rural). Soil concentrations were found to be consistent with earlier sampling in the United Kingdom (Hassanin et al., 2004). Total soil concentrations (which included BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154) taken at a 0 to 5-cm depth for 10 sites ranged from 0.07 to 3.89 ng/g dwt. The maximum concentration of 3.89 ng/g was found in the city of Birmingham, while other concentrations were 0.84 ng/g dwt and less. The ratio of the two primary congeners found, BDE 47 to BDE 99, ranged from 0.51 to 0.85.

Cai and Jiang (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 (monoBDE); BDE 15—0.76 ng/g dwt (DiBDE); BDE 28—5.01 ng/g dwt (triBDE); BDE 47—217 ng/g dwt; BDE 99—552 ng/g dwt; BDE 139—35 ng/g dwt; BDE 153—74 ng/g dwt; BDE 154—49 ng/g dwt; and

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BDE 183—12.3 ng/g dwt. The total concentration is 945 ng/g dwt. The authors state that the isomer pattern of BDE 47, BDE 99, BDE 139, BDE 153, and BDE 154 is consistent with the pentaBDE formulation. No measurements of BDE 209 were made in this study.

In summary, the sparse literature suggests background soil concentrations of total BDEs in the United States 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 pentaBDE formulation. However, BDE 209 mostly has not been sampled 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. DUST CONCENTRATIONS

4.4.1. House Dust Concentrations

House dust was the focus of several U.S. studies because of the concern for indoor exposures in residences. Data were also available in a study on concentrations in a computer laboratory. As will be shown, house dust from both homes and places of work are dominated by BDE 47, BDE 99, and BDE 209. Table 4-1 provides congener-specific concentrations for U.S. house 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 in house dust 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, BDE 47, BDE 99, and BDE 209. The mean concentrations of these three congeners were 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 televisions and personal computers (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 (i.e., the larger the home, the smaller the BDE 209 concentration). Clothes dryer lint, examined in five of the homes, showed concentrations ranging from 480 to 3,080 ng/g dwt. The two house dust

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Table 4-1. Congener-specific concentrations of PBDEs in house dust in the United States (units in ng/g dwt)

Congener	Concentration (ng/g dwt)	Comment	Reference
DiBDE			
15	11	Mean 10 homes throughout U.S., but 9 ND, 1 at 109	Sharp and Lunder (2004)
TriBDE			
17	9	Mean 17 homes, Washington, 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	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom., and vacuum	Allen et al. (2008)
25	2	Carpet dust (n = 2) from computer labs in CA	CARB (2005)
28	21	Mean 17 homes, Washington, DC, also listed as BDE Congener 33	Stapleton et al. (2005)
	ND (50)	Mean 10 homes throughout U.S.	Sharp and Lunder (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, Texas from 2006 (n = 20)	Harrad et al. (2008b)
	0.25, <0.1	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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	Sharp and Lunder (2004)
28/33	16.3, 10.5, 6.4	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	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)

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Table 4-1. Congener specific concentrations of PBDEs in house dust in the United States (units in ng/g dwt) (continued)

Congener	Concentration (ng/g dwt)	Comment	Reference
TetraBDE			
47	1,220	Mean 17 homes, Washington, DC	Stapleton et al. (2005)
	1857	Mean 10 homes throughout U.S.	Sharp and Lunder (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. (2008)
	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	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
	669, 14613	Median, maximum from 11 homes in the Boston, MA area	Wu et al. (2007)
	810, 470	Mean, geometric mean from homes in Amarillo/Austin, Texas from 2006 (n = 20)	Harrad et al. (2008b)
	3750	Median, n = 49 homes in Northern CA	Zota et al. (2008)
	2920; 655−8030	Median, range, n = 11 homes in Davis, CA	Hwang et al. (2008)
	93, 40	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
49	ND	Carpet dust (n = 2) from computer labs in CA	CARB (2005)
	29.6, 23.6, 12.4	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
66	28.5	Mean 17 homes, Washington, DC	Stapleton et al. (2005)
	21	Mean 10 homes throughout U.S., but 8 ND, 2 at about 100	Sharp and Lunder (2004)
	20.3	Carpet dust (n = 2) from computer labs in CA	CARB (2005)

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Table 4-1. Congener specific concentrations of PBDEs in house dust in the United States (units in ng/g dwt) (continued)

Congener	Concentration (ng/g dwt)	Comment	Reference
	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	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
	ND, 293.0	Median, maximum from 11 homes in the Boston, MA area	Wu et al. (2007)
	1.2, <1.3	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
71	ND	Mean 17 homes, Washington, 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	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	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)
	1.9, <1.3	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
PentaBDE			
85	83	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	100	Mean 10 homes throughout United States, but 8 ND, 2 at 453 and 544	Sharp and Lunder (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, maximum from 11 homes in the Boston, MA area	Wu et al. (2007)

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Table 4-1. Congener specific concentrations of PBDEs in house dust in the United States (units in ng/g dwt) (continued)

Congener	Concentration (ng/g dwt)	Comment	Reference
	8.8, 4.7	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
85/155	124.0, 51.8, 19.2	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
99	1,700	Mean 17 homes, Washington 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	Sharp and Lunder (2004)
	176, 95	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
	880 (70–3,700)	Median; range from 10 homes in Atlanta	Sjodin et al. (2008)
	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	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
	1014, 14979	Median, maximum from 11 homes in the Boston, MA area	Wu et al. (2007)
	1400, 840	Mean, geometric mean from homes in Amarillo/Austin, Texas from 2006 (n = 20)	Harrad et al. (2008b)
	3830	Median, n = 49 homes in Northern CA	Zota et al. (2008)
	4430; (807–11700)	Median, range, n = 11 homes in Davis, CA	Hwang et al. (2008)
100	274	Mean 17 homes, Washington 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	Sharp and Lunder (2004)

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Table 4-1. Congener specific concentrations of PBDEs in house dust in the United States (units in ng/g dwt) (continued)

Congener	Concentration (ng/g dwt)	Comment	Reference
	150 (<8–660)	Median; range from 10 homes in Atlanta	Sjodin et al. (2008)
	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	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
	174, 2776	Median, maximum from 11 homes in the Boston, MA area	Wu et al. (2007)
	240, 160	Mean, geometric mean from homes in Amarillo/Austin, Texas from 2006 (n = 20)	Harrad et al. (2008b)
	756	Median, n = 49 homes in Northern CA	Zota et al. (2008)
	793; 165–2,000	Median, range, n = 11 homes in Davis, CA	Hwang et al., 2008
	30.6, 16.1	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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)
	<0.04, <0.04	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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, Washington DC	Stapleton et al. (2005)
	181	Mean 10 homes throughout U.S., but 8 ND, 1 at 1,668	Sharp and Lunder (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)

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Table 4-1. Congener specific concentrations of PBDEs in house dust in the United States (units in ng/g dwt) (continued)

Congener	Concentration (ng/g dwt)	Comment	Reference
	20.9, 12.1, 5.2	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
	ND, 77	Median, ng/g, from 11 homes in the Boston, MA area	Wu et al. (2007)
	2.28, <0.04	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
153	181	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	243	Mean 10 homes throughout U.S., but 7 ND, 1 at 1,510	Sharp and Lunder (2004)
	140 (5−650)	Median; range from 10 homes in Atlanta	Sjodin et al. (2008)
	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	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
	107, 563	Median, maximum from 11 homes in the Boston, MA area	Wu et al. (2007)
	240, 120	Mean, geometric mean from homes in Amarillo/Austin, Texas from 2006 (n = 20)	Harrad et al. (2008b)
	30.2, 25.9	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
154	156	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	156	Mean 10 homes throughout U.S., but 8 ND, 1 at 1,050	Sharp and Lunder (2004)
	80 (<4−260)	Median; range from 10 homes in Atlanta	Sjodin et al. (2008)
	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)

Table 4-1. Congener specific concentrations of PBDEs in house dust in the United States (units in ng/g dwt) (continued)

Congener	Concentration (ng/g dwt)	Comment	Reference
	182.8, 94.4, 35.0	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	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, Texas from 2006 (n = 20)	Harrad et al. (2008b)
	17.4, 9.6	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
155	8	Carpet dust (n = 2) from computer labs in CA	CARB (2005)
156	ND	Mean 17 homes, Washington 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, Washington DC	Stapleton et al. (2005)
	60	Mean 10 homes throughout United States, but 9 ND, 1 at 604	Sharp and Lunder (2004)
	70 (<4–4,000)	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	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
	28, 16	Mean, geometric mean from homes in Amarillo/Austin, Texas from 2006 (n = 17)	Harrad et al. (2008b)
	18, <21	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
184	ND	Mean 17 homes, Washington DC	Stapleton et al. (2005)

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Table 4-1. Congener specific concentrations of PBDEs in house dust in the United States (units in ng/g dwt) (continued)

Congener	Concentration (ng/g dwt)	Comment	Reference
190	5	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	24	Carpet dust (n = 2) from computer labs in CA	CARB (2005)
191	ND	Mean 17 homes, Washington DC	Stapleton et al. (2005)
OctaBDE			
196	15	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	3.6, 2.6, 3.9	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
197	17	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	2.7, 3.3, 5.6	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
203	109	Carpet dust (n = 2) from computer labs in CA	CARB (2005)
	3.6, 3.6, 4.9	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
	<3, <3	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
NonaBDE			
206	51	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	76.3, 48.1, 40.5	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
207	30	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	45.9, 25.3, 26.6	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)

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Table 4-1. Congener specific concentrations of PBDEs in house dust in the United States (units in ng/g dwt) (continued)

Congener	Concentration (ng/g dwt)	Comment	Reference
	<0.1, <0.1	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
208	35	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	35.6, 17.5, 29.4	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
DecaBDE			
209	2,090	Mean 17 homes, Washington DC	Stapleton et al. (2005)
	2,394	Mean 10 homes throughout United States	Sharp and Lunder (2004)
	2000 (120–21,000)	Median; range from 10 homes in Atlanta	Sjodin et al. (2008)
	7500	Carpet dust (n = 2) from computer labs in CA	CARB (2005)
	665, 8,567	Median, mean (n = 9) from vacuum samples in Dallas, TX in 2004	Schecter et al. (2005)
	4,502, 1,703, 1,811	Geometric mean of 3 locations/home from 20 homes in Boston, MA area; living room, bedroom, and vacuum	Allen et al. (2008)
	ND, 9,020	Median, ng/g, from 11 homes in the Boston, MA area	Wu et al. (2007)
	1,600, 1,300	Mean, geometric mean from homes in Amarillo/Austin, Texas from 2006 (n = 17)	Harrad et al. (2008b)
	2,810, 903	Mean, median from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)

samples with the highest concentration had the commercial pentaBDE commercial ration of 0.6 for BDE 47/BDE 99.

Clothes dryer lint was studied from U.S. and German sources (Schecter et al., 2009b). Dryer lint from 12 U.S. and 7 German homes were analyzed for 13 congeners: BDE 17, BDE 28, BDE 47, BDE 66, BDE 77, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154,

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BDE 183, and BDE 209. The U.S. homes were in Dallas, and the German homes were in Hamburg. The U.S. household lint samples ranged from 321 to 3,073 ng/g dwt, with a median of 803 and a mean of 1,138 ng/g dwt. The German households ranged from 30 to 3,069 ng/g dwt, with a median of 71 and a mean of 361 ng/g dwt. BDE 47, BDE 99, and BDE 209 comprised more than 90% of the sum of the 12 congeners in the U.S. samples. For the median, for example, BDE 47, BDE 99, and BDE 209 comprised 180, 204, and 273 ng/g dwt of the total of 803 ng/g dwt.

Ten women from a prior Environmental Working Group 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 ranged 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 typical 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, BDE 47, BDE 99, and BDE 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, Germany, Australia, and the United Kingdom (Sjodin et al., 2008; first partially reported in Sjodin et al., 2004) for a total of 40 samples. These samples were analyzed for BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209. The U.S. concentrations were uniformly higher than the German and Australian data: the range of total PBDEs (sum of the 7 congeners) in Germany was 17–550 ng/g dwt (median = 74) and in Australia was 500–13,000 ng/g dwt (median = 1,200), while in United States, the range was 530–29,000 ng/g dwt (median = 4,200). The UK data were the most unique. Concentrations of six congeners, not

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including BDE 209, were generally low (medians less than 30 ng/g dwt), but the median for BDE 209 was 10,000 ng/g dwt with a range of 910–54,000 ng/g dwt. 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 from those made by others, who note that the ratio of BDE 47 to BDE 99 in commercial penta formulations is about 0.60.

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 between 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 BDE 99 and BDE 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 three BDEs (BDE 47, BDE 99, and BDE 100) were analyzed, and the reporting limits were high—400 ng/g dwt for BDE 47 and BDE 99 and 300 ng/g dwt for BDE 100. Because of this, the data had limited value. 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 with a maximum of 3,400 ng/g dwt.

Johnson-Restrepo and Kannan (2009) collected dust samples from the vacuum bags of residents of 12 homes in Albany, NY, between December 2007 and January 2008. Twenty congeners from BDE 28 to BDE 209 were measured in the dust. BDE 209 was the major congener found, accounting for between 26 and 99% of the total concentration. The mean of the 12 samples was 3,190 ng/g, the median was 1,910 ng/g, and the range was 380 to 9,340 ng/g.

Four computer wipe and nine 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

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because no other data were available with which to compare these units, 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, BDE 99 and BDE 47 were next in predominance, accounting for 25% and 11%, respectively. In nine 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 seven of nine samples (BDE 99 was the dominant in the other two 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 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 4,702, 1,866, and 1,811 ng/g dwt, respectively. BDE 99 was second highest at 2,460, 1,170, and 536, respectively, and BDE 47 was the third most prevalent at 1,865, 837, and 338, respectively. The geometric means of the total concentrations (including BDE 17, BDE 28/33, BDE 47, BDE 49, BDE 66, BDE 75, BDE 85/155, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, BDE 196, BDE 197, BDE 203, BDE 206, BDE 207, BDE 208, and BDE 209) of the three locations were 13,732, 6,255, and 4,269 ng/g dwt, respectively. They found a correlation between air and dust concentrations of the penta formulation congeners (BDE 17 up to BDE 154), but did not find a correlation between BDE 209 air and dust concentrations. Their highest concentration of 544,000 ng/g dwt was dominated by BDE 209, at 527,000 ng/g dwt.

In another study of the Boston, MA, area, Wu et al. (2007) sampled 11 houses and measured for BDE 17, BDE 28/33, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 209. This was a study in which women's 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 BDE 47 were

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quantified in nearly all samples, with median concentrations of 1,010 and 670 ng/g dwt, respectively. BDE 209 was quantified in 5 of 11 samples, with quantified concentrations ranging between 1,360 and 9,020 ng/g dwt. The median total concentration was 1,910 ng/g dwt. They found a correlation of $r = 0.76$ ($p = 0.003$) between concentrations of pentaBDE congeners in the breast milk of first time mothers and dust sampled from their homes. There was also a statistically significant correlation between dust levels and with reported dietary habits, particularly the consumption of dairy products.

Harrad et al. (2008b) 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 BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209. The most interesting finding was 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 have 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 were found in homes in the United Kingdom, at 520,000 and 100,000 ng/g dwt (Harrad et al., 2008b). The concentrations of the tri-hexa congeners (BDE 28 through BDE 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 concentrations were highest for the United Kingdom, similar to the findings of Sjodin et al. (2008). The geometric mean total concentrations for the United Kingdom, United States, 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 BDE 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 United Kingdom.

Zota et al. (2008) took dust samples in 49 homes in Northern California and measured them for BDEs 47, 99, and 100. Median and range of concentrations were (in ng/g dwt): BDE 47—3,750 and 11−107,000; BDE 99—3,830 and 102−170,000; and BDE 100—756 and <MRL—30,900. As Zota et al. (2008) notes, these concentrations were 4−10 times higher than concentrations found for these congeners in U.S. studies summarized here (Rudel et al., 2003; Wu et al., 2007; Stapleton et al., 2005; Harrad et al., 2008b) and orders of magnitude higher than

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studies abroad. The authors also identified blood samples from NHANES 2001/2002 that were from California and compared them to the entire NHANES 2001/2002 statistical extrapolations for the United States. Their geometric mean for the sum of six congeners (BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154) from samples in California was 62 ng/g lipid weight (lwt), while the total for all of the United States was 38.6 ng/g lwt. They suggest this might have resulted from more stringent standards for flammability in California as compared to other states. The NHANES database is discussed further in Chapter 5.

Hwang et al. (2008) measured seven congeners (BDE 47, BDE 66, BDE 99, BDE 100, BDE 138, BDE 153, and BDE 154) in dust collected from vacuum bags provided by residents in 10 apartments and 1 community hall in Davis, California. They found that the total ranged from 1,780–25,500 ng/g dwt, with a median of 9,240 ng/g dwt. Median and range of the three dominant congeners were (in ng/g dwt) BDE 47—2,920 and 655–8,830; BDE 99—4,430 and 807–11,700; and BDE 100—793 and 165–2,000.

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.[1] 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 BDE 77, BDE 138, BDE 155, BDE 183, and BDE 203 (BDE 203 and BDE 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.

Wilford et al. (2005) studied house dust concentrations from 68 of 74 homes in Canada that previously had been studied for air concentrations of PBDEs (see Section 4.5) in the winter of 2002/2003. They found that the data were log normally distributed, with a geometric mean of 2,000 ng/g, a median of 1,800 ng/g, and an arithmetic mean of 5,500 ng/g. For the arithmetic mean, BDE 209 comprised 1,000 ng/g of the total, or 18%, while for the median, BDE 209 comprised half, at 900 ng/g. This suggests that BDE 209 dominates the highest concentrations found. After BDE 209 for the mean, the highest concentrations were BDE 99 at 1,800 ng/g and BDE 47 at 1,100 ng/g, with other concentrations less than 500 ng/g.

[1]See http://www.arb.ca.gov/toxics/pbde.htm.

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Sampling of house 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 heptaBDE 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 unknown if these mean values are significantly different. The highest value from Spain was from a dust sample that had been sucked out from the inside of a computer. Urban house dust 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 a ratio of 0.6, suggesting these samples were dominated by this commercial mixture.

Harrad et al. (2006) measured BDEs in house dust at eight homes sampled in 2005 in the United Kingdom. They reported an average of 215 ng/g total (maximum of 625 ng/g total), which included BDE 28, BDE 47, BDE 49, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 154 (BDE 209 not measured). The ratio of BDE 47 to BDE 99 was about 0.5. The study that Harrad et al. (2008b) later published on different locations around the world (United Kingdom, United States, New Zealand, and Canada) included these earlier data.

Harrad et al. (2008a) later measured dust samples taken from cars, homes, and offices in locations in southern United Kingdom (West Midlands area), and found the highest BDE 209 measurements in dust samples that have been found as of the date of this report, including findings of 2,200,000 ng/g in a home and 2,600,000 ng/g dwt in a car (further discussion on the car data provided in Section 4.4.2 below). The average total BDE concentration (sum of BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209) of 30 dust samples taken in homes was 260,000, which was the same concentration provided as the average for BDE 209. This was because the concentrations of the other congeners were all less than 100 ng/g dwt (sum of average congener concentrations was about 150 ng/g dwt), and, hence, BDE 209 overwhelmed the average. The average of 18 office samples was 31,000 ng/g dwt total, with BDE 209 explaining 30,000 ng/g dwt of that average. They went back to sample one

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of the two homes with high BDE 209, 1,400,000 ng/g dwt, and found a similar concentration: 900,000 ng/g dwt

Karlsson et al. (2007) report on measurements of PBDEs in air, house dust, and blood from five households in Sweden. 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 BDE 99 and BDE 47, at 99 ng/g dwt 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 2/29/2004 and 4/11/2004. Individual congeners measured included BDEs 28, BDE 47, BDE 100, BDE 99, BDE 85, BDE 154, BDE 153, and BDE 183 (BDE 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. BDE 47 and BDE 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. House 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 those 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, 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 U.S. 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 have higher BDE 209 concentrations than the measurements summarized in Pless-Mulloli et al. (2006).

In summary, the most common indoor congeners include those associated with the penta formulations, BDE 47 and BDE 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

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10,000 ng/g dwt. Concentrations of BDE 209 have been found in the hundreds of thousands to over two million of ng/g dwt in the United Kingdom (Harrad et al., 2008a) and in the hundreds of thousands in one study in the U.S. (from Allen et al., 2008). Most authors reported that the results suggested the presence of the penta formulation, based on the ratio of the two congeners, BDE 47 and BDE 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 BDE 47 and BDE 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, and as noted, some well above that in the hundreds of thousands to millions of ng/g dwt. Dust concentrations of BDEs appear much higher in U.S. dust, well into the range of thousands of ng/g dwt for total concentrations, compared with limited measurements in Europe, which appear to be limited to the hundreds of ng/g dwt. Exceptions to this generalization include data from the United Kingdom showing substantial concentrations of BDE 209, and one study from Kuwait, which had a high concentration of total BDEs just above 100 ng/g dwt.

4.4.2. Car and Airplane Dust Concentrations

High concentrations of BDE congeners in cars and airplanes recently have been found. The authors of the studies have noted that the concentrations found are higher than those typically found in homes. This becomes important even though the time spent in these vehicles is small relative to the time spent in a home.

The Harrad et al. (2008a) West Midland, United Kingdom study mentioned in the previous section found the highest BDE 209 measurements in dust samples that have been found as of the date of this report, including a finding of 2,600,000 ng/g dwt in a car. The average concentration for 20 car samples was 410,000 ng/g dwt (std. deviation 770,000; personal communication with Harrad). Cars had statistically significantly higher concentrations of lower BDE congeners, tri-hexaBDEs, in the hundreds of ng/g for BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154, as compared to offices and homes. They went back to sample the high car finding of BDE 209, 2,600,000 ng/g dwt, and found a similar concentration: 2,200,000 ng/g dwt.

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Lagalante et al. (2009) sampled dust within 60 cars in the United States and, like Harrad et al. (2008a), found a dominance of BDE 209. The cars were manufactured mostly in the 2000s; one car each was manufactured in 1990, 1998, and 1999. The median BDE 209 level was 48,100 ng/g dwt, and it comprised 95% of total BDEs measured. Median concentrations of the other congeners measured were BDE 28—118 ng/g dwt, BDE 47—880 ng/g dwt, BDE 99—1,130 ng/g dwt, BDE 100—211 ng/g dwt, BDE 153—163 ng/g dwt, and BDE 183—73 ng/g dwt. All of these cars were from car dealerships and were cleaned. Significant relationships between concentrations and car descriptors were found for BDE 209 and these descriptors: vehicle model year, vehicle manufacturer, and country of origin; there was no relationship by dealership, suggesting that cleaning procedures might not influence BDE levels in the cars. Even though there was significant differences between concentrations by manufacturers, up to 3 orders of magnitude difference were seen within some of the manufacturers, suggesting that there were variables explaining the concentrations not determined, or else there simply was variability within the cars.

Christiansson et al. (2008) studied levels of dust in planes making intercontinental flights out of Sweden to Japan, China, Poland, Canada, United States, United Kingdom, and to India. Nine individuals provided the dust samples on their intercontinental trips, collecting the dust mostly from ventilation ducts in the airplane restrooms (no further detail provided). These nine individuals also provided blood samples before and after their trips to determine whether exposures may have resulted in an elevation of PBDEs. Two additional individuals provided dust samples from one of their frequent trips within Sweden, and one blood sample each. All the results in this study were provided in units of pmol/g dwt for dust and pmol/g lwt for blood. To convert to the units used in this report, the results were multiplied by the molecular weight and 0.001 (to convert pmol to pg, multiply pmol × pg/pmol, or essentially, g/mol, which is the molecular weight; and then from pg to ng, multiply by 0.001). The median and maximum of the congeners in dust were (in ng/g dwt) BDE 28—140 and 2,800; BDE 47—3,500 and 228,000; BDE 99—3,800 and 291,000; BDE 100—900 and 179,000; BDE 153—502 and 23,000; BDE 154—370 and 63,000; BDE 183—490 and 193,000; and BDE 209—17,000 and 190,000. The authors display a table and discuss how these concentrations are higher than those found in house dust from other countries, including the United States, citing three U.S. studies (all discussed above). They note that the travelers showed blood levels similar to Swedish general

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population, but that levels of most of the congeners increased upon return from their intercontinental trip. BDE 209 had the highest mean and median concentration of all congeners, at 3.4 and 2.6 ng/g lwt, respectively. The second highest was BDE 47 at 1.8 and 1.2 ng/g lwt, respectively. As discussed in Chapter 5, NHANES pooled blood samples showed a level of 2.0 ng/g lwt of BDE 209, but that the median for individual samples for BDE 47 was 20.5 ng/g lwt.

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 others. 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, composed 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 were 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

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Table 4-2. Outdoor and indoor congener-specific air concentrations of PBDEs in the United States (units in pg/m^3)

Congener	Concentration (pg/m^3)	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	Geometric mean of 20 homes, 3 locations/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)

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Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
	80, 50	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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	Geometric mean of 20 homes, 3 locations/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 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)
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)

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Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
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), 2002/2003	Hoh and Hites (2005)
	17.4	Chicago (n = 28), 2002/2003	Hoh and Hites (2005)
	7.0	Indiana (n = 38), 2002/2003	Hoh and Hites (2005)
	9.2	Arkansas (n = 30), 2002/2003	Hoh and Hites (2005)
	6.9	Louisiana (n = 26), 2002/2003	Hoh and Hites (2005)
	5.0	3 Rural/remote sites in MI, NY (n = 36), 1997−1999	Strandberg et al. (2001)
	33	Chicago, n = 12, 1997−1999	Strandberg et al. (2001)

ED_002435_00009767-00193

Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
	175 (671)	Lewes, Del Marva, MD (n = 95) geom. mean (max), vapor phase only	Goel et al. (2006)
	9.7 (26)	Horn Point, Del Marva, MD (n = 98) geom. mean (max), vapor phase only	Goel et al. (2006)
	17 (52)	Dover, Del Marva, MD (n = 47) geom. mean (max), vapor 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; average = 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	Geometric mean of 20 homes, 3 locations/home in Boston, MA area; "personal", bedroom, and living room	Allen et al. (2007)
	740, 420	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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)
	449−2,860	Range, n = 6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; average = 1,764	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	Geometric mean of 20 homes, 3 locations/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)

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Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
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	Geometric mean of 20 homes, 3 locations/home in Boston, MA area; "personal", bedroom, and living room	Allen et al. (2007)
	110, 90	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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 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)
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)

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Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
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)
	ND, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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	Geometric mean of 20 homes, 3 locations/home in Boston, MA area; "personal", bedroom, and living room	Allen et al. (2007)
	ND, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
99	5.1	Michigan (n = 35), 2002/2003	Hoh and Hites (2005)
	7.4	Chicago (n = 28), 2002/2003	Hoh and Hites (2005)
	5.1	Indiana (n = 38), 2002/2003	Hoh and Hites (2005)

ED_002435_00009767-00196

Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
	5.4	Arkansas (n = 30), 2002/2003	Hoh and Hites (2005)
	3.0	Louisiana (n = 26), 2002/2003	Hoh and Hites (2005)
	3.4	3 Rural/remote sites in MI, NY (n = 36), 1997−1999	Strandberg et al. (2001)
	16	Chicago, n = 12, 1997−1999	Strandberg et al. (2001)
	26 (178)	Lewes, Del Marva, MD (n = 95) geometric mean (maximum), vapor phase only	Goel et al. (2006)
	5.3 (26)	Horn Point, Del Marva, MD (n = 98) geometric mean (maximum), vapor phase only	Goel et al. (2006)
	7.7 (17)	Dover, Del Marva, MD (n = 47) geometric mean (maximum), vapor 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)
	402−3,210	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	Geometric mean of 20 homes, 3 locations/home in Boston, MA area; "personal", bedroom, and living room	Allen et al., 2007
	210, 160	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
100	1.1	Michigan (n = 35), 2002/2003	Hoh and Hites (2005)
	1.8	Chicago (n = 28), 2002/2003	Hoh and Hites (2005)
	1.0	Indiana (n = 38), 2002/2003	Hoh and Hites (2005)

ED_002435_00009767-00197

Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
	1.1	Arkansas (n = 30), 2002/2003	Hoh and Hites (2005)
	0.7	Louisiana (n = 26), 2002/2003	Hoh and Hites (2005)
	0.5	3 Rural/remote sites in MI, NY (n = 36), 1997−1999	Strandberg et al. (2001)
	2.0	Chicago, n = 12, 1997−1999	Strandberg et al. (2001)
	17 (73)	Lewes, Del Marva, MD (n = 95) geometric mean (maximum), vapor phase only	Goel et al. (2006)
	5.4 (5.4)	Horn Point, Del Marva, MD (n = 98) geometric mean (maximum), vapor phase only	Goel et al. (2006)
	5.3 (5.3)	Dover, Del Marva, MD (n = 47) geometric mean (maximum), vapor 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	Geometric mean of 20 homes, 3 locations/home in Boston, MA area; "personal," bedroom, and living room	Allen et al. (2007)
	60, 50	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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)

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Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
	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)
	ND, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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)

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Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
	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)
	ND, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
153	0.20	3 Rural/remote sites in MI, NY (n = 36), 1997–1999	Strandberg et al. (2001)
	0.53	Chicago, n = 12, 1997–1999	Strandberg et al. (2001)
	2.0	Outdoors at UC Davis, 2004; average of 2 days of filter/PUF and filter/XAD samplers	CARB (2005)
	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; average = 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	Geometric mean of 20 homes, 3 locations/home in Boston, MA area; "personal", bedroom, and living room	Allen et al. (2007)
	60, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)

ED_002435_00009767-00200

Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
154	0.12	3 Rural/remote sites in MI, NY (n = 36), 1997−1999	Strandberg et al. (2001)
	041	Chicago, n = 12, 1997−1999	Strandberg et al. (2001)
	ND	For Lewes, Horn Point, Del Marva, MD (n = 240), vapor 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)
	1,230−5,260	Range, n = 6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; average = 3,455	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	Geometric mean of 20 homes, 3 locations/home in Boston, MA area; "personal," bedroom, and living room	Allen et al. (2007)
	ND, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
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)

ED_002435_00009767-00201

Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
	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 electronics recycling facility in CA	CARB (2005)
	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)
	5,610−36,700	Range, n = 6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; average = 23,813	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)
	ND, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
190	<0.06 (DL)	3 Rural/remote sites in MI, NY (n = 36), 1997−1999	Strandberg et al. (2001)

ED_002435_00009767-00202

Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
	<0.06 (DL)	Chicago, n = 12, 1997−1999	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)
	288−1,300	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)
OctaBDE			
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)
	ND, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
NonaBDE			
207	ND, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)
DecaBDE			
209	1.4	Michigan (n = 35), 2002/2003	Hoh and Hites (2005)
	60.1	Chicago (n = 28), 2002/2003	Hoh and Hites (2005)
	2.2	Indiana (n = 38), 2002/2003	Hoh and Hites (2005)
	9.0	Arkansas (n = 30), 2002/2003	Hoh and Hites (2005)
	2.6	Louisiana (n = 26), 2002/2003	Hoh and Hites (2005)

ED_002435_00009767-00203

Table 4-2. Outdoor and indoor congener specific air concentrations of PBDEs in the United States (units in pg/m^3) (continued)

Congener	Concentration (pg/m^3)	Comment	Citation
	<0.10 (DL)	3 Rural/remote sites in MI, NY (n = 36), 1997−1999	Strandberg et al. (2001)
	0.30	Chicago, n = 12, 1997−1999	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)
	140−11,400	Range, n = 12 (3 days, 4 samplers) outdoors surrounding electronics recycling facility in CA; mean = 2,764	CARB (2005)
	79,700−833,000	Range, n = 6 (3 days, 2 samplers) indoors in electronics recycling facility in CA; average = 423	CARB (2005)
	123−1,940	Range, n = 9 (3 days, 3 samplers) outdoors in auto shredder facility in CA; average = 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	Geometric mean of 20 homes, 3 locations/home in Boston, MA area; "personal," bedroom, and living room	Allen et al. (2007)
	ND, ND	Mean, median indoor concentrations from 12 homes in Albany, NY	Johnson-Restrepo and Kannan (2009)

site/statewide averages are available from the Web site. Twelve congeners were measured, including BDE 17, BDE 28, BDE 47, BDE 65, BDE 77, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209. BDE 47 and BDE 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). This overall research program was also published in the open literature in Cahill et al. (2007). 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 2 consecutive days, 3/17/2004 and 3/18/2004, providing a total of eight 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. BDE 47 and BDE 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 five locations from Lake Michigan to the Gulf of Mexico, between August 2002 and December 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 were only available for BDE 47, BDE 99, BDE 100, and BDE 209 (these four contributed about 80% of the total PBDE). The highest congener individually was BDE 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 including 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 3 years (1997, 1998, and 1999) resulted in a total of 48 samples. Congeners included BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 190, and BDE 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 three rural/remote sites but detected at levels only at 0.3 pg/m^3 in Chicago. This is in stark contrast to the monitoring of Hoh and Hites (2005), which showed an average of 60 pg BDE 209/m^3 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

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the time when the penta and octa formulations were taken off the U.S. 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) note much higher BDE 209 in their data and attribute 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 BDE 99 and BDE 100, and only about 30% in vapor phase for BDE 153 and BDE 154.

The 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 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/m^3, 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/m^3, respectively, and the mean concentration at the "remote" site of Eagle Harbor was 5.8 pg/m^3. The authors also studied the time trends of BDE 47, BDE 99, and BDE 209 from data starting in 2003 and observed that BDE 47 and BDE 99 were declining rapidly, but that BDE 209 was not declining at any of the five sites.

Three sites within the Chesapeake 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 vapor phases for BDE 47, BDE 99, BDE 100, and BDE 154. BDEs were detected in 75% of the samples, but detection frequency was highest at Lewes, DE 98%, compared to Dover, DE 77%, and Horn Point, MD 52%. Vapor phase concentrations were presented and were highest at Lewes, with geometric mean concentrations over the 3-year period of 174 pg/m^3, followed by Dover and Horn Point at 19 and 10 pg/m^3, 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 BDE 99 and BDE 100, in Horn Point and Dover (10–20 pg/m^3 for BDE 47 compared to 5–8 pg/m^3 for BDE 99 and BDE 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 BDE 99 and BDE 100). BDE 154 was essentially not detected in either vapor 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 four samplers, including two upwind and two 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 offgassing from previously deposited BDEs from the nearby source. The highest measurement was for BDE 209, averaging over 2,400 pg/m^3 for four 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 pg/m^3, 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). Guy Fawkes Day (also referred to as "Bonfire Festival" in the article), which is centered on the use of fireworks and the lighting of bonfires, occurs every year on November 5. Three samplers were set up in the garden of a home in a

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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 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 BDE 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 BDE 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 BDE 66, BDE 85, BDE 100, BDE 181, and BDE 190. The authors suggest that 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 BDE 154, which is different from the more typical predominance of BDE 47 and BDE 99 (as seen in background air).

Gouin et al. (2006) studied particle bound air transport of BDE 209 in Southern Ontario by air sampling with a PS-1 sampler during the period between 1/23/2002 and 6/5/2002. While the focus was on BDE 209, BDE 47, BDE 99, and BDE 100 were also measured in 28 samples taken roughly weekly during this time frame. Concentrations of BDE 209, exclusively found in the particle phase, ranged from nondetect (detection limit = 3 pg/m^3) to 105 pg/m^3, with a mean of 19 pg/m^3. BDE 99 and BDE 100 were rarely detected, while BDE 47 was detected in nearly every sample with concentrations of mostly less than 5 pg/m^3, dominated by the vapor phase (as measured in the PUF portion of the sampler) in the warmer time frames, but with higher concentrations in the particle phase (as measured by the amounts on the glass fiber filter) from January to about the end of March. Blanchard et al. (2004) also reported on the concentrations of BDE 209 in Canadian air. They found air concentrations averaging 1.8 pg/m^3 of BDE 209 (predominantly vapor phase) for 72-hour samples collected every month during the year.

Wilford et al. (2008) sampled air at a semirural site in northwest England in April to May of 2004. Their analysis focused almost exclusively on characterizing the particle phase of BDEs, with a particular emphasis on BDE 209 and other higher-brominated congeners. They did measure for the vapor phase using a 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

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was dominated by tri- to hexa BDE congeners, and that these congeners were found at vapor phase concentrations at least 2 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 concentrations of total BDEs in the particle phase over 28 samples were 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 BDE 183, BDE 196, BDE 197, BDE 206, BDE 207, BDE 208, and BDE 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.

Harrad and Hunter (2006) used PUF disk passive samplers to measure the concentration of six BDE congeners—BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154—in the West Midlands area of the United Kingdom, which includes the city of Birmingham and neighboring rural locations. They took air samples at 10 sites over the course of 1 year between 2003 and 2004. The found average total concentrations (sum of the 6 congeners) at these 10 sites to range from 2.84 to 23.3 pg/m^3, with the highest concentration associated with the city of Birmingham. They found ratios of BDE 47 to BDE 99 to range narrowly from 2.95 to 3.57. They suggested that by using PUF samplers, they might be capturing mostly the vapor phase of the BDE congeners in air.

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 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 analyzed 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 of 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 9/21/2004 and 11/16/2004. Rural and urban sites were sampled in China (32 samples), Japan (20), South Korea (15), and Singapore (10). Congeners measured include BDE 17, BDE 28, BDE 32, BDE 47, BDE 49, BDE 75, BDE 99, and BDE 100. Total BDEs ranged from 0.1 to tens of pg/m^3, which the authors note is

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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 Kingdom, 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 all four locations; BDE 99: 50 in China, <10 otherwise; BDE 100: 5.5 in China, <2.3 otherwise. It was stated that BDE 47 and BDE 99 dominated the profile, contributing around 75% to the overall 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 in 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; and 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, BDE 47 and BDE 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 6/15/2004–6/30/2004. High volume polyurethane foam/glass fiber filter PS-1 samplers were used, and detection limits were 0.14–0.58 pg/m^3 for BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, and BDE 183, and 14.3 pg/m^3 for BDE 209. BDE 209 dominated the profile, averaging 4,200, 750, 264, and 478 pg/m^3 for the two industrial, urban, and background settings while the sum of the other PBDEs correspondingly averaged 3,673, 230, 89, and 105 pg/m^3. 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 (which is used in computer

circuitry and plastic housing) 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 BDE 47 and BDE 99 comprising greater than 50% of the total.

In summary, the major trends seen from the air monitoring studies include the following: (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 BDE 47 and BDE 99, suggesting a penta formulation influence; however, when BDE 209 was measured, it was seen to have concentrations equal or greater than BDE 47 and BDE 99. One study in Chicago had BDE 209 at concentrations six times higher than BDE 47 or BDE 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). (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 Concentrations 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 persistent and bioaccumulative toxics (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

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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. 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. Thus, while the combustion of PBDEs appears to produce other brominated compounds, unlike dioxins, PBDEs are not formed themselves from combustion.

Unfortunately, U.S. researchers have not measured indoor air as much as house dust, or as much as researchers from Canada and abroad. Only three studies have been located that measured indoor air. The most recent involved sampling the air in 12 homes in Albany, NY, between December 2007 and January 2008 (Johnson-Restrepo and Kannan, 2009). They used a low-volume pump placed in the main room, the living room, and collected samples for 12 hours with an air flow of 25 L/min. Analysis of fractions on a glass fiber filter as well as a polyurethane foam plug allowed for collection of both the particle and vapor phases. The mean total concentration of 15 congeners (from BDE 28 to BDE 209) was 1.26 ng/m^3, the median was 0.76 ng/m^3, and the range was 0.21 to 3.98 ng/m^3. Unlike dust samples taken as part of the same study, BDE 209 could not be quantified at the detection limit of 0.1 ng/m^3, and the samples were dominated by BDE 47.

The second was an indoor air study in which dust was sampled in 20 urban residences in the Boston, MA area, 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 were reported in 2006). Congeners include BDE 17, BDE 28/33, BDE 47, BDE 49, BDE 66, BDE 85/155, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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

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samplers when returning in the evening and turned them off when leaving for work. The sums 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 third U.S. study found for indoor air measurement of PBDEs was conducted by CARB (2005). This study was also published in the open literature in Cahill et al. (2007). 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 nonaBDEs were not measured, however, for most of the samples because there was analytical interference. The sampling for the indoor training facility included sampling when the computers were *on* versus *off* and, also, it included carpet dust samples. The dominant congener found in the air was BDE 47, followed by BDE 99, BDE 100, and BDE 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/m^3 when the computers were *off* to concentrations from 56–74 pg/m^3 when they were *on* in one set of tests and from 18, when *off*, to 47–56 pg/m^3, when *on*, in the second test. The largest absolute concentration difference was for BDE 47, which was 213 and 112 pg/m^3 in the two tests (starting from concentrations in the 800-pg/m^3 range in the *off* condition and then increasing by 213 and 112 pg/m^3 when the computers were turned *on*), followed by BDE 99, BDE 100, and then BDE 209. The average concentrations of total BDEs *on* (three measurements) averaged with *off* (one measurement) on the 2 days of measurement were 1,550 and 2,010 pg/m^3, with BDE 209 averaging 50 and 65 pg/m^3 for the 2 days.

A second site studied by CARB was an electronics recycling facility. Outdoor measurements from two samplers in front and two in back suggested concentrations in the range of 2,000–3,000 pg/m^3, dominated by BDE 209. Generally, sampling indoors showed

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substantially higher BDE concentrations compared to outdoors at this facility. A total of six samples were obtained from two samplers over 3 consecutive days in 2004. Concentrations ranged from 316,000 to 833,000 pg/m^3. The overwhelmingly dominant congener was BDE 209, ranging from 79,000 to 833,000 pg/m^3, with an average over 423,000 pg/m^3. The next highest congeners were BDE 183, averaging 23,813 pg/m^3, BDE 153 averaging over 5,600 pg/m^3, BDE 154 averaging over 3,400 pg/m^3, and then BDE 99, BDE 47, and BDE 49, all at about 1,770 pg/m^3. 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/m^3 might represent nonoccupational 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 November/December of 2001 and then again in March of 2003. Congeners included BDE 17, BDE 28/33, BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 154. Total concentrations outdoors were 39 and 48 pg/m^3 (two locations, three samples) and indoors were 410, 358, 490, 2,088, 381, and 76 pg/m^3 (six locations, seven samples), leading to an average ratio difference of 15 between indoor and outdoor samples. BDE 47 represented about 46% of the sample, followed by BDE 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%, BDE 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 2002/2003 in Ottowa, Canada. Indoor air concentrations of PBDEs were log-normally distributed, with a geometric mean of 120 pg/m^3 (high of

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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 BDE 17, BDE 28, BDE 47, BDE 66, BDE 71, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 154. The highest mean concentration was BDE 47 at 160 pg/m^3, followed by BDE 99 at 42 pg/m^3, and BDE 28 at 24 pg/m^3. Wilford et al. (2004) stated that the indoor passive samplers are sampling mainly the vapor phase, with only a small contribution from particulates. The study found that the technical formulations tend to be enriched with heavier congeners, BDE 99 and BDE 100, while the indoor air was dominated by the lighter, more volatile congeners, BDE 47 and BDE 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. 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 1 year, for sampling events that took between 4 and 6 weeks. Congeners sampled included BDE 28, BDE 47, BDE 49, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 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 2/29/2004 and 4/11/2004 (Gevao et al., 2005). Individual congener concentrations included BDE 28, BDE 47, BDE 85, BDE 99, BDE 100, BDE 154, BDE 153, and BDE 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 congener data on BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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; BDE 153 and BDE 154 were comparable in both environments, but BDE 47, BDE 99, and BDE 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.

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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 BDE 28, BDE 47, BDE 49, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 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 noncar 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 BDE 47 and BDE 99.

Karlsson et al. (2007) reported on measurements of PBDEs in indoor air, house dust, 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 one of four samples, at 257 pg/m^3 (DL = 173 pg/m^3). BDE 47 was found in three of four samples, ranging from 126 to 171 pg/m^3. The only other two congeners quantified in air were BDE 28 and BDE 66, and they were found in seven of eight 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 that 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 BDE 47 and BDE 99. The maximum found was over an order of magnitude higher than this median at 2,644 pg/m^3.

In summary, only two studies could be located that measured indoor air concentrations of BDEs in the United States in residences rather than in occupational settings. One study in Albany, NY, showed a mean total concentration of 1,260 pg/m^3 and the other was of different rooms within urban residences in the Boston, MA, area, where the mean total household concentration was in the range of 200–500 pg/m^3. 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

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industrial/occupational concentrations were substantially higher, with measurements in the hundreds of thousands of pg/m^3. In United States, Canada, 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 study where BDE 209 was measured in the indoor environment, it was the second highest concentration, followed by BDE 99. In a study of PBDE concentrations inside cars, BDE 209 dominated 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 U.S. 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 BDE 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 (wwt) 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 wwt and lipid bases. While the authors reported results are provided below, wwt or lipid-based (lwt) concentrations are concurrently provided in parentheses 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., 2008). They represented salmon from three regions: two farms in eastern Maine, three farms in eastern Canada, and one farm in Norway. Samples were composited so that the Maine sample results were displayed for the two farms.

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Table 4-3. Congener-specific concentrations of PBDEs for fish caught in the United States (units in ng/g wwt, or lwt, in parentheses if available)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
MonoBDE			
1	0.007, 0.006 0.007, 0.006	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
2	0.005, 0.004 0.005, 0.004	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
3	0.004, 0.004 0.004, 0.004	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
DiBDE			
7	0.001, 0.0002 0.001, 0.001	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
8/11	0.0002, 0.0002 0.0004, 0.0003	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
10	0.0002, 0.0002 0.0004, 0.0003	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
12/13	0.0003, 0.0002 0.0003, 0.0002	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
15	0.006, 0.003 0.002, 0.002	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
TriBDE			
17	0.011	n = 24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al. (2006a)
	0.002	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%	Schecter et al. (2009b)
	0.10 (0.77 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)
	0.010, 0.003 0.003, 0.003	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
25	0.007, 0.002 0.001, 0.001	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
28	0.01	Average of 18 farm and wild-caught salmon from Maine, Alaska, Canada; eyeball estimate from graph	Shaw et al. (2008)
	0.01	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%	Schecter et al. (2009b)
	0.026	n = 24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al. (2006a)
	1.67 (10.21 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)

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Table 4-3. Congener specific concentrations of PBDEs for fish caught in the United States (units in ng/g wwt, or lwt, in parentheses if available) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	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)
28/33	0.036, 0.014 0.006, 0.006	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
30	0.0008, 0.0007 0.0008, 0.0006	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
32	0.0007, 0.0006 0.0007, 0.0005	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
35	0.005, 0.003 0.003, 0.0006	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
37	0.001, 0.001 0.0005, 0.0004	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
TetraBDE			
47	0.60	n = 24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al. (2006a)
	0.20	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%	Schecter et al. (2009b)
	3.6	n = 17 locations in SF Estuary; bivalve (clam, oyster, mussel) average	Oros et al. (2005)
	20.6, 10.9	Means from 2004 (n=132) and 2005 (n=213) from national study on rivers and streams in the US	Blocksom et al. (2010)
	0.4	Average of 18 farm and wild-caught salmon from Maine, Alaska, Canada; eyeball estimate from graph	Shaw et al. (2008)
	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 and 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 Washington State survey in rivers and lakes	Johnson et al. (2006)
	2.6, 1.9 0.1, 0.1	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
49	4.78 (35.35 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)

Table 4-3. Congener specific concentrations of PBDEs for fish caught in the United States (units in ng/g wwt, or lwt, in parentheses if available) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	0.059	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%	Schecter et al. (2009b)
	ND, 1.3, 33%	n = 60; median, mean, frequency detection in Washington State survey in rivers and lakes	Johnson et al. (2006)
	0.1, 0.03 0.01, 0.01	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
66	1.82 (11.29 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)
	0.005	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; 2 detects	Schecter et al. (2009b)
	0.021	n = 24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al. (2006a)
	ND	Average of 18 farm and wild-caught salmon from Maine, Alaska, Canada; eyeball estimate from graph	Shaw et al. (2008)
	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 Washington State survey in rivers and lakes	Johnson et al. (2006)
	0.03, 0.02 0.002, 0.001	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
71	ND	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)
	ND, <0.5, 3%	n = 63; median, mean, frequency detection in Washington State survey in rivers and lakes	Johnson et al. (2006)
75	0.008, 0.008 0.001, 0.001	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
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. (2006a)
	0.001, 0.0005 0.0008, 0.0005	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
PentaBDE			
85	0.67 (4.31 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)

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Table 4-3. Congener specific concentrations of PBDEs for fish caught in the United States (units in ng/g wwt, or lwt, in parentheses if available) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	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. (2006a)
	0.0006	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; 1 detect at 0.002	Schecter et al. (2009b)
	ND	Average of 18 farm and wild-caught salmon from Maine, Alaska, Canada; eyeball estimate from graph	Shaw et al. (2008)
	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)
	0.03, 0.01 0.003, 0.003	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
88	0.03	Average of 2 farm salmon farm composited from Maine; eyeball estimate from graph	Shaw et al. (2008)
99	22.96 (142.20 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and 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. (2006a)
	0.04	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%	Schecter et al. (2009b)
	0.1	Average of 18 farm and wild-caught salmon from Maine, Alaska, Canada; eyeball estimate from graph	Shaw et al. (2008)
	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)
	7.9, 2.7	Means from 2004 (n=132) and 2005 (n=213) from national study on rivers and streams in the US	Blocksom et al. (2010)
	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 Washington State survey in rivers and lakes	Johnson et al. (2006)
	1.6, 0.9 0.1, 0.1	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
100	17.50 (109.51 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)

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Table 4-3. Congener specific concentrations of PBDEs for fish caught in the United States (units in ng/g wwt, or lwt, in parentheses if available) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	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. (2006a)
	0.04	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%	Schecter et al. (2009b)
	0.08	Average of 18 farm and wild-caught salmon from Maine, Alaska, Canada; eyeball estimate from graph	Shaw et al. (2008)
	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)
	4.8, 2.0	Means from 2004 (n=132) and 2005 (n=213) from national study on rivers and streams in the US	Blocksom et al. (2010)
	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 Washington State survey in rivers and lakes	Johnson et al. (2006)
	0.6, 0.4 0.03, 0.03	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
116	0.004,0.003 0.002, 0.001	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
119	0.009, 0.007 0.0008, 0.0006	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
	0.002	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; 2 detects	Schecter et al. (2009b)
126	0.002, 0.001 0.00080.0006	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
HexaBDE			
138	ND	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and 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. (2006a)
	0.0007	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; 2 detects	Schecter et al. (2009b)

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Table 4-3. Congener specific concentrations of PBDEs for fish caught in the United States (units in ng/g wwt, or lwt, in parentheses if available) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	ND	Average of 18 farm and wild-caught salmon from Maine, Alaska, Canada; eyeball estimate from graph	Shaw et al. (2008)
	<0.001	Average of 88 specimens, 9 species of marine fish, 2 species of dolphin, off Florida coast	Johnson-Restrepo et al. (2005)
	ND, <0.9, 2%	n = 63; median, mean, frequency detection in Washington State survey in rivers and lakes	Johnson et al. (2006)
	0.01, 0.003 0.001, 0.001	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
153	0.021	n = 24; consumer fish including tuna, salmon, shark, trout, catfish, herring;	Schecter et al. (2006a)
	0.006	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%	Schecter et al. (2009b)
	4.43 (27.48 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)
	1.2, 0.3	Means from 2004 (n=132) and 2005 (n=213) from national study on rivers and streams in the US	Blocksom et al. (2010)
	0.1−3.6	Time/spatial trend study for trout/smelt in Great Lakes; range for last sampled, ~2003	Batterman et al. (2007)
	ND	Average of 18 farm and wild-caught salmon from Maine, Alaska, Canada; eyeball estimate from graph	Shaw et al. (2008)
	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 Washington State survey in rivers and lakes	Johnson et al. (2006)
	0.3, 0.2 0.01, 0.01	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
154	0.049	n = 24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al. (2006a)
	0.015	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%	Schecter et al. (2009b)
	7.63 (46.84 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)
	0.9, 0.3	Means from 2004 (n=132) and 2005 (n=213) from national study on rivers and streams in the US	Blocksom et al. (2010)
	ND	Average of 18 farm and wild-caught salmon from Maine, Alaska, Canada; eyeball estimate from graph	Shaw et al. (2008)
	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)

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Table 4-3. Congener specific concentrations of PBDEs for fish caught in the United States (units in ng/g wwt, or lwt, in parentheses if available) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	0.48, 0.88, 49%	n = 63; median, mean, frequency detection in Washington State survey in rivers and lakes	Johnson et al. (2006)
	0.2, 0.2 0.01, 0.01	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
155	0.2, 0.2 0.01, 0.01	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
156	0.003, 0.003 0.002, 0.002	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
166	0.003, 0.003 0.002, 0.002	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
HeptaBDE			
181	0.002, 0.002 0.002, 0.002	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
183	0.002	n = 24; consumer fish including tuna, salmon, shark, trout, catfish, herring	Schecter et al. (2006)
	0.001	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; 1 detect at 0.004	Schecter et al. (2009b)
	0.13 (0.73 lwt)	Time/spatial trend study for lake trout in several Great Lakes; average for 2000 only	Zhu and Hites (2004)
	0.02, 0.01	Means from 2004 (n=132) and 2005 (n=213) from national study on rivers and streams in the US	Blocksom et al. (2010)
	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 Washington State survey in rivers and lakes	Johnson et al. (2006)
	0.02, 0.01 0.003, 0.003	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
184	ND, <0.9, 2%	n = 60; median, mean, frequency detection in Washington 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 and Hites (2004)
	0.003, 0.003 0.003, 0.002	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
191	ND, <0.9, 0%	n = 60; median, mean, frequency detection in Washington State survey in rivers and lakes	Johnson et al. (2006)

Table 4-3. Congener specific concentrations of PBDEs for fish caught in the United States (units in ng/g wwt, or lwt, in parentheses if available) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
196	ND	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; DL < 0.003	Schecter et al. (2009b)
197	0.009, 0.008 0.002, 0.002	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
	0.0002	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; 1 detect at 0.001	Schecter et al. (2009b)
OctaBDE			
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)
	0.003, 0.002 0.003, 0.002	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
NonaBDE			
206	0.0004	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; 1 detect at 0.003	Schecter et al. (2009b)
207	0.007, 0.005 0.005, 0.005	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)
	0.0007	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; 1 detect at 0.005	Schecter et al. (2009b)
DecaBDE			
209	0.092	n = 24; consumer fish including tuna, salmon, shark, trout, catfish, herring; majority ND but one catfish at 1,269	Schecter et al. (2006a)
	0.008	n = 8, consumer fish including salmon, tuna, catfish, canned sardines, frozen fish sticks, lipid = 8.7%; 2 detects at 0.007 and 0.051.	Schecter et al. (2009b)
	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 Washington State survey in rivers and lakes	Johnson et al. (2006)
	0.1, 0.04 0.1, 0.08	Mean, median of wild-caught (n = 33) and farmed salmon (n = 28) in southern Mississippi	Staskal et al. (2008)

Results were provided for nine congeners including BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, and BDE 154. The samples were analyzed with skin on and skin off to see if that made a difference; the results suggested essentially no difference. There was also no correlation between lipid content and wwt concentrations, which is counterintuitive. It was found that total concentrations between the farmed and wild-caught were essentially identical, and the values ranged between 0.42 and 1.37 ng/g total wwt. This is four to five 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. BDE 47 dominated the profiles, contributing 38−58% of the total measured PBDEs, with BDE 99 plus BDE 100 accounting for another 21−26% of the total.

Jacobs et al. (2002) measured PCBs, dichlorodiphenyltrichloroethane (DDT), and PBDEs in farmed and wild European Atlantic salmon, aquaculture feeds, and fish oils used to supplement the feed. Seven British salmon samples, five additional salmon samples (two from Ireland, and three purchased from a Belgian market), eight salmon feeds (from four different Scottish sources), five fish oils, and one vegetable oil were analyzed. Congeners analyzed included BDE 28, BDE 71, BDE 47, BDE 75, BDE 66, BDE 100, BDE 99, BDE 153, and BDE 154. Total BDEs ranged from 1.1−85.2 ng/g 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 eight 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.

Staskal et al. (2008) collected 60 samples of catfish from southern Mississippi, including 28 that were farm-raised and store-bought, and 33 that were wild-caught. Samples were analyzed for 43 congeners including BDE 209. The wild-caught catfish were significantly higher ($p < 0.01$) than the store-bought fish: the median total concentration (sum of 43 congeners) from the wild-caught fish was 10.6 ng/g wwt (1,670.4 ng/g lwt), while the median for the farm-raised catfish was 0.5 ng/g wwt (5.8 ng/g lwt). BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154 were detected in all samples and dominated the concentrations. BDE 209 was detected in 30% of the samples, and the average concentration in samples above the LOD was 0.2 ng/g wwt. BDE 209 was detected in the three catfish nugget samples of the

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study and contributed about 36% to the total concentration in these nuggets. Congener-specific data from this study are provided in Table 4-3.

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. BDE 1, BDE 2, BDE 3, BDE 7, BDE 8, BDE 10, BDE 11, BDE 12, BDE 13, BDE 15, BDE 17, BDE 25, BDE 28, BDE 30, BDE 32, BDE 33, BDE 35, BDE 37, BDE 47, BDE 49, BDE 66, BDE 71, BDE 75, BDE 77, BDE 85, BDE 99, BDE 100, BDE 105, BDE 116, BDE 119, BDE 126, BDE 138, BDE 140, BDE 153, BDE 154, BDE 155, BDE 166, BDE 191, BDE 183, BDE 190, BDE 206, BDE 207, and BDE 208 were found. BDE 209 was tested 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%), BDE 28/33 and BDE 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 four 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 BDE 209, but only BDE 28, BDE 47, BDE 49, BDE 99, BDE 100, BDE 153, and BDE 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,

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and sockeye) at 0.4 ng/g wwt (n = 6). BDE 47 had the highest concentrations, explaining 55–70% of the total, followed by BDE 99 and BDE 100, both of which took turns being second behind BDE 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. Freshwater and Marine Fish Concentrations

Fish from the major river systems of the central US - the Upper Mississippi, Missouri, and Ohio Rivers - were sampled as part of EPA's Environmental Monitoring and Assessment Program for Great River Ecosystems (EMAP-GRE; Blocksom et al., 2010). The overall design was a probability design aimed at characterizing these systems on a geographic basis. A first set of results from this study pertaining to sampling years 2004 and 2005 included overall results for legacy organochlorines including pesticides and PCBs as well as emerging compounds of concern, including PBDEs. Both "large" and "small" fish were sampled and composited per "site" of sampling. The large species represent fish targeted by recreational anglers and could include bottom (catfish, e.g.) as well as column fish (largemouth bass, e.g.). The small fish were meant to be representative of prey for piscivorous wildlife. For purposes here, the large fish PBDE results were of interest. A total of 345 large samples (132 in 2004 and 213 in 2005) were measured for 6 PBDEs, including BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183. Individual congener concentrations were not presented in Blocksom et al (2010), but were provided by personal communication (JM Lazorchak, US EPA, 26 W. Martin Luther King Dr Cincinnati, OH 45268). The mean concentrations of all congeners (calculated assuming ND = 0, DL = 1 ng/g wwt) each year are provided in Table 4.1. Overall, BDE 47 was detected the most frequently (nearly 100% of the time) at the highest average concentration, 20.6 ng/g wwt in 2004 and 10.9 ng/g wwt in 2005. In contrast, BDE 183 was detected less than 1% of the time, and when detected, at concentrations near 1 ng/g wwt. The total concentration (sum of the 6 congeners) averaged 35.4 ng/g wwt in 2004 and 16.2 ng/g wwt in 2005.

Oros et al. (2005) measured 22 BDE congeners but detected only BDE 47, BDE 99, and BDE 100 in bivalves (clams, mussels, and oysters) in composited samples originating from 16 locations, at wwt 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 congener found most

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abundantly was BDE 47; it is found in all samples above its DL and comprising about 50−70% of the total concentration, while BDE 99 and BDE 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 six PBDEs: BDE 47, BDE 66, BDE 99, BDE 100, BDE 153, and BDE 154. These samples are 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%.

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 congeners, including BDE 17, BDE 28, BDE 47, BDE 49, BDE 66, BDE 71, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, BDE 190, and BDE 209. The levels of BDE 209 found in all fish samples were in the undetectable range of 3.6 ng/g lwt, thus it was deemed to be not detected in all samples and not reported. Total PBDEs (sum of BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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 octaBDE product, which was high in BDE 153 and BDE 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 pentaBDE use divided by octaBDE in the mid-1980s was 0.7 (more octa used). However, 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 BDE 47, BDE 99, BDE 100, and BDE 153 and found consistent

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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 wwt 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 wwt. BDE 47 dominated the total, contributing about 70% of total concentration, with BDE 99 and BDE 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), six congeners, including BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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 >21.1 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 BDE 99 and BDE 100, which were of similar magnitude; then BDE 153 and BDE 154 at lower and sometimes nondetect values. BDE 183 was only detected in channel catfish at 0.805 ng/g wwt; BDE 47 was detected at 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 BDE 47, BDE 49, BDE 66, BDE 71, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, BDE 184, BDE 191, and BDE 209. Results for total ranged from not detected 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 BDE 100, BDE 154, BDE 153, and BDE 99, detected 51%, 49%, 40%, and 38% of the time, respectively. 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 the most impacted river, with three samples taken measuring 76, 417, and the survey maximum of 1,059 ng/g wwt. 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

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under 100 pg/L. Johnson et al. (2006) reported on an earlier study done 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 freshwater bodies, with totals at 10 to >300 ng/g lwt (which would equal about 1/10 as much on a fwt basis). The interesting finding from this study was that the predominant congener was identified as BDE 30 in all three locations—a congener that was not looked for in any other fish study. The second most predominant congeners were the familiar BDE 47 and BDE 99.

Ashley et al. (2006) analyzed samples of eel and sediment from the Delaware River (sediment samples described in Section 4.2). 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 biotal 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 congener 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 BDE 47, BDE 49, BDE 99, BDE 100, BDE 153, and BDE 154 in 70 samples of fish from the Roanoke River, the Dan River, and the Hyco River, which are within two large watersheds in Virginia. Overall 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 1,000 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 BDE 99 and BDE 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.

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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 BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, BDE 203, and BDE 209. The overall mean concentrations 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 teleost 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 nondetects. Johnson-Restrepo et al. (2005) also did a temporal study, evaluating nine samples of dolphin blubber collected between 1991 and 1996, with six analogous samples collected between 2001 and 2004. The authors similarly 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. Mean total PBDEs in the dolphins rose from 363 to 1,190 ng/g lwt, and mean 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, large scale 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 six sucker fish samples were taken in 2001 and 2000, respectively. Total PBDE concentrations in whitefish , increased by a factor of 11.8 at one location over the period from 1992 and 2000: they went from 6.1 ng/g wwt (average) in 1992 to 19.1 ng/g wwt in 1994 and 1995 to 71.8 ng/g wwt in 2000. At a second location, total PBDE concentrations in whitefish increased by a factor of 6.5: the starting concentration of 4.5 ng/g wwt in 1992 rose to 29.2 ng/g wwt by 2000. At a remote site,

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total PBDE was 0.9 ng/g wwt in the whitefish. The authors attributed the higher concentrations seen at the other two sites to domestic wastewater draining directly into the river. Sucker fish sampled in 2000 had lower concentrations than whitefish at 5.0 ng/g wwt. The sediment congener pattern was somewhat different from the fish. The primary congeners in the sediment and fish were BDE 47, BDE 99, and BDE 100, but BDE 47 was the major congener in sediments (46–63% total), followed by BDE 99 (23–39%), and BDE 100 (6–8%), while for whitefish, BDE 99 was the dominant congener, followed by BDE 47 and BDE 100. This is contrary to other literature, which showed similar dominance of BDE 47 in both sediment and fish.

Vives et al. (2004) collected fish from 11 mountain lakes in Europe and 1 in Greenland. The importance of these lakes 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 composed of 39 individual congeners up to BDE 190 (not including BDE 209), but they only presented results for BDE 28, BDE 33, BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154, while not providing any information on other congeners, including those that they looked for but did not detect. 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 BDE 99, BDE 100, BDE 153, BDE 154, and BDE 28. This pattern 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 six rivers and three estuaries in 2003 in China. BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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 was 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 BDE 99 and BDE 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 BDE 47 at 92 ng/g lwt. In all other locations, BDE 154 was 10 or less ng/g lwt.

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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 are from 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 BDE 183 and BDE 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) reported the results of a comprehensive market basket survey entailing 62 samples that encompassed meat products, dairy products, and fish. BDE 17, BDE 28, BDE 47, BDE 66, BDE 77, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 209 were reported, although the average results reported, which were based on the substitution of 1/2 DL for ND, could be misleading because there was a high frequency of ND (not detected) or NA (not analyzed) for many congeners. 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. The high mean was driven by two samples at 1.27 ng/g wwt 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 ng/g wwt to 0.17 ng/g wwt. The data regarding the more commonly found BDEs at higher concentrations (congeners BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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 these data represent national trends. A large proportion of the food supply is national in scope, such as nonperishable or frozen food items, but others might be locally produced (fruits and vegetables) or perhaps regionally produced (animal food products). Still, this data set represents one of the most comprehensive surveys 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

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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.

Schecter et al. (2009b) provided an update to this data by conducting a similar market basket survey in Texas in 2009. They obtained 310 samples of 31 food types, made composites of 10 for each food type, and analyzed the 31 final samples for 24 BDE congeners as well as hexabromocyclododecane (HBCD). Seven of these 31 samples were of fish, ranging from canned tuna to cod to frozen fish sticks. There was a somewhat similar issue relating to detection limits for this data set as was found for the earlier data set, in that the detection limit for some congeners approached the detected concentrations, particularly for BDE 209. Still, a decline in concentrations might be seen with this data. For example, the average concentrations of these four congeners (which were the congeners having the highest concentrations in 2006) were: BDE 47 at 0.2 ng/g wwt (it was 0.6 ng/g wwt in the earlier survey as noted above), BDE 99 at 0.04 ng/g wwt (compared to 0.17 ng/g wwt), BDE 100 at 0.04 (compared to 0.13 ng/g wwt), and BDE 209 at 0.008 ng/g wt (calculated assuming ND = 0 for 5 nondetects; compared to 0.098 ng/g wwt). As in 2006, BDE 153 and BDE 154 were detected frequently: 23 of 24 in 2006, and 5 of 7 in 2009. Declines were seen in these congeners as well. BDE 153 declined from 0.02 to 0.006 ng/g wwt, and BDE 154 declined from 0.05 to 0.015 ng/g wwt.

Fish, meat, and fowl products were purchased in December 2003 and February 2004 from three different food markets in Sacramento 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 seven PBDEs in samples of chicken, trout, and palm oil. The eight BDEs were BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 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 eight congeners, respectively, totaling 236 ng/g wwt. These results appear anomalous, not only because the concentration were so much higher than

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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 oils 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 three 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 congeners include BDE 15, BDE 17, BDE 28, BDE 47, BDE 66, BDE 71, BDE 75, BDE 77, BDE 85, BDE 99, BDE 100, BDE 119, BDE 126, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 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 contents 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 Arctic char, and not unexpectedly, Arctic 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 BDE 100, BDE 28, BDE 153, BDE 154, and BDE 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 congeners evaluated included BDE 28, BDE 47, BDE 99, BDE 153, and BDE 154. Concentrations in 19 fish samples (4 young yellowtail, 4 mackerel, 2 natural yellowtail, 3 salon, 3 yellow-fin 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. In a "high" group of fish consumers (n = 5), the average breast milk concentration of BDEs was 1,724 pg/g lwt. This was significantly higher than the average concentration of BDEs in a "low" group of fish consumers (n = 3), which was 774 pg/g lwt, suggesting that breast milk concentrations of BDEs were related to fish consumption.

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

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for mussel, trout, and salmon, respectively. BDE 47 dominated the results, comprising over 60% for the three samples, followed by BDE 99 (in mussel and trout) or BDE 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 10 BDEs, not including BDE 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, it covers critical studies on fish from open aquatic settings (rivers and lakes), as well as from retail markets and fish farms. Measurements from open aquatic settings provide information on the understanding of environmental fate for this class of compounds, while sampling from retail markets or fish farms provide more direct information on exposure. Unfortunately, most of this sampling did not include the higher-brominated marker congeners BDE 183 and BDE 209. The very limited sampling for BDE 183 suggested insignificant concentrations, but the market basket surveys by Schecter et al. (2006a; 2009b) 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 wwt 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 Section

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3.8) and body burden measurements (described in Section 5.1), 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

U.S. data on food other than fish are highlighted by four market basket surveys: two in Texas (Schecter et al., 2006a, 2009b), one in California (Luksemburg et al., 2004), and one sampling from several states in the United States (Huwe and Larsen, 2005). 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 or wwt. 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) reported on a sampling of 62 food items, split between samples of meat, dairy, and fish. BDE 17, BDE 28, BDE 47, BDE 66, BDE 77, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 209 were detected, and, as described in Section 4.6.3, there may have been some analytical issues, given the high detection limits and a high frequency of nondetects in this data set. In addition to these limitations, all the food originates from Texas supermarkets only. Section 4.6.3 above describes the results for the 24 fish samples. Of 18 meat samples, total BDE ranged from 0.04 to 1.4 ng/g wwt. BDE 209 was detected in eight meat samples, with concentrations ranging from 0.01 to 0.25 ng/g wwt. Generally, the highest congeners were BDE 47 and BDE 99, with BDE 99 the highest in meat, averaging 0.16 ng/g wwt; BDE 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 nondetect (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. Two samples of infant formula were included in this survey, and these were the only infant formula data that could be found. The wwt concentrations in the samples were 32 and 25 pg/g wwt, respectively, and BDE 209 made up about half of the concentration. No data on solid baby foods could be found in the literature.

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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)	Comment	Reference
TriBDE			
17	0.0008	n = 18 meat (lipid = 26.3%) including pork, chicken, beef; dominated by NDs at DL = 0.7	Schecter et al. (2006a)
	0.0003	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al. (2006a)
	ND	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef, DL < 0.0002	Schecter et al. (2009b)
	ND	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, DL < 0.001	Schecter et al. (2009b)
	<0.00001	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), one detect for apple at 0.00004	Schecter et al. (2009b)
	ND	n = 1 egg (lipid = 10%), DL = 0.0001	Schecter et al. (2009b)
17/25	0.008	9 chicken fat (90% lipid) samples from three production noncontaminated 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	Schecter et al. (2006a)
	0.0008	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al. (2006a)
	0.0006	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; one ND	Schecter et al. (2009b)
	0.0034 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt	Schecter et al. (2009b)
	0.0009	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), two detects including cereal at 0.003	Schecter et al. (2009b)
	ND	n = 1 egg (lipid = 10%), DL = 0.00009	Schecter et al. (2009b)
33	0.017	9 chicken fat (90% lipid) samples from three production noncontaminated 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 and Larsen (2005)
	0.004 (lwt)	Market basket bacon (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)

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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) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	0.003 (lwt)	Market basket chicken fat (n = 22), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.007 (lwt)	Market basket pork fat (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.002 (lwt)	Market basket beef fat (n = 10), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
TetraBDE			
47	2.26	9 chicken fat (90% lipid) samples from 3 production noncontaminated 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. (2006a)
	0.032	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al. (2006a)
	0.016	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef	Schecter et al. (2009b)
	0.134 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt	Schecter et al. (2009b)
	0.007	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit)	Schecter et al. (2009b)
	0.009	n = 1 egg (lipid = 10%)	Schecter et al. (2009b)
	0.18 (lwt)	Market basket hamburger (n = 11), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
	0.23 (lwt)	Market basket bacon (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.81 (lwt)	Market basket chicken fat (n = 22), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	1.07 (lwt)	Market basket pork fat (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.07 (lwt)	Market basket beef fat (n = 10), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
49	ND	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; DL < 0.0004	Schecter et al. (2009b)

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) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	ND	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, DL < 0.0005 (DL for butter at 0.003)	Schecter et al. (2009b)
	0.00003	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), 1 detect for apple at 0.0002	Schecter et al. (2009b)
	ND	n = 1 egg (lipid = 10%), DL = 0.0002	Schecter et al. (2009b)
66	0.001	9 chicken fat (90% lipid) samples from three production noncontaminated sites	Huwe et al. (2003)
	0.018	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe et al. (2003)
	0.0001	n = 18 meat (lipid = 26.3%) including pork, chicken, beef	Schecter et al. (2006a)
	0.0006	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al. (2006a)
	ND	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; DL < 0.0004	Schecter et al. (2009b)
	ND	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, DL < 0.0005 (DL for butter at 0.003)	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit); DL < 0.003	Schecter et al. (2009b)
	ND	n = 1 egg (lipid = 10%), DL = 0.0002	Schecter et al. (2009b)
77	0.0008	n = 18 meat (lipid = 26.3%) including pork, chicken, beef; mostly ND or NA	Schecter et al. (2006a)
	0.0001	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream; all ND or NA	Schecter et al. (2006a)
PentaBDE			
85	0.11	9 chicken fat (90% lipid) samples from three production noncontaminated sites	Huwe et al. (2003)
	0.52	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe et al. (2003)
	0.005	n = 18 meat (lipid = 26.3%) including pork, chicken, beef	Schecter et al. (2006a)
	0.001	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. (2006a)

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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) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	0.022 (lwt)	Market basket hamburger (n = 11), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
	0.023 (lwt)	Market basket bacon (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.047 (lwt)	Market basket chicken fat (n = 22), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.034 (lwt)	Market basket pork fat (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.014 (lwt)	Market basket beef fat (n = 10), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
	0.0004	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; 1 detect; DL < 0.0006	Schecter et al. (2009b)
	0.0007 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, 1 detect; DL <0.001 except butter at 0.005	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.008	Schecter et al. (2009b)
	ND	n = 1 egg (lipid = 10%), DL = 0.0004	Schecter et al. (2009b)
99	3.11	9 chicken fat (90% lipid) samples from three production noncontaminated 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. (2006a)
	0.028	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al. (2006a)
	0.26 (lwt)	Market basket hamburger (n = 11), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
	0.018	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef	Schecter et al. (2009b)
	0.098 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt	Schecter et al. (2009b)
	0.0076	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit)	Schecter et al. (2009b)
	0.019	n = 1 egg (lipid = 10%)	Schecter et al. (2009b)

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) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	0.30 (lwt)	Market basket bacon (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	1.38 (lwt)	Market basket chicken fat (n = 22), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	1.04 (lwt)	Market basket pork fat (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.11 (lwt)	Market basket beef fat (n = 10), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
100	0.45	9 chicken fat (90% lipid) samples from three production noncontaminated sites	Huwe et al. (2003)
	1.47	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe et al. (2003)
	0.023	n = 18 meat (lipid = 26.3%) including pork, chicken, beef	Schecter et al. (2006a)
	0.005	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al. (2006a)
	0.003	n=8 meat (lipid = 13.3%) including pork, chicken, turkey, beef	Schecter et al. (2009b)
	0.016 (lwt)	n=8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt	Schecter et al. (2009b)
	0.0001	n=7 vegetable based (lipid = 50 -100% for oil/peanut butter; 0 for cereals/fruit), one detect for apple at 0.0009 ng/g wwt	Schecter et al. (2009b)
	0.007	n=1 egg (lipid = 10%)	Schecter et al. (2009b)
	0.042 (lwt)	Market basket hamburger (n = 11), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
	0.041 (lwt)	Market basket bacon (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.28 (lwt)	Market basket chicken fat (n = 22), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.18 (lwt)	Market basket pork fat (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.02 (lwt)	Market basket beef fat (n = 10), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
119	ND	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; DL < 0.0006	Schecter et al. (2009b)

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) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	ND	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, DL < 0.001 except for butter at 0.004	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.006	Schecter et al. (2009b)
	ND	n = 1 egg (lipid = 10%), DL = 0.0004	Schecter et al. (2009b)
126	ND	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; DL < 0.0004	Schecter et al. (2009b)
HexaBDE			
138	0.02	9 chicken fat (90% lipid) samples from three production noncontaminated 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. (2006a)
	0.0005	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; 2 detects; DL < 0.0006	Schecter et al. (2009b)
	ND	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, DL < 0.001 except for butter at 0.006	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.006	Schecter et al. (2009b)
	ND	n = 1 egg (lipid = 10%), DL = 0.0004	Schecter et al. (2009b)
140	0.003	9 chicken fat (90% lipid) samples from three production noncontaminated 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 noncontaminated 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., (2006a)
	0.004	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al. (2006a)

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) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	0.004	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef	Schecter et al. (2009b)
	0.0128 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt	Schecter et al. (2009b)
	0.00006	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), one detect for apple at 0.0004 ng/g wwt	Schecter et al. (2009b)
	0.007	n = 1 egg (lipid = 10%)	Schecter et al. (2009b)
	0.10 (lwt)	Market basket hamburger (n = 11), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
	0.078 (lwt)	Market basket bacon (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.22 (lwt)	Market basket chicken fat (n = 22), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.12 (lwt)	Market basket pork fat (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.019 (lwt)	Market basket beef fat (n = 10), 10 locations around the country, sampled in 2004	Huwe and Larsen, (2005)
154	0.18	9 chicken fat (90% lipid) samples from three production noncontaminated 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. (2006a)
	0.002	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al. (2006a)
	0.002	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef	Schecter et al. (2009b)
	0.0048 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.007	Schecter et al. (2009b)
	0.004	n = 1 egg (lipid = 10%), DL = 0.00011	Schecter et al. (2009b)
	0.029 (lwt	)Market basket hamburger (n = 11), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)

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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) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	0.042 (lwt)	Market basket bacon (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.088 (lwt)	Market basket chicken fat (n = 22), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.087 (lwt)	Market basket pork fat (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.011 (lwt)	Market basket beef fat (n = 10), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
156	ND	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; DL < 0.001	Schecter et al. (2009b)
HeptaBDE			
183	0.19	9 chicken fat (90% lipid) samples from three production noncontaminated sites	Huwe et al. (2003)
	0.34	4 chicken fat (90% lipid) samples from a contaminated production site	Huwe et al. (2003)
	0.01	n = 18 meat (lipid = 26.3%) including pork, chicken, beef	Schecter et al. (2006a)
	0.002	n = 15 dairy (lipid = 10.2%) including cheese, milk, infant formula, yogurt, ice cream	Schecter et al. (2006a)
	0.008	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; one ND	Schecter et al. (2009b)
	0.0103 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt; 3 detects; butter DL at 0.008	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.012	Schecter et al. (2009b)
	ND	n = 1 egg (lipid = 10%), DL = 0.0008	Schecter et al. (2009b)
	0.029 (lwt)	Market basket hamburger (n = 11), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)
	0.12 (lwt)	Market basket bacon (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.13 (lwt)	Market basket chicken fat (n = 22), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.084 (lwt)	Market basket pork fat (n = 11), 10 locations around the country, sampled in 2005	Huwe and Larsen (2005)
	0.012 (lwt)	Market basket beef fat (n = 10), 10 locations around the country, sampled in 2004	Huwe and Larsen (2005)

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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) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
196	0.002	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; 2 detects	Schecter et al. (2009b)
	0.002 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, 1 detect butter at 0.014	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.007	Schecter et al. (2009b)
	0.0006	n = 1 egg (lipid = 10%)	Schecter et al. (2009b)
197	0.004	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; 2 detects	Schecter et al. (2009b)
	0.002 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, 1 detect butter at 0.011	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.007	Schecter et al. (2009b)
	ND	n = 1 egg (lipid = 10%), DL = 0.0004	Schecter et al. (2009b)
OctaBDE			
206	0.001	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; 2 detects	Schecter et al. (2009b)
	0.037 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, 2 detects including butter at 0.224	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.013	Schecter et al. (2009b)
	0.005	n = 1 egg (lipid = 10%)	Schecter et al. (2009b)
207	0.003	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; one ND	Schecter et al. (2009b)
	0.059 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, 2 detects including butter at 0.359	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.013	Schecter et al. (2009b)
	0.003	n = 1 egg (lipid = 10%)	Schecter et al. (2009b)
DecaBDE			
209	1.24	9 chicken fat (90% lipid) samples from three production noncontaminated sites	Huwe et al. (2003)

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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) (continued)

Congener	Concentration (ng/g wwt or lwt)	Comment	Reference
	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. (2006a)
	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. (2006a)
	0.017	n = 8 meat (lipid = 13.3%) including pork, chicken, turkey, beef; 5 detects	Schecter et al. (2009b)
	0.954 (lwt)	n = 8 dairy (lipid = 26%) including butter, cheese, milk, ice cream, yogurt, 6 detects including butter at 5.19	Schecter et al. (2009b)
	ND	n = 7 vegetable based (lipid = 50−100% for oil/peanut butter; 0 for cereals/fruit), DL < 0.08	Schecter et al. (2009b)
	0.034	n = 1 egg (lipid = 10%), DL = 0.00011	Schecter et al. (2009b)

Schecter et al. (2009b) provided an update to this data by conducting a similar market basket survey in Texas in 2009. They obtained 310 samples of 31 food types, made composites of 10 for each food type, and analyzed the 31 final samples for 24 BDE congeners as well as hexabromocyclododecane. Twenty-four of these 31 samples were of meat, dairy, and products of vegetable origin. Declines were seen in most food types. Total BDEs (sum of 20 congeners with sufficient detections, including BDE 196, BDE 197, BDE 206, BDE 207, and BDE 209) for meats ranged from 0.008 to 0.2 ng/g wwt, with BDE 209 being found at no higher than 0.04 ng/g wwt. Dairy concentrations of total PBDEs in seven of eight samples were lower than 0.3 ng/g wwt, but was 6.2 ng/g wwt in a sample of butter (90% lipid) with BDE 209 at 5.2 ng/g wwt. As in the previous survey, BDE 47 tended to dominate the concentrations, but with significant contributions also from BDE 99, BDE 100, BDE 153, and BDE 154. BDE 209 was detected in five of eight meat samples ranging from 0.009 to 0.04 ng/g wwt, and in six of eight dairy samples ranging from 0.004 to 5.2 ng/g wwt. It was the highest congener found in eggs at 0.03 ng/g wwt.

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Fish, meat, and fowl products were purchased in December 2003 and February 2004 from three different food markets in Sacramento 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 concentrations 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 BDE 47, BDE 99, and BDE 100, although decaBDE, which is the single congener BDE 209, most often was the highest homologue group found.

A nonstatistical 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 and Larsen, 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 BDE 28/33, BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183. Beef had the lowest average concentrations 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 average concentrations—2.96 ng/g lwt and 2.62 ng/g lwt, respectively. These average concentrations were elevated 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 to BDE 99 ratio of 0.6 found in a penta formulation. This contrasts with a BDE 47 to BDE 99 ratio of sometimes greater than 2, which was identified in several studies in fish as described in Section 4.6. As will be described in the next chapter, humans also have more BDE 47 congeners than BDE 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

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congeners analyzed included the following: BDE 17/25, BDE 33, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 138, BDE 140, BDE 153, BDE 154, BDE 183, and BDE 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 from the noncontaminated production site. The nine samples from these three locations were averaged, and the four samples from the contaminated site, where levels were higher, were 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 production site where BDE levels were higher, a factory producing pentaBDE formulations was located in the same city, which may have explained the higher levels found there. The average total BDE concentration of nine "uncontaminated" chicken samples was 8.3 ng/g lwt, and the average of four "contaminated" chicken samples 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. BDE 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 BDE 47 and BDE 99, with sparse data on BDE 209. As noted below, however, three studies have 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; in the study from Spain, BDE 209 was found at the highest level in several food products; and in the study from Japan, BDE 209 dominated oils and fats. In all three studies, high concentrations of BDE 209 were 10 times or more higher than other congeners when BDE 209 was found.

A recent study commissioned by WWF (formerly World Wildlife 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 tested for but not found, although it cannot be

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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 nonfish 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.

Three recent surveys showed high levels of BDE 209, one from United Kingdom (FSA, 2006), one from Spain (Gomara et al., 2006), and one from Japan (Akutsu et al., 2008). 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 BDE 183 and BDE 209. The meat product concentration of BDE 209 was exceedingly high at 3.64 ng/g wwt. In fact, BDE 209 had the highest concentration of all congeners in all but two 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 BDE 49 and BDE 99, both reported at 0.08 ng/g wwt. Although summary statistics were not supplied, generally BDE 47 and BDE 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, 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 184, BDE 191, BDE 196, and BDE 197, in addition to BDE 183 and BDE 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

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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 wwt, and shellfish at 7 pg/g wwt. Otherwise, BDE 47 and BDE 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). The study from Japan was a total dietary survey, like the UK survey, and entailed measurements of 36 BDE congeners in 14 food groups. Detectable levels of one or more congeners were found in four food groups. Total concentrations (sum of 36 congeners) in these were 1.8 ng/g wwt in "fats and oils" dominated by BDE 209 at 1.8 ng/g lwt, with detectable levels (0.01 ng/g wwt) of BDE 28/33 and BDE 47, "legumes and their products" at a low level of 0.03 ng/g wwt, "fish, shellfish, and their products" at a level of 0.48 ng/g wwt, dominated by BDE 47 at 0.22 ng/g wwt with detections of eight other congeners, and "meat and eggs" at a low level of 0.01 ng/g wwt. Eighteen samples of vegetable oil were resampled to verify the high BDE 209 finding, and ng/g levels of BDE 209 (0.7–2.4 ng/g wwt) were found in seven of them.

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, dairy, 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 that 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: BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209, were more in line with other

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values from around with world. Levels of these eight congeners in chicken 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. 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 BDE 17, BDE 28, BDE 47, BDE 47, BDE 66, BDE 71, BDE 77, BDE 85, BDE 99, BDE 100, BDE 119, BDE 126, BDE 138, BDE 153, BDE 154, and BDE 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 at 3.85 ng/g lwt, margarine (with 5% fish oil) at 3.08 ng/g lwt, liver paste at 1.17 ng/g lwt, and pork liver at 1.08 ng/g lwt. Levels were much higher in fish, with wwt 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 BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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 47, BDE 99, and BDE 100 were higher in meat diets as compared to vegan diets—BDE 153 and BDE 154 were very similar. Meat diet samples averaged 0.07, 0.07, and 0.02 ng/g dwt for BDE 47, BDE 99, and BDE 100, while the analogous concentrations for vegan samples were 0.05, 0.06, and 0.01 ng/g dwt.

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Schecter et al. (2006b) looked at the changes in PBDE levels when beef, lamb, and fish were cooked. While they reported a reduction in the amount of total BDEs in the samples, concentrations before and after cooking were not reported. 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). As 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 BDE 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 with the relationship between these two congeners for fish, where BDE 47 is about twice as high as BDE 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), 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 products. The

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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 that would provide a rigorous estimate of exposure media concentrations to which the general population of the United States is exposed. Most studies are nonstatistical targeted surveys, conducted within a small geographic area, and driven by a limited budget.

3. Key exposure matrices remain sparsely studied, including drinking water, 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 that did obtain samples from several states (Huwe and Larsen, 2005) did not include measurements for BDE 209.

Therefore, it is not possible to derive media concentrations that are statistically representative of general population exposures. Nonetheless, reasonable assumptions can be made for exposure media concentrations to use in making exposure estimates (see 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 BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154. BDE 183 and BDE 209 remain of interest for their representation of the octa and deca formulations, as described above. The U.S. Environmental Protection Agency (EPA) PBDE project plan[2] 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 triBDE 17, tetraBDE 66, and hexaBDE 85 in their measurements; so these final congeners will be added, bringing the total to 14 congeners for which profiles will be derived, if the data permit. 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.

[2]See http://www.epa.gov/oppt/pbde.

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Determining final concentrations to represent the general U.S. 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, do 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, BDE 183 and BDE 209, are preferable to studies that have the limited set of BDEs associated with the penta formulation—BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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 those data have 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 BDE 47, BDE 99, etc, as well as critical BDE 183 and BDE 209, would be preferable to only using the BDE 183 and BDE 209 data from CARB, while averaging the CARB data on other congeners with data from other urban or rural locations in the United States.

5. EPA has not provided an independent review of the quality of data contained in the studies that it has reviewed in this chapter, nor will EPA attempt to independently verify the quality of data in the studies it selects to represent exposure media concentrations. Rather, best judgment is used to select studies that contain high quality data. In addition to the factors cited above, additional considerations that speak to data quality include identification of a laboratory of known high quality, reputation of the authors of the study, and generally, the publication of the study in a respected peer-reviewed journal.

With that as backdrop, Table 4-5 contains the final derived profiles for water, surface soil, house dust, indoor and outdoor air, and categories of food products for use in the exposure calculations in the next chapter. 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. (2009b), 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., 2009b effort). Following now is a brief justification for each of the media:

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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 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. However, the Estuary is not a drinking water source, and issues of turbidity and salinity could influence the results found. Since concentrations were similar in all studies available, this is not expected to present a problem for intake calculations; the more important issue is that drinking water data are not available. Total PBDE concentrations ranged 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. House 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 these data originated from nine different states, it was judged that this might be the most representative data set. The average total PBDEs of 8,275 ng/g dwt for the 14 congeners compares with an average of 5,811 ng total PBDEs/g dwt from 17 homes in the Washington, DC area (Stapleton et al., 2005); the 9,271 ng total PBDE/g dwt average from two 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 13,732; 6,255; and 4,269 ng/g dwt, respectively (Allen et al., 2008).

4. Outdoor air: The CARB data of 84 samples taken in 2004 from seven monitors on 12 dates from locations in the Bay Area and the South Coast were used for the profile of outside air. 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/m^3 as an average of 28 samples in 2002/2003. Strandberg et al. (2001) measured 0.30 pg/m^3 for BDE 209, but their measurements pertain to 1997–1999, which was before the prominent use of the deca formulation. Strandberg et al. (2001) did measure concentrations of 33 and 16 pg/m^3 for BDE 47 and BDE 99, respectively, which compares well with Hoh and Hites (2005), measuring 17 and 7 pg/m^3 for these two congeners, and with the CARB data showing 53 and 51 pg/m^3 for these two congeners, respectively. Therefore, it would appear that the CARB data capture 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.

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Table 4-5. Exposure media concentrations

Exposure media	Congeners														Total	Reference; n
	17	28	47	66	85	99	100	138	153	154	183	197	206	209		
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 dwt	---	---	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)
House dust, ng/g	---	ND	1,857	21	100	2,352	911	181	243	156	60	---	---	2,394	8,275	Sharp & Lunder (2004); n=10
Outdoor air, pg/m^3	2	3	53	---	2	51	13	---	4	4	1	---	---	25	158	CARB (2005); n = 84
Indoor air, pg/m^3	8	27	177	4	3	79	16	---	5	7	---	---	---	121	447	Allen et al. (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	0.002	0.01	0.20	0.005	0.0006	0.04	0.04	0.0007	0.006	0.015	0.001	0.0002	0.0004	0.008	0.32	Schecter et al. (2009b) ; n=7[d]
Beef, ng/g wwt	ND	0.0006	0.009	ND	ND	0.01	0.001	ND	0.003	0.0009	0.0004	ND	ND	0.003	0.028	Schecter et al. (2009b); n=3[d]
Pork, ng/g wwt	ND	0.0007	0.031	ND	0.0006	0.033	0.005	0.0009	0.007	0.003	0.02	0.01	0.0008	0.016	0.13	Schecter et al. (2009b); n=3[d]
Poultry, ng/g wwt	ND	0.0003	0.005	ND	ND	0.007	0.001	ND	0.001	0.001	0.001	ND	0.001	0.004	0.02	Schecter et al. (2009b); n=2[d]
Dairy, ng/g wwt	ND	0.001	0.04	ND	0.0006	0.032	0.005	ND	0.004	0.002	0.001	0.002	0.029	0.675	0.79	Schecter et al. (2009b); n=8[d]
Eggs, ng/g wwt	ND	ND	0.009	ND	ND	0.018	0.007	ND	0.007	0.004	ND	ND	0.005	0.034	0.084	Schecter et al. (2009b); n=1[d]

Table 4-5. Exposure media concentrations (continued)

[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) 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.
[d]All the samples in Schecter et al (2009) are composites of 10 samples, so 10 times the "n" is represented in the averages presented. In calculation of averages, nondetects were set equal to zero due to the range of detection limits presented, including many which exceeded the positive detections.

--- = no data available; ND = data available but not detected

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 three 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/m^3 for the "personal" samples, which were taken near the breathing zone in the bedroom, 392 pg/m^3 in the bedroom, and 366 pg/m^3 in the living room.

6. Shellfish: The only shellfish data available were on clams, oysters, and mussels from the San Francisco Estuary (Oros et al., 2005), so these data were used for the profile. While measurements were made for all 14 congeners, it was stated that nondetects were found for all but BDE 47, BDE 99, and BDE 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. (2009b) were thought to be the most representative for use in this exposure assessment. Seven composite (each including 10 individual samples of the same fish type) samples were taken, ranging from canned tuna to cod to frozen fish sticks. These data showed a small decline from a similarly designed retail market place survey from the same research group and geographic location. For example, the average concentrations of these four congeners (which the highest found in 2006) were: BDE 47 at 0.2 ng/g wwt (it was 0.6 ng/g wwt in the earlier survey), BDE 99 at 0.04 ng/g wwt (compared to 0.17 ng/g wwt), BDE 100 at 0.04 (compared to 0.13 ng/g wwt), and BDE 209 at 0.008 ng/g wt (compared to 0.098 ng/g wwt). The total concentration (sum of key congeners) declined from 1.2 to 0.3 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 4.6, similarly showed concentrations mostly below 10 ng/g wwt and near the value of 0.3 ng/g wwt measured by Schecter et al. (2009b).

8. Beef: The retail market place data from Schecter et al. (2009b) were used for beef congener data. The total concentration of 0.028 ng/g wwt, which was the average of three composite beef samples (hamburger, roast beef, canned chili), was lower than similar retail market basket surveys: Schecter et al (2006a) earlier found an average of 0.13 ng/g wwt in three samples, Huwe and Larsen's (2005) beef samples (n = 11) averaged 0.42 ng/g lwt (roughly 0.08 ng/g wwt) for the five major congeners (BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154), and Luksemburg et al. (2004) beef samples (n = 4) from Northern California averaged 0.15 ng/g wwt total. The data of Schecter et al (2009b) represents the most recent sampling and his study was comprehensive with

regard to congeners measured (24) and food types samples (composites of 10, of 31 different food types).

9. Pork: The retail market place data from Schecter et al. (2009b) were used for pork congener data. The total concentration of 0.14 ng/g wwt, which was the average of 3 composite samples (of bacon, sausages, and ham) was generally lower than comparable studies. For example, the earlier, similarly designed retail market basket survey by Schecter et al (2006a) found an average total of 0.28 ng/g wwt in seven pork samples. Huwe and Larsen (2005), whose pork samples include 11 bacon and 11 pork fat samples, showed a total of 1.59 ng/g lwt for the five major BDE congeners (BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154). Assuming about a 15% lipid weight of pork, this translates to 0.24 ng/g wwt. The data of Schecter et al. (2009b) represents the most recent sampling and his study was comprehensive with regard to congeners measured (24) and food types samples (composites of 10, of 31 different food types).

10. Poultry: The retail market basket data from Schecter et al. (2009b) was used for poultry congener data. The total concentration of 2 composite samples (sliced turkey and sliced chicken breast) of 0.06 ng/g wwt is generally lower than comparable studies. For example, the earlier, similarly designed retail market basket survey by Schecter et al (2006a) found an average total of 0.36 ng/g wwt for three poultry samples. Luksemburg et al. (2004).found an average of 0.41 ng/g wwt for seven poultry samples (four chicken and three turkey samples), and Huwe and Larsen (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 et al. (2004) 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 averages for their studies. Duck was not included in the recent Schecter et al (2009b) study. This high concentration occurred because of the high fat content of duck, listed as 75% lipid in Schecter et al. (2006a). The lipid content of the chicken and turkey samples in the four studies noted were between 2 and 11% lipid, and these wet weight concentrations are felt to better represent consumed poultry. The data of Schecter et al (2009b) represents the most recent sampling and his study was comprehensive with regard to congeners measured (24) and food types samples (composites of 10, of 31 different food types).

11. Dairy: The retail market place data from Schecter et al. (2009b) will be used for dairy congener data. A total of eight composite samples were taken, including three cheeses, whole milk, butter, ice cream, and two yogurts. The highest concentration was found in the butter sample, at 6.2 ng/g wwt. However, this sample was 91% fat, so for this category, lipid average concentrations were determined from the eight samples, and then, exposure was determined based on a whole weight concentration. It was assumed that the milk lipid content was 0.018 (1.8%) and that all other dairy had a lipid content of 0.12 (12%). More detail on the procedure to determine PBDEs via dairy intake is provided in Chapter 5. The lipid weight average total PBDE concentration of total PBDEs (sum of 18 congeners found in sufficient frequency in Schecter et al., 2009b) was 1.3 ng/g lwt, which would translate to about 0.16 ng/g wwt assuming 12% lipid content. By

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comparison, the average over 15 samples in the Schecter et al. (2006) retail market survey (with a mean lipid content of 10%) was similar at 0.11 ng/g wwt. The data of Schecter et al (2009b) represents the most recent sampling and his study was comprehensive with regard to congeners measured (24) and food types samples (composites of 10, of 31 different food types).

12. Eggs: Schecter et al. (2009b) included a composite sample of ten eggs. The total concentration of this sample was 0.09 ng/g lwt, which is the same as their reported concentration of about 0.09 ng/g wwt in the earlier study (Schecter et al, 2006a). The data of Schecter et al (2009b) represent the most recent sampling and his study was comprehensive with regard to congeners measured (24) and food types samples (composites of 10, of 31 different food types).

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5. HUMAN EXPOSURE

5.1. INTRODUCTION

The rise in polybrominated diphenyl ether (PBDEs) in breast milk in the United States throughout the 1990s and into the 2000s, coupled with the finding that milk concentrations in American women exceed those of European women by factors of 10 or more, has served to focus attention on U.S. 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 human 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 pharmacokinetic (PK) model was used to evaluate the impact of breast-feeding on the PBDE body burden of infants. Other exposures of interest, including fetal exposures, childhood exposures, and occupational exposures, are examined. The chapter concludes with discussions on the uncertainties in the procedures used in this chapter, and a series of findings from this examination of U.S. 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. Sections on blood and human 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.

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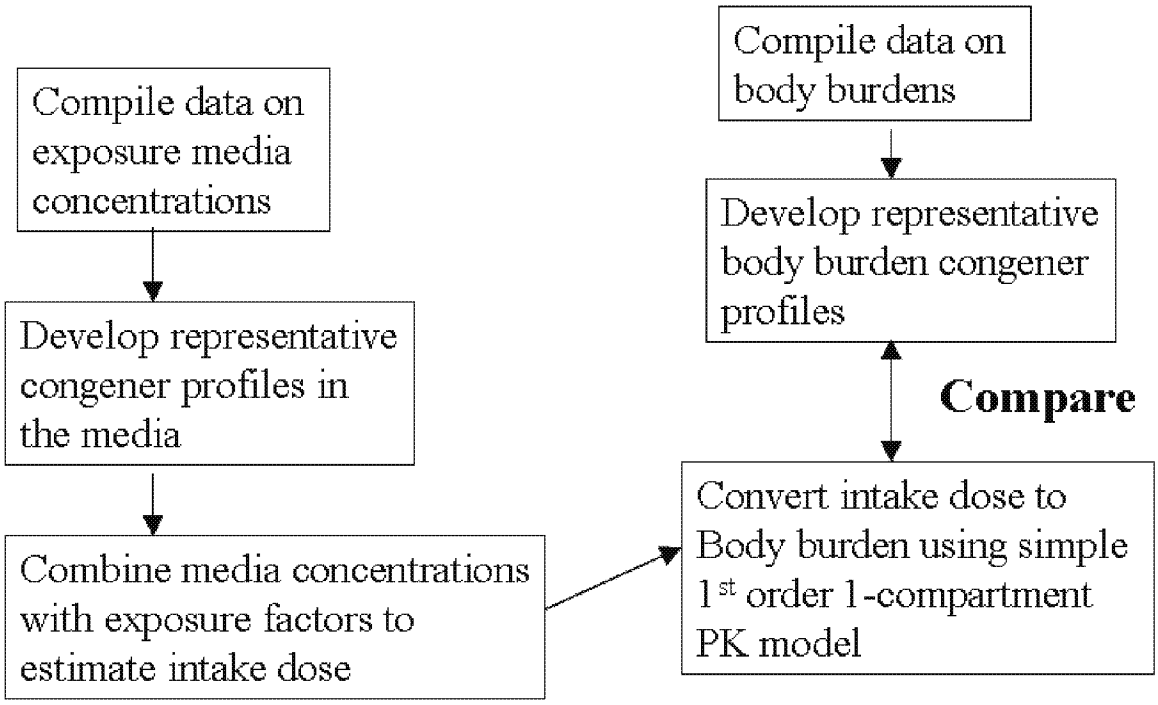

Figure 5-1. Approach for characterizing exposure to PBDEs in this report.

5.2.1. Blood Data

Seventeen studies that contained data on PBDEs in blood in the United States are reviewed in this section. Table 5-1 shows congener-specific data from these studies.

The most statistically rigorous and expansive study of background exposures to PBDEs is a recent analysis of 2003/2004 National Health and Nutritional Examination Survey (NHANES) data by Sjodin et al. (2008). Unfortunately, brominated diphenyl ether (BDE) 209 was not measured in this NHANES study, although Sjodin et al. (2008) note that the concentration of BDE 209 from pooled NHANES 2001/2 samples was 2 ng/g lipid weight (lwt; unpublished data; no further details supplied). A total of 2,040 serum samples from individuals 12 years of age and older were analyzed for 10 BDE congeners, including BDE 17, BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183. The geometric mean concentrations over the entire population were, in ng/g lwt, in 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

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Table 5-1. Blood concentrations of PBDE congeners in Americans

Congener	Concentration (ng/g lwt)	Comment	Citation
DiBDE			
15	0.2; 0.09–0.5	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
TriBDE			
17	0.3	N = 1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. (2004)
	0.1, 0.03, 0.05	Three 2003 results: one pooled serum (n = 100) from TX, one 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)
	0.1, ND–0.7	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
28	1.2	N = 2040; NHANES 2003/2004; 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	Three 2003 results: one pooled serum (n = 100) from TX, one 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. (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)
	0.4, 1.0	N = 20 mothers and 20 toddlers pairs; median from mother, then child (ages 1.5−4)	EWG (2008)
	0.6; ND	Medians from "high" (n = 22) and "low" (n = 14) New York State sportfish consumers	Spliethoff et al. (2008)
	1.3, 0.09–5.6	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
30	0.2; ND–0.06	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
37	<0.1; ND–0.06	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)

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Table 5-1. Blood concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
TetraBDE			
47	20.5	N = 2040; NHANES 2003/2004; geometric mean	Sjodin et al. (2008)
	0.6 (<0.4–23.8)	Median, range; 6 of 12 blood donor samples collected in IL in 1988 quantified	Sjodin et al. (2001)
	13.2, 0.7–1388.6	Geometric mean; range of 93 urban angler samples in NY and NJ; 93% detected	Morland et al. (2005)
	28.1	N = 1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. (2004)
	34 (29–98)	Median from 2000/2002 (range) in trend study from pooled blood from around United States	Sjodin et al. (2004a)
	20.8; 0.7–109.0	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
	28, 9.1–310	Median; range from maternal/umbilical cord blood (n = 12); 2001; umbilical cord blood was identical	Mazdai et al. (2003)
	50.6 (<10–511)	Mean, range from Laotian reproductive age women in San Francisco area, 1997–1999	Petreas et al. (2003)
	32.5, 44.2, 25.0	Three 2003 results: one pooled serum (n = 100) from TX, one 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 two parents, one 5 year-old, and one 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. (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. (2007)
	8.8, 30.6	N = 20 mothers and 20 toddlers pairs; median from child, then child (ages 1.5–4)	EWG (2008)
	12.8, 1.3	Medians from "high" (n = 22) and "low" (n = 14) New York State sportfish consumers	Spliethoff et al. (2008)
	18, 17	Geometric means for men (n = 350) and women (n = 150) from a study of sport fishers	Anderson et al. (2008)

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Table 5-1. Blood concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
49	0.1; ND–0.8	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
66	0.3	N = 1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. (2004)
	0.3, NA, 0.4	Three 2003 results: one pooled serum (n = 100) from TX, one 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)
	0.2, ND	Medians from "high" (n = 22) and "low" (n = 14) New York State sportfish consumers	Spliethoff et al. (2008)
	0.2; ND–0.9	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
71	<0.1; ND–0.1	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
75	<0.1; ND–0.1	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
77	ND, NA, 0.01	Three 2003 results: one pooled serum (n = 100) from TX, one 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	Morland et al. (2005)
	0.8	N = 1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. (2004)
	0.7 (0.5−1.4)	Median from 2000/2002 (range) in trend study from pooled blood from around United States	Sjodin et al. (2004a
	NA, 1.1, 1.2	Three 2003 results: one pooled serum (n = 100) from TX, one 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. (2007)

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Table 5-1. Blood concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
	ND, 0.4	N = 20 mothers and 20 toddlers pairs; median from mother, then child (ages 1.5−4)	EWG (2008)
	0.4, ND	Medians from "high" (n = 22) and "low" (n = 14) New York State sportfish consumers	Spliethoff et al. (2008)
	0.3; ND–1.8	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
99	5.0	N = 2,040; NHANES 2003/2004; geometric mean	Sjodin et al. (2008)
	0.3, <0.2−3.7	Median, range; 8 of 12 U.S. blood donor samples collected in IL 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	Morland et al. (2005)
	9.2	N = 1; Pool of 15 individuals from Philadelphia, Memphis, Miami	Focant et al. (2004)
	11 (6.8−26)	Median from 2000/2002 (range) in trend study from pooled blood from around United States	Sjodin et al. (2004a)
	5.7, 2.4−68	Median; range from maternal/umbilical cord blood (n = 12); 2001; umbilical cord blood was identical	Mazdai et al. (2003)
	8.4, 12.8, 11.1	Three 2003 results: one pooled serum (n = 100) from TX, one 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 two parents, one 5 year-old, and one 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	Wolff et 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. (2007)
	1.5, 6.2	N = 20 mothers and 20 toddlers pairs; median from mother, then child (ages 1.5−4)	EWG (2008)
	3.4, ND	Medians from "high" (n = 22) and "low" (n = 14) New York State sportfish consumers	Spliethoff et al. (2008)
	5, 5	Geometric means for men (n = 350) and women (n = 150) from a study of sport fishers	Anderson et al. (2008)
	4.3; 0.6–24.2	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)

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Table 5-1. Blood concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
100	3.9	N = 2040; NHANES 2003/2004; geometric mean	Sjodin et al. (2008)
	0.2, <0.1−2.4	Median, range; 10 of 12 U.S. blood donor samples collected in IL 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 2000/2002 (range) in trend study from pooled blood from around United States	Sjodin et al. (2004a)
	4.2, 1.9−110	Median; range from maternal umbilical cord blood (n = 12); 2001; umbilical cord blood was identical	Mazdai et al. (2003)
	5.7, 5.2, 4.7	Three 2003 results: one pooled serum (n = 100) from TX, one 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 two parents, one 5 year-old, and one 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. (2007)
	1.2, 6.2	N = 20 mothers and 20 toddlers pairs; median from mother, then child (ages 1.5−4)	EWG (2008)
	3.0, ND	Medians from "high" (n = 22) and "low" (n = 14) New York State sportfish consumers	Spliethoff et al. (2008)
	3.3	Geometric means for men (n = 350) and women (n = 150) from a study of sport fishers	Anderson et al. (2008)
	2.9; 0.1–19.7	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
HexaBDE			
138	NA, 0.3, 0.2	Three 2003 results: one pooled serum (n = 100) from TX, one 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)

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Table 5-1. Blood concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
	0.04	N = 11; mean from blood/milk study (ND = 0)	Schecter et al. (2006c)
	ND, ND	Medians from "high" (n = 22) and "low" (n = 14) New York State sportfish consumers	Spliethoff et al. (2008))
	0.1; ND–0.4	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
153	5.7	N = 2040; NHANES 2003/2004; geometric mean	Sjodin et al. (2008)
	0.3, 0.1–2.0	Median, range; 12 of 12 U.S. blood donor samples collected in IL 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 2000/2002 (range) in trend study from pooled blood from around United States	Sjodin et al. (2004a)
	2.9, 1–83	Median; range from maternal/umbilical cord blood (n = 12); 2001; umbilical cord blood was identical	Mazdai et al. (2003)
	12.3, 11.7, 5.7	Three 2003 results: one pooled serum (n = 100) from TX, one 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 two parents, one 5 year-old, and one 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. (2007)
	5.8, 15.3	N = 20 mothers and 20 toddlers pairs; median from mother, then child (ages 1.5–4)	EWG (2008)
	3.0, 0.7	Medians from "high" (n = 22) and "low" (n = 14) New York State sportfish consumers	Spliethoff et al. (2008)
	6.6	Geometric means for men (n = 350) and women (n = 150) from a study of sport fishers	Anderson et al. (2008)
	5.2; 0.1–20.9	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)

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Table 5-1. Blood concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
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 2000/2002 (range) in trend study from pooled blood from around United States	Sjodin et al. (2004a)
	0.3 (ND−6.1)	Median, range from maternal/umbilical cord blood (n = 12); 2001; umbilical cord blood was identical	Mazdai et al. (2003)
	0.8, 0.8, 1.0	Three 2003 results: one pooled serum (n = 100) from TX, one 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 two parents, one 5 year-old, and one 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 24 pregnant Latina women in CA	Bradman et al. (2007)
	0.6, ND	Medians from "high" (n = 22) and "low" (n = 14) New York State sportfish consumers	Spliethoff et al. (2008)
	0.3; 0.05–1.5	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
155	0.1; ND–0.2	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States.	PSR (2009)
HeptaBDE			
183	0.2, 0.1−1.3	Median, range; 12 of 12 U.S. blood donor samples collected in IL 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	Morland et al. (2005)
	ND, 0−2.7	Median; range from maternal/umbilical cord blood (n = 12); 2001; umbilical cord blood was identical	Mazdai et al. (2003)
	0.3, 0.4, 0.4	Three 2003 results: one pooled serum (n = 100) from TX, one 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)

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Table 5-1. Blood concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
	ND, ND	N = 20 mothers and 20 toddlers pairs; median from mother, then child (ages 1.5−4)	EWG (2008)
	0.3; 0.05–0.8	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States.	PSR (2009)
190	<0.1; ND–0.2	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
OctaBDE			
197	0.3, 0.5	N = 20 mothers and 20 toddlers pairs; median from mother, then child (ages 1.5−4)	EWG (2008)
203	ND(0.1), <0.1−0.2	Median, range; 5 of 12 U.S. blood donor samples collected in IL in 1988 quantified	Sjodin et al. (2001)
	0.2; 0.07–0.3	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States.	PSR (2009)
NonaBDE			
206	0.4; 0.2–0.8	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
207	0.6; 0.4–1.0	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
208	0.3; 0.2–0.7	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)
DecaBDE			
209	ND(1.0), <1.0−33.6	Median, range; 5 of 12 U.S. blood donor samples collected in IL in 1988 quantified	Sjodin et al. (2001)
	NA, 1.4, 2.7	Three 2003 results: one pooled serum (n = 100) from TX, one 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 two parents, one 5 year-old, and one 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)
	ND, 1.7	N = 20 mothers and 20 toddlers pairs; median from mother, then child (ages 1.5−4)	EWG (2008)
	3.1; ND–9.0	Mean (ND = 0), range; samples from 20 health care providers from 10 states around the United States	PSR (2009)

ND = not detected.

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for the other BDE congeners because they were detected at less than 60%, including the low frequencies of 5%, 15%, 21%, and 23% for BDE 17, BDE 183, BDE 66, and BDE 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 BDE 28, BDE 47, BDE 99, BDE 100, and BDE 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 concentrations of BDE 47 for these four age categories, respectively, were 28.2, 21.5, and 17.7, and then rose to 19.7 ng/g lwt. Although not statistically significant, the geometric mean concentrations for males were higher than females for BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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. In addition, 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 congeners including BDE 17, BDE 28, BDE 47, BDE 66, BDE 77, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 209. All congeners were nondetects for the 1973 sample. The two pooled samples collected in 2003, one serum and one whole blood, both 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 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 BDE 153 comprising about the same amounts—BDE 99 was

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between 14 and 21% and BDE 153 was between 11 and 20%. From the individual samples, an interesting trend was that women had higher concentrations than men did: 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 nondetects in 19 of 39 individual samples.

The public interest group, Physicians for Social Responsibility, measured a suite of contaminants in the blood of 20 volunteers in the medical sector (nurses, physicians, and others) from 10 states around the country (PSR, 2009). They measured for perfluorinated compounds and PBDEs in blood, and phthalate metabolites, triclosan, bisphenol A, and mercury in urine. They sampled between February and April of 2009, and measured 28 BDE congeners from BDE 15 to BDE 209. Assuming ND = 0, they found an average total PBDE concentration of 41.2 ng/g lwt, dominated by BDE 47 at 20.8 ng/g lwt. BDE 209 was found in half the samples, with an average concentration of 3.1 ng/g lwt (assuming again ND = 0).

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 frames: 1985–1989 (n = 9), 1990–1994 (n = 14), 1995–1999 (n = 10), and 2000–2002 (n = 7), and the samples were measured for BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 154. A clear trend was seen, with total concentrations rising from 9.6 ng/g lwt in the 1985–1989 time frame, to 48 ng/g lwt in 1990–1994, 71 ng/g lwt in 1995–1999, and 61 ng/g lwt in 2000–2002. BDE 47 comprised 55–65% of the total in the four time frames. In contrast to BDEs, BB-153 (2,2',4,4',5,5'-hexabromobiphenylhexabrominated biphenyl, a marker for polybrominated biphenyls) and CB-153 (2,2',4,4',5,5'-hexachlorobiphenyl, 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 U.S. donors who provided blood

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at a commercial blood collection facility in the state of Illinois in 1988, and then stored at −70°C, were retrieved for analysis. Seven congeners were quantified: BDE 47, BDE 99, BDE 100, BDE 153, BDE 183, BDE 203, and BDE 209. Three unidentified octa BDE and three unidentified nona BDEs were noted. The data were reported in units of pmol/g lwt, converted to ng/g lwt by multiplying by the congener's molecular weight in pg/pmol, 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. Sjodin et al. (2001) 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 were sampled from two disparate cohorts of women who were sampled in the late 1990s, and they were 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 1998. The second set was serum from a group of 50 Laotian women of reproductive age living in the San Francisco Bay area, taken in the 1997−1999 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 nondetects 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. Since PBDEs were not produced commercially until the 1970s, this is not an unexpected result. The mean and median from the adipose samples were 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

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contrasts with 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 BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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 (ND) 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 arithmetic average 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.

In contrast, Spliethoff et al. (2008) found a significant correlation between levels of BDE congeners in the blood of New York state anglers and the frequency with which they consumed fish from Lake Ontario and it tributaries. Samples were obtained from 38 individuals who were part of a larger New York State Angler Cohort Study whose blood was sampled between 1995 and 1997. Statistically significant relationships were noted between blood concentrations of BDE congeners (BDE 28, BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 154) and total BDE (sum of congeners) and number of years consuming sportfish between 1980 and 1990, and number of meals eaten in the previous year to the analysis. When separating "high" consumers (median of 7−11 sportfish meals, maximum of 24−36 meals, in the previous year) from "low" consumers (median of 0 meals, maximum of 1−6 meals), the median total concentration of high consumers (n = 22) was 23.1 ng/g lwt, while the median for low consumers (n = 14) was 1.9 ng/g lwt. BDE 47 was the highest congener found, at a median of 12.8 ng/g lwt for "high" consumers and 1.9 ng/g lwt for "low" consumers.

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In a third study on anglers consuming fish from the Great Lakes, Anderson et al. (2008) also found statistically significant relationships between total PBDE levels in blood (sum of BDE 28, BDE 47, BDE 49, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153; BDE 209 measured but not detected) and years of sportfish consumption as well as catfish and shellfish consumption. Multivariate as well as highest quartile associations were also found with variables such as computer use as well as sportfish consumption. This study entailed collection of blood from 508 volunteers (350 men, 158 women) from a full cohort of 4,107 frequent and infrequent consumers of Great Lakes fish who were sampled in 2004 and 2005. The geometric mean total PBDE from this sample was 27 ng/g lwt for men and 25 ng/g lwt for women, of which BDE 47 comprised almost two-thirds. Anderson et al. (2008) compared these results to derived geometric means for Caucasian men and women over 30 years from NHANES 2003/2004 and found the NHANES results to be higher: 34 ng/g lwt for men and 30 ng/g lwt for women.

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, 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. BDE 17, BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 154 were analyzed. The blood sample results were expressed in pg/g wet weight (wwt) 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 one sample was 52.7 ng/g lwt, dominated by BDE 47 at 28.1 ng/g lwt, with comparable contributions by BDE 99, BDE 100, and BDE 153 at 9.2, 6.9, and 6.2 ng/g lwt, respectively.

Wolff et al. (2005) conducted a study of exposures among pregnant women who were near the World Trade Center (WTC) Site on 9/11/2001. The study involved a complex evaluation of exposures, including measurement of key persistent contaminants including polycyclic aromatic hydrocarbons, PCBs, dioxins, and PBDEs in blood. The authors did not find

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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 BDE 28, BDE 47, BDE 99, BDE 100, and BDE 153 at 0.65, 9.7, 1.5, 1.8, and 1.8 ng/g lwt, respectively.

Bradman et al. (2007) 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 congeners were measured, including BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183. The resulting concentrations were 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 showed 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 four, 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 BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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 five 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 at values below 10 days, while the half-lives of lower-brominated congeners

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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 human milk, although Fisher et al. (2006) suggest that it might also be due to exposure to house dust. Of the non-BDE 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-third to one-half of the concentration of BDE 47.

A second and more recent study providing data on PBDE concentrations in young children was conducted by the Environmental Working Group (EWG, 2008). Twenty pairs of mothers and children from 16 states were sampled for 20 congeners; positive occurrences were found for these 10: BDE 28, BDE 47, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 183, BDE 197, and BDE 209 (no detections for BDE 17, BDE 66, BDE 71, BDE 196, BDE 201, BDE 203, BDE 206, BDE 207, and BDE 208; BDE 154 was not included because it coeluted with PCB 153). The children ranged in age from 1.5 to 4 years old. The median total concentration (sum of the 10 congeners found) from the 20 children was nearly four times that of the mother, at 69.3 ng/g lwt for the children versus 18.2 ng/g lwt for the mother. BDE 209 was found in 13 of 20 children, with a median of 1.7 ng/g lwt, while it was found in only 9 of 20 mothers (median at ND). The congener found at the highest concentration was BDE 47 (medians of 30.6 ng/g lwt in children and 8.8 ng/g lwt in mothers), followed by BDE 153 (medians of 12.5 ng/g lwt in children and 5.8 in mothers).

Schecter et al. (2006a) measured the concentrations of 12 BDEs (BDE 17, BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 209) in the blood of eight 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. BDE 99 and BDE 100 totaled about 6 ng/g lwt. BDE 209 was not detected, although detection limits (DLs) 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

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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.

Schecter et al. (2006c) provided important insight as to the relative partitioning of PBDEs between blood and milk. Eleven mothers donated samples of their blood and milk, which were simultaneously measured for BDE 17, BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 209. The location was not specified, but it was assumed to be in Texas, where the primary author is located (and within the article, it was stated that the specimens were packed in dry ice and shipped to the analytical lab in Germany—coauthors on the paper originate from Germany), and the date of sampling is not specified, but is assumed to be 2004 or 2005, since the paper was published in 2006. The focus of the paper was the relationship between concentrations found in blood and milk. Tables 5-1 and 5-2 provide average concentrations of the congeners in blood and milk, and the sum of these congener averages (the article did not provide total concentrations or statistics for totals or individual congeners; congener-specific averages were generated for this report) is about the same in milk and blood at about 33 ng/g lwt. However, there were differences in partitioning between blood and milk among the congeners, most importantly for BDE 209 as compared to other congeners. The average concentration in blood was 3.7 ng/g lwt, while in milk it was 0.42. For other congeners, the concentrations were more nearly similar. The congener found at the highest average concentration in blood (B) and milk (M) was BDE 47 at 13.1 ng/g lwt (B) and 17.1 ng/g lwt (M); the second highest was BDE 153 at 8.4 ng/g lwt (B) and 7.0 ng/g lwt (M). Schecter et al. (2006c) generated ratios of blood: milk concentrations; when nondetected, the value was set at 1/2 DL. The highest ratio (greatest disparity between blood and milk, with blood much higher) was with BDE 209, with the median and mean over the 11 individuals in the 35–40 range. The ratio was second highest for BDE 183, with median and mean ratios of 1.8 and 2.3. All other median and mean ratios for the congeners frequently detected (BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154) were between 0.7 and 1.5. These data support the argument that BDE 209 does not partition equally to all body lipids.

A second study providing some insight into partitioning of BDE congeners between blood and milk is LaKind et al. (2009). In this study, a group of 10 women were sampled post partum, at several intervals and for both blood and milk. Milk was sampled 4 times (1, 2,

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3 months, and at cessation of breast-feeding) and blood sampled twice (1 month and final milk sampling event). Contaminants measured included dioxins and furans, PCB, and PBDEs. Summary statistics were provided in LaKind et al. (2009) for BDE 47, BDE 153, and BDE 100. The median concentrations over all milk samples (n = 35, 100% detection) for the three congeners was 26, 4, and 3.5 ng/g lwt for BDE 47, BDE 100, and BDE 153, respectively. The median concentration for the blood samples (n = 17, 100% detection for BDEs 47 and 100; 59% for 153) was 15.5, 2.4, and 4 ng/g lwt for BDE 47, BDE 100, and BDE 153, respectively. In studying the relationship between blood and milk concentrations, only concurrently measured samples collected one month post partum were studied, and only BDE 47 and BDE 100 were included because they were detected in all samples. Individual results were not available, but in a subgroup of all contaminants measured which includes these BDE congeners (and also pesticides/metabolites and 9 PCB congeners), a best-fit line was found at, milk = 1.74 * serum (r^2 = 0.98), where milk and serum were both expressed on a lipid basis. This suggests that for these two BDE congeners, milk concentrations tended to be just under twice the concentration in blood.

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 hundreds and thousands of ng/g lwt.

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 Australia, Sweden, Norway, New Zealand, the United Kingdom (UK), the Faroe Islands, Japan, and Nicaragua.

Toms et al. (2008) report on results from pooled samples collected from 8,132 residents of Australia in 2002/2003 and 2004/2005. The pools were constructed to correspond to ages 0−4, 5−15, 16−30, 31−45, 46−60, and >60 years old. A total of 35 congeners were measured, making this study the most comprehensive found in terms of the number of congeners measured, although BDE 209 was not included. Looking only at results for all age categories greater than 16 years old (adults essentially), the mean total PBDE concentration for 2002/2003 was 15 (standard deviation ±5, median=13) ng/g lwt, and for 2004/2005, it was 18 (S.D.±5, median=16) ng/g lwt. Like the Fischer et al. (2006) study, there was a clear age effect in the data, with the highest concentrations found in the youngest individuals. For 2004/2005 (when all age group

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data were available), the mean was 73 (S.D.±7, median=75) ng/g lipid for 0–4 years of age, 28 (S.D.±7, median=29) for 5–15 years of age, 20 (S.D.±5, median=18) ng/g lipid for 16–30 years of age, and 15–18 ng/g lipid for age categories above 30.

Blood from 37 Swedish men were sampled in 1991 and 2001 and measured for BDE 28, BDE 47, BDE 99, BDE 100, BDE 128, BDE 154, BDE 153, BDE 183, BDE 196, BDE 197, BDE 203, BDE 206, BDE 207, BDE 208, and BDE 209 (Jakobsson 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 were 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. BDE 154 and BDE 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 BDE 209 and BDE 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 PCB-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 (BDE 28, BDE 58, BDE 66, BDE 99, BDE 100, BDE 153, and BDE 154) were near the detection limit for four of the five 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 four of five individuals, at concentrations ranging from about 9.4 to 17.4 ng/g lwt. One individual also had quantified measurements of BDE 197/204 (coeluting), BDE 196, BDE 206, and BDE 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

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decaBDE congeners. A positive relationship was found when plotting a sum of BDE concentrations (including BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154, but not BDE 209) for each individual against the sum BDE dust concentration found in that individual's home; however, that relationship was driven by one individual having the highest plasma and dust sum BDE concentrations. This relationship did not hold up when adding BDE 209 to the dust and plasma sumBDE. There was no similar relationship found between air and plasma concentrations.

Pooled samples of about 20 individuals each from five hospitals in Norway (total number of samples analyzed was not provided) were analyzed for 11 BDEs: BDE 28, BDE 37, BDE 47, BDE 85, BDE 99, BDE 100, BDE 119, BDE 138, BDE 153, BDE 154, and BDE 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 (BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183) showed a concentration range of 0.5 to 5.5 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." Also reported in this study are age trends for pooled samples stratified by age for age ranges 0–4, 5–14, 15–24, 25–59, and >60. Data were presented for 1998 and 2002, with the 1998 data from an earlier study by the same team of researchers (Thomsen et al., 2002). The youngest age group, 0–4 years, had the highest concentration, at total concentrations at about 11.5 ng/g lwt in 1998 and 10 ng/g lwt in 2002, while other results for both years and age ranges were between about 3 to 6 ng/g lwt.

Harrad and Porter (2007) report on concentrations of congeners including BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183 in the blood of 23 individuals (10 males, 13 females ages 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

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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. BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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.

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. BDE 47, BDE 99, BDE 100, BDE 153, BDE 209, and BDE 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 coeluting BDEs 153/154, accounting for 26% each. For children, the predominant congener was BDE 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.

The study that 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 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 who 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). BDE 47, BDE 99, BDE 100, BDE 153, BDE 183, BDE 203, and BDE 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

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eight pools was 38 ng/g lwt. BDE 47 was the most prominent congener, contributing nearly 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 generally were in the range of 30–100 ng/g lwt. The most robust study of U.S adults and older children was based upon NHANES 2003-2004 data (2,040 serum samples, all greater than 12 years of age), and reported geometric mean concentrations of key congeners BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154 at 1.2, 20.5, 5.0, 3.9, 5.7, and 2.3 ng/g lwt, respectively (Sjodin et al, 2008).

2. U.S. levels were higher than levels found in nearly every other study done outside of the United States, with most non-U.S. data suggesting total PBDEs to be less than 10 ng/g lwt total PBDEs.

3. Studies in both the US and abroad showed higher concentrations in toddlers (less than 4 years old) compared to older ages of children and adults. In paired sampling studies, including mother and child, the child's body burden exceeded the mother's by about a factor of four, exceeding 100 ng/g lwt total PBDEs in some cases. Higher levels in toddlers were attributed to ingestion of mother's milk and high exposures to house dust.

4. The predominant congener in the US and abroad was BDE 47, which comprised about 50% of the total concentration. The second most commonly found congeners were BDE 99 and BDE 153, both explaining about 10–20% of total concentrations. Most studies did not measure BDE 209, but, when measured, BDE 209 was found at low levels near 5 ng/g lwt. Low levels of BDE 209 have been attributed to the rapid half-life of under 10 days in humans.

5.2.2. Human Milk Data

While blood data suggested concentrations of total PBDEs in the range of 30–100 ng/g lwt, data on PBDEs in human 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

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Table 5-2. Breast milk concentrations of PBDE congeners in Americans

Congener	Concentration (ng/g lwt)	Comment	Citation
MonoBDE			
3	0.15	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
DiBDE			
7	0.01	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
8/11	0.02	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
12	0.01	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
13	0.01	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
15	1.46	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
	1.5	WHO interlab calibration study with results from 17 of 24 countries; United States listed here	Kotz et al. (2005)
TriBDE			
17	0.1	Mean from 20 primaparae women from around United States	Lunder and 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 United States	Lunder and Sharp (2004)
28	7.4	Mean of 3 pooled sample analyses from CO, CA, and NC	Focant et al. (2004)
	8.6	WHO interlab calibration study with results from 17 of 24 countries; United States 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)
	2 (97%)	Median (% detect) from a sample of 303 women sampled 3 months post partum in NC	Daniels et al. (2009)

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Table 5-2. Breast milk concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
28/32	3.8	Mean of 40 samples from primaparae women from Pacific Northwest	NEW (2004)
28/33	6.2	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
32	0.2	Mean of 40 samples from primaparae women from Pacific Northwest	NEW (2004)
35	0.02	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
37	0.06	Mean from 20 primaparae women from around United States	Lunder and 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 United States	Lunder and Sharp (2004)
	50; 26	Mean; median of 40 samples from primaparae women from Pacific Northwest	NEW (2004)
	233.9	WHO interlab calibration study with results from 17 of 24 countries; United States 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)
	28 (99%)	Median (% detect) from a sample of 303 women sampled 3 months post partum in NC	Daniels et al. (2009)
49	0.6	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
66	1.1	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
	0.3	Mean of 40 samples from primaparae 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)

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Table 5-2. Breast milk concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
	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)
	ND (46%)	Median (% detect) from a sample of 303 women sampled 3 months post partum in NC	Daniels et al. (2009)
71	0.05	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
	0.5, 0.2	Mean, median of 40 samples from primaparae women from Pacific Northwest	NEW (2004)
75	0.07	Mean from 20 primaparae women from around United States	Lunder and 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 United States	Lunder and Sharp (2004)
	1.1, 0.6	Mean, median of 40 samples from primaparae 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)
	0.5 (65%)	Median (% detect) from a sample of 303 women sampled 3 months post partum in NC	Daniels et al. (2009)
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 United States	Lunder and Sharp (2004)
	10, 5.4	Mean, median of 40 samples from primaparae women from Pacific Northwest	NEW (2004)
	58.7	WHO interlab calibration study with results from 17 of 24 countries; United States listed here	Kotz et al. (2005)

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Table 5-2. Breast milk concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
	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)
	5 (92%)	Median (% detect) from a sample of 303 women sampled 3 months post partum in NC	Daniels et al. (2009)
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 United States	Lunder and Sharp (2004)
	12, 5.2	Mean, median of 40 samples from primaparae women from Pacific Northwest	NEW (2004)
	39.2	WHO interlab calibration study with results from 17 of 24 countries; United States 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)
	5 (92%)	Median (% detect) from a sample of 303 women sampled 3 months post partum in NC	Daniels et al. (2009)
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 United States	Lunder and Sharp (2004)
126	0.04	Mean from 20 primaparae women from around United States	Lunder and 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)

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Table 5-2. Breast milk concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
	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 United States	Lunder and Sharp (2004)
140	0.21	Mean from 20 primaparae women from around United States	Lunder and 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 United States	Lunder and Sharp (2004)
	16, 4.8	Mean, median, of 40 samples from primaparae women from Pacific Northwest	NEW (2004)
	17.6	WHO interlab calibration study with results from 17 of 24 countries; United States 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)
	6 (99%)	Median (% detect) from a sample of 303 women sampled 3 months post partum in NC	Daniels et al. (2009)
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 United States	Lunder and Sharp (2004)
	0.8, 0.4	Mean, median, of 40 samples from primaparae women from Pacific Northwest	NEW (2004)
	3.0	WHO interlab calibration study with results from 17 of 24 countries; United States 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)
	0.3 (54%)	Median (% detect) from a sample of 303 women sampled 3 months post partum in NC	Daniels et al. (2009)

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Table 5-2. Breast milk concentrations of PBDE congeners in Americans (continued)

Congener	Concentration (ng/g lwt)	Comment	Citation
155	0.3	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
HeptaBDE			
183	0.15	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
	0.3, 0.2	Mean, median, of 40 samples from primaparae 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)
	ND (12%)	Median (% detect) from a sample of 303 women sampled 3 months post partum in NC	Daniels et al. (2009)
190	0.01	Mean from 20 primaparae women from around United States	Lunder and 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 United States	Lunder and Sharp (2004)
207	0.06	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
208	0.01	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
DecaBDE			
209	0.24	Mean from 20 primaparae women from around United States	Lunder and Sharp (2004)
	0.8, 0.4	Mean, median, of 40 samples from primaparae 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)

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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 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 six having levels above 100 ng/g lwt and two exceeding 700 ng/g lwt. These results were the highest found to date and significantly higher than for European women. The most common BDE was BDE 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 BDE 32, BDE 28/32, BDE 47, BDE 66, BDE 71, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209. Levels ranged from 6 to 321 ng/g lwt in the human 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 human milk from 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.

Daniels et al (2009) obtained samples from women participating in the Pregnancy Infection and Nutrition studies which were conducted in North Carolina between 2001 and 2005. Between 2004 and 2006, a subset of the 589 eligible women remaining in the study were sampled twice: months post partum. Nine BDE congeners (BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE183) were measured, and summary statistics were provided for these congeners for 303 of the 304 sampled at 3 months post partum (one sample was discarded). BDE 47 dominated the profile, with a median of 28 ng/g lwt. Other congeners had medians at 6 ng/g lwt (BDE 153, which was the second highest) or lower. Total

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BDEs equaled the sum of the 5 congeners detected 70% or more of the time (BDE 28, BDE 47, BDE 99, BDE 100, and BDE 153), and the median total was 51 ng/g lwt, with a range of 1.2 to 2,010 ng/g lwt. Looking at the samples from the 83 women providing samples at 3 and 12 months post partum, there was a wide range of change in total BDE concentrations, with the median showing an increase of 14%, an inter-quartile range of −26 to +50%. BDE 153 increased the most between sampling dates, and the authors could not provide an explanation for this finding.

Schecter et al. (2003) collected milk from 47 volunteer donors between August and December 2002. Twenty-four donors were from Austin, TX, and 23 were from Dallas, TX. Samples were measured for 13 BDEs including BDE 17, BDE 28, BDE 47, BDE 66, BDE 77, BDE 85, BDE 99, BDE 100, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 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 seven 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. BDE 17, BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, and BDE 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 gas chromatography and isotope dilute high resolution mass spectrometry (GC-IDHRMS) analysis were compared with the newer GC/GC-IDTOFMS, 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.

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Two studies have been conducted in the Boston, MA area. In one, 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 3 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 human milk. The second study in the Boston area was conducted by Johnson-Restrepo et al. (2007), who measured 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 three women for BDE 47 and BDE 99 (Sjodin et al., 2005). These data originate from an ongoing study at 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 two of three women from Postpartum Day 40 to 120, and from 10 to about 15 ng/g lwt from Day 40 to 60 in the other woman. BDE 99 increased from 50 to100 ng/g lwt in one participant from Day 40 to 120, increased slightly from approximately 5 to 6 ng/g lwt from Day 40 to 60 in another, and decreased from 8 to about 5 ng/g lwt from Day 40 to 90 in the third participant. A more comprehensive longitudinal study did show depuration over the course of up to 85 weeks after birth (Hooper et al., 2007). Summary statistics of individual congeners (BDE 32, BDE 28/33, BDE 47, BDE 66, BDE 71, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183) were not provided, and the box and whisker plots for total concentrations (sum of the congeners) said little more than that means appeared to be less than 100 ng/g lwt. However, overall results suggested that concentrations of all congeners decreased between 1 and 3% per month over the course of the study, over the entire range of total concentrations, which was provided at between 21 and 1,330 ng/g lwt.

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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 were 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 2 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 suggest much higher concentrations in America compared to European and Asian countries. Extracted milk fat from the 3rd round of the World Health Organization (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 BDE 15, BDE 28, BDE 47, BDE 77, BDE 99, BDE 100, BDE 126, BDE 153, and BDE 183. The highest level by far was found in a sample from the United States at 373.6 ng/g lwt; the second highest level was 10.3 ng/g lwt from a sample from Ireland. The predominant congener was BDE 47, at 63% in U.S. 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) reported on concentrations of 15 BDE congeners (although the specific congeners were not identified except to note that samples were fortified with BDE 28, BDE 77, BDE 99, BDE 153, and BDE 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) in Catalonia, Spain. 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. This same group of 15 women were revisited in 2007, and little change in concentrations were noted (Schulmacher et al, 2009). Now the mean for the two groups were 2.4 and 2.7 ng total PBDE/g lwt for the urban and industrial groups, respectively.

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Thomsen et al. (2005b) sampled milk of 151 women representing the northern, southwestern, and eastern parts of Norway. Samples were analyzed for BDE 28, BDE 37, BDE 47, BDE 85, BDE 99, BDE 100, BDE 119, BDE 138, BDE 153, BDE 154, and BDE 183. The sum of the seven most abundant BDE congeners (BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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 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 milk. Fifteen individual congeners were measured in all samples, including BDE 209. 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 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 BDE 15, BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, BDE 196, BDE 197, BDE 206, BDE 207, and BDE 209 (Inoue et al., 2006). The geometric means for the total amounts of the 13 PDEs in human milk and serum were 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,

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accounting for 8%. In milk, BDE 47 and BDE 153 were the major contributors, accounting for 28% and 23% of total BDEs, respectively.

Fangstrom et al. (2008) conducted a temporal study on Swedish mother's milk. Fourteen pooled milk samples representing 1980 (116 mothers pooled), 1984/1985 (102 mothers), several of the years between 1988 and 2002 (20 mothers), 2003 (15 mothers), and 2004 (20 mothers) were sampled for BDE 47, BDE 77, BDE 99, BDE 100, BDE 153, and BDE 209. It was impossible to quantify BDE 209 in milk, and the authors suggest this could be due to the short half-life of this compound, which has been noted for BDE 209 in serum. From the middle of the 1990s, the concentrations of the lower-brominated BDE congeners (BDE 47, BDE 99, and BDE 100) were decreasing, while BDE 153 appeared 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 (2001) to 1.4 (2002) to 1.2 (2003) to 0.9 (2004). Very similar trends were seen for BDE 99 and BDE 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 until 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 decreasing lower-brominated BDEs and increasing higher-brominated 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.

Lignell et al. (2009) obtained samples from 335 randomly recruited primiparas who lived in Uppsala County, Sweden, and delivered between 1996 and 2006. They measured the samples for PCBs, PCDD/Fs, hexabromocyclododecane (HBCD), and PBDEs. While declining trends over time were noted for PCBs and PCDD/Fs, the results for PBDEs were inconsistent. BDEs 47 and 99 declined by 4.2% and 7.6%, respectively, while BDE 153 increased 4.9% each year. BDEs 100 and 154 were also measured, and BDE 100 showed little change (decline 0.9% each year) and results for BDE 154 were not provided. The sum of the five congeners, the total concentration, declined 1.7% each year. The mean and median total concentration for all years was 3.5 and 2.9 ng/g lwt, respectively. The mean for BDE 47 over all years was the highest at 1.9 ng/g lwt, while the means for BDEs 99, 100, and 153 were all near 0.5 ng/g lwt.

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In a temporal study of organohalogen compounds, including PCBs, PCDD/Fs, and PBDEs, in the 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 BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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 frame.

Ohta et al. (2002) determined the concentration of PBDEs in milk of 12 primaparae women at 1 month after delivery in Japan. The PBDEs evaluated included BDE 28, BDE 47, BDE 99, BDE 153, and BDE 154. Concentrations ranged between 0.7 and 2.8 ng/g lwt in milk. Samples were also taken of numerous food products, including 20 fish samples, spinach, potatoes, 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 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.

A similar study measuring PBDE congeners in breast milk and tying them to dietary and lifestyle habits occurred in North China in 2006 (Zhu et al., 2009). A total of eight congeners, including BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209, were measured in 80 primiparas living in Tianjin, China. The total concentration ranged from 1.7 to 4.5 ng/g lwt. The concentration of BDE 209 was the same in blank samples as measured in the breast milk, and hence BDE 209 results were not reported. BDE 47 was the most predominant congener found, explaining between 22 and 34% of the concentration, although the authors noted that this predominance was less than for measurements found around the world. A significant correlation was found between concentrations in human milk and the time the woman used electronic appliances during the day (hours/day). There were no findings of note relating breast milk to diet.

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

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6.1–18.7 ng/g lwt. BDE 47 dominated the profile, explaining over 50% of the total, followed by BDE 99, BDE 100, and BDE 153—all of which contributed between 10 and 20%.

In summary, data suggest concentrations of total BDEs in women's milk in the United States exceeded that in blood, perhaps averaging near 100 ng/g lwt, in contrast to a range more like 30–100 ng lwt in blood. 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 milk of U.S. women exceeded that of women outside of the United States. Milk concentrations of total PBDEs outside of the United States were mostly below 10 ng/g lwt. As for blood, BDE 209 was usually not measured, or when measured, not found in most instances. BDE 47 was the predominant congener, followed by either BDE 99 or BDE 153.

5.2.3. Adipose Tissue Data

Most of the body burden data originate from either blood or human milk. The very limited data on adipose are generally consistent with blood and milk data—U.S. data are higher than data from other countries; BDE 47 predominates, and BDE 209 is either not measured or not found when measured.

Johnson-Restrepo et al. (2005) measured BDEs in adipose tissue from 52 individuals who were undergoing liposuction during October 2003–October, 2004 in New York City. BDEs sampled included BDE 28, BDE 30, BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, BDE 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

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(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 BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 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.

Fernandez et al. (2007) reported on the concentrations of 14 BDE congeners (BDE 28, BDE 47, BDE 66, BDE 71, BDE 75, BDE 77, BDE 85, BDE 99, BDE 100, BDE 119, BDE 138, BDE 153, BDE 154, and BDE 183) in adipose tissue from 20 women who were undergoing surgery for malignant and benign diseases in Granada, Spain. Three congeners were never found above the quantitation limit: BDE 71, BDE 77, and BDE 119. BDE 47, BDE 99, BDE 100, BDE 153, and BDE 183 were present at the highest levels and constituted 96% of total concentrations. Of these, BDE 153 contributed about 42%, with BDE 47 and BDE 99 2nd and 3rd most common at 24% and 10%, respectively. The mean concentration of total BDEs (including the 11 found above the quantitation limit) was 3.84 ng/g lwt.

Medina et al. (2008) describe an analytical method for measurement of PBDEs in human breast adipose tissue. Their study focused on development and validation of the method; limited information was presented on "real" samples they obtained and measured. They measured BDE congeners in 15 samples taken in Valencia, Spain, and found monoBDEs to hexaBDE congeners (BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154) at concentrations ranging from 0.08 to 0.23 ng/g whole weight (percent lipid in the breast adipose tissue not supplied). BDE 183 and BDE 209 could not be quantified because of their low concentrations, but they were detected in 2 of 15 samples.

5.2.4. Selection of Representative Body Burden Profiles

In Chapter 4 (see Section 4.8), exposure media concentrations were assigned for a select group of PBDE congeners in order to conduct the exposure assessment. Specifically, these

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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). 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 (where occupational exposures pertain to elevated concentrations found in individuals working in facilities producing PBDEs or in electronic recycling facilities; not involved in everyday office work); 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 in Figure 5-3 were from Sjodin et al. (2008). Of all the U.S. blood data available, this evaluation of NHANES 2003/4 was the most representative of recent, national trends. Geometric mean concentrations were provided for BDE 28, BDE 47, BDE 99, BDE 100, and BDE 153, but not for BDE 17, BDE 66, BDE 85, BDE 154, and BDE 183, because these were only quantified in 40% of the samples. Arithmetic means were not provided in Sjodin et al. (2008). The median of BDE 99 was reported as less than detection limit, while the geometric mean was reported for BDE 99. There were five studies on PBDEs on U.S. human milk that could be used to represent background conditions. These included two by public interest research groups—the EWG (Lunder and Sharp, 2004) and the 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 had a large sample size. The EWG study included 20 samples from around the United States, so it had broad coverage, but it also had the highest concentrations of any U.S. study on human milk. The NEW study represented several states in the Northwest, had a sample size of 40, and its concentrations track well with EWG, but they were consistently lower. The Schecter et al. (2003) data on 47 samples were all from Texas. Their data also track with the two studies, but it was the lowest of all the data and

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Table 5-3. Representative body burden levels of PBDEs in Americans

Description	Blood[a] (ng/g lwt)	Mother's milk[b] (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

[a]Blood data from Sjodin et al. (2008). These were geometric means from NHANES.
[b]Mother's milk data from NEW (2004). These were medians from a limited opportunistic sample

NA = not available.

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 was used to characterize human milk; it is displayed in Table 5-3. It was chosen because it represented several states, it had as large a sample size as the others, and the concentrations were consistent among the U.S. 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

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total concentration was 97 ng/g lwt. Because geometric means were chosen to represent blood concentrations, the most analogous statistical representation of human 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 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.

Johnson-Restrepo and Kannan (2009) conducted a comprehensive intake assessment for the United States that was structured very similarly to the analysis in this report, using some of the same data. They combined dietary intake factors with food data from Schecter et al. (2006b) to derive intakes from food. They conducted their own analysis of indoor air and dust from homes in Albany, NY, and combined these results with contact rates to derive estimates for inhalation, dust ingestion, and dust dermal contact. They combined breast milk concentrations from Johnson-Restropo et al. (2007) with infant breast milk ingestion rates to estimate exposure to infants from this pathway. They also estimated intake rates for toddlers, children, and teenagers. While they concluded that the dust-related pathways dominated exposures for children and adults, as did this study, their intake estimates were lower mainly because their dust concentrations, at 1,910 ng total/g dust, were lower than the concentrations used in this assessment, 8,275 ng total/g dust. Total intakes from all pathways assessed were, in units of ng total/kg-day: 86.4 for infants, 13.3 for toddlers, 5.3 for children, 3.5 for teenagers, and 2.9 for adults.

As discussed in Chapter 4, studies have shown house dust to contain much higher concentrations of PBDEs compared to outdoor soils. Subsequently, the "soil ingestion" pathway

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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		
Total, from dust ingestion/dermal contact, inhalation, and diet, ng/kg-day	86.4 for infants 13.3 for toddlers 5.3 for children 3.5 for teenagers 2.9 for adults	Johnson-Restropo and Kannan (2009). They combined air/dust measurements from homes in Albany, NY with previously published food/breast milk to develop comprehensive intake estimates.
Dust ingestion plus dust dermal contact for BDE 209 only; ng/kg-day	Mean & 95th percentile: Child—6.0 and 21.0 Adult—0.66 and 2.4	Petito Boyce et al. (2009); probabilistic using data from several U.S. studies for dust concentration distribution; distribution and point estimates for other exposure parameters
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. (2008b); measurements on hands on 33 individuals; empirical model to estimate dust ingestion
Dust ingestion, ng/d	400 for adults	Sjodin et al. (2004b); 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. (2008a); dust conc. from TX; median & average dust conc.; 50, 200 mg/d dust ingestion for adults, toddlers
Diet, Dust, Inhalation, ng/d	155 for adults; 264 for toddlers (4 mos−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

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Table 5-4. Estimates of general population intake or exposure dose of total PBDEs provided in the literature (continued)

Pathway/units	Exposure dose/target individual	Reference/comment
Diet, ng/kg-d	<1 for children/adults for fish; 0.04−20 for children/adults for beef/chicken	Luksembourg et al. (2004); used market basket survey data on fish, beef, chicken from Northern CA
Catfish, ng/kg-d	0.17−1.05; avg. and 95th fish consumption of 3.6 and 21.7 g/day	Staskal et al. (2008); catfish consumption using measured concentrations of 43 congeners, 61 fish
Diet + dust + air, ng/day	67−474 ng/day for adults, 227−886 ng/day for children	Allen et al. (2007); high and low in range based on different dust ingestion rates; total includes BDE 209
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
Total dose (i.e., all pathways), ng/kg-d for BDE 209 only	1200 for "mid-range" and 340,000 for "upper" exposure estimates	Hays and Pyatt (2006) based on 0.96 and 33.6 ng/g lwt BDE 209 in blood, and backward PK modeling
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 plus dust dermal contact for BDE 209 only; UK only and Europe, ng/kg-day	Mean & 95th percentile: Child/UK—451 and 1154 Child/Europe—2.2 and 8.5 Adult/UK—54 and 140 Adult/Europe—0.26 and 0.96	Petito Boyce et al. (2009); probabilistic using data from several European studies for dust concentration distribution; distribution and point estimates for other exposure parameters
Dust ingestion and inhalation, ng/d	0.9−22 (dust ingested) and 2 (inhaled) for adults; 12−43 (dust ingested) and 0.4 (inhales) 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; did not include BDE 209

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Table 5-4. Estimates of general population intake or exposure dose of total PBDEs provided in the literature (continued)

Pathway/units	Exposure dose/target individual	Reference/comment
Dust ingestion, ng/d	<600; <11,000 for adults <2500; <55,000 for toddlers	Harrad et al. (2008a, b); UK; median & average dust conc.; 50, 200 mg/d ingestion for adults, toddlers; BDE 209 dominates
Inhalation, ng/d	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. (2003); 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	Gevao 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 for adults	Bakker et al. (2008); 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 for adults	FSA (2006); UK, based on total diet survey food samples + consumption data
Diet, ng/kg-d	About 1.2 ng/kg-d for age groups >15; BDE 209 is 1/4 to 1/3 total	Akutsu et al. (2008); Japan; based on total diet survey + consumption data
Diet, ng/d	35 for adults	Voorspoels et al. (2007); Belgium, based on market basket samples + consumption data; includes BDE 209
Fish, species specific, ng/kg-d	0.03 for shrimp, 1.35 for sole + flounder, 5.46 for eels	Bragigand et al. (2006); France; ingestion rate + measured BDE congeners from Seine/Loire Estuaries
Fish, ng/d Nursing, ng/d Inhalation, ng/d	1.7−12.9 for child to adult 48.2 for infants 2.7−9.2 for child to adult	Meng et al. (2007); China; median values for fish intake/inhalation for different ages, 0−1 for nursing infant

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has focused on house 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 listed 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 the U.S. Environmental Protection Agency (EPA)'s *Exposure Factors Handbook* (U.S. EPA, 1997), where Hawley (1985) is cited for using a value of 0.56 mg/day to characterize adult exposure to house dust 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 on 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 1,380 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 that they cite from the literature. Harrad et al. (2008a) 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 estimated a "high" total intake of 228 ng/day, which includes BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209. They also provided 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

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91 ng/day. They also provided estimates for toddlers and adults in the United Kingdom, 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 United States. They continued their work by sampling dust in homes, offices, and cars in the West Midland area in the southern part of the United Kingdom, and then estimated intakes for adults and toddlers assuming different times in the home (72% of the time), office (23.8%), and cars (4.2%) (Harrad et al., 2008b). They found totals comparable to their earlier work, including "median" (meaning they used median concentrations and high and low dust ingestion rates) intakes for adults to be less than 5 ng/day for the sum of tri-hexa BDE congeners and 200–600 ng/day for BDE 209. Median toddler intakes were less than 10 for the tri-hexa congeners and between 600 and 2,400 ng/day for BDE 209. 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 house dust measurements from eight 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.

Unlike these previous studies on dust ingestion intakes of PBDEs which relied on point estimates for intake rates and dust concentrations, Petito Boyce et al. (2009) used a probabilistic exposure framework to estimate ranges of intakes of BDE 209 for young children and adults. They used a lognormal distribution for the BDE 209 in dust, which assumed a geometric mean of 1,325 ng/g and a 95th percentile of 8,495 ng/g. They also assumed a log normal distribution for child soil ingestion assuming 45 and 124 mg/day as geometric mean and 95th percentile, respectively, and for adult soil ingestion, assumed half that of children. They used a combination of point estimates and distributions for other necessary parameters (body weight, fraction of total ingestion that is dust, etc) and to model soil dermal contact. The mean and 95th percentile exposure to BDE 209 via dust ingestion and dermal contact was 0.66 and 2.4 ng/kg-day for adults and 4.8 and 18 ng/kg-day for children. Interestingly, they conducted the same analysis for European and United Kingdom conditions. For Europe, other than the United Kingdom, they found lower intakes, but for the United Kingdom, they found intakes about 100 times higher for both children and adults. While BDE 209 concentrations have generally been found at higher

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levels in the United Kingdom as compared to the United States, this particular result was driven by a finding of two very high concentrations of BDE 209 (both above 500,000 ng/g) found in a study of 30 homes in the United Kingdom. These findings were also found to be high in follow-up sampling, and were retained in the analysis by Petito Boyce et al. (2009).

Similar to the relationship between outdoor soils and house dust, outdoor air concentrations were typically lower than indoor air measurements, and, therefore, literature estimates of inhalation exposures to PBDEs have focused on indoor air concentrations. Allen et al. (2007) provides the most comprehensive evaluation of indoor air exposures to PBDEs. They used their own measurements from 20 urban residences in Boston, MA, measured from January to March 2006. They measured 11 congeners, including BDE 17, BDE 28/33, BDE 47, BDE 49, BDE 66, BDE 85/155, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 209. They used their air measurements, along with data on dust concentrations from Stapleton et al. (2005) and food data from Schecter et al. (2006b), and combined these with exposure factors from the *Exposure Factors Handbook* (U.S. EPA, 1997), to estimate adult and child exposures to air, food, and dust. They estimated total exposures with "mean" dust ingestion rates of 4.16 mg/day for adults and 45 mg/day for children, and "high" dust ingestion rates of 100 mg/day for adults and 200 mg/day for children. Finally, they estimated BDE 209 exposures separate from non-BDE 209 intakes. They estimated total intake (non BDE 209 plus BDE 209) for adults as 67 ng/day using "mean" dust ingestion and 474 ng/day using "high" dust ingestion, with BDE 209 explaining 23% ("mean") and 31% ("high") of these totals. For children (ages 1–5, generally, for assignment of exposure factors), total intakes were 227 ("mean") and 886 ng/day ("high"), with BDE 209 explaining 30% ("mean") and 31% ("high"). They found inhalation exposures to all congeners to be 13% in the "mean" scenario, but much lower at 2% for "high" dust scenario for adults; the influence of air for children was much lower, at less than 2%. BDE 209 tended to be the predominant congener for inhalation exposures, generally explaining about 40% of total inhalation exposures.

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

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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. 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 the 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 inhalation 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 were developed mostly by individuals who also measured food concentrations. Estimates of dose originate from the United States and overseas, with U.S. studies summarized here first. Schecter et al. (2006b) combined their measured average food concentrations with average food consumption rates to calculate average intakes for various age ranges (2–5, 6–11, 12–19, 20-39, 40-59, and $\geq$ 60) and for males and females separately. Total PBDE intakes ranged from about 0.9–1.5 ng/kg body weight/day for males/females above the age of 12 (this is the range of average intakes, derived as noted above over the 4 age ranges, 12-19, 20-39, 40-59, and $\geq$ 60). For ages 2–5 (males and females), the intake was estimated at 2.7 ng/kg-day, and for ages 6–11 (males and females), the intake was 1.8 ng/kg-day. Individual intakes between BDE 47 and BDE 99 were nearly identical for males and females, ranging from 0.4 to 0.7 ng/kg-day after age 12, 0.9–1.5 ng/kg-day 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-day, while a "higher fat meats" consumer had an intake of 0.8 ng/kg-day. 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 three different food markets in Sacramento and El Dorado Hills in

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Northern California (Luksemburg et al., 2004). Using average daily intake by adults and children taken from the *Exposure Factors Handbook* (U.S. 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-day in children and between 0.4 and 10 ng/kg-day in adults. Staskal et al. (2008) estimated the dietary intake of PBDEs from consumption of catfish that were store-bought or wild-caught (see Chapter 4 for discussion of the sampling and measurement of PBDEs in these catfish). Assuming ingestion rates of 3.6 and 21.7 g/day (which they describe as the mean and the 95th percentile rate for consumption of all freshwater finfish in the United States), they derived a range of ingestion of 0.17 to 1.05 ng/kg-day. This was based on the mean total concentration of PBDEs (sum of 43 congeners including BDE 209) in the sample of 61 catfish.

Twelve 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 vegan and omnivorous diets. They estimated a dietary exposure average of 107 ng/day (median = 91 ng/day) using consumption data from the survey. Additionally, 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%

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exposure of 149.0 ng/day for Norway. Without fish, these numbers were about one-third as much, with the mean at 20.0 ng/day and the 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 95th percentile 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 the 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 the 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. Bragigand et al. (2006) measured six BDE congeners (BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154) in several fish species in two major estuaries in France, the Loire and the Seine. Combining ingestion rates (on a 60-kg adult basis) for key species with measured concentrations, they noted total intakes of PBDEs to be 0.03 ng/kg-day for shrimp, 1.35 ng/kg-day for sole and flounder, and 5.46 ng/kg-day for eels.

BDE 209 was not included in any of these dietary intakes and surveys. Instead, the standard suite of BDE congeners (BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154) was included. Four studies were found that included BDE 209 as well: one on fish consumption in China, one in Belgium, one in Japan, and one 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 BDE/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 the limit of quantification in any food sample. The average total adult intake was only 35 ng/day, generally low but in line with other European surveys that did not

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include BDE 209 (Voorspoels et al., 2007). A total dietary survey from Japan included BDE 209 among 36 congeners measured, and it was found at ng/g lwt levels in "fats and oils" (Akutsu et al., 2008). Overall, intake of PBDEs from fish dominated the total dietary intake of PBDEs, which averaged about 1.2 ng/kg-day for age groups above 15 years old. Fish concentrations were dominated by BDE 47 but had concentrations of eight other congeners. BDE 209 in fats and oil explained about 1/4 to 1/3 the total intake for all age groups. Childhood intakes, not including formula or breast milk, totaled 2.27 ng/kg-day for ages 1–6 (BDE 209 was 0.90 ng/kg-day) and 1.56 ng/kg-day for ages 7–14 (BDE 209 was 0.59 ng/kg-day).

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 that did include BDE 209 arrived at substantially different results. The UK 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 (see Section 4.7). 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 that 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 is incomplete because BDE 209 was not considered.

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, multimedia 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 BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154. BDE 209 was excluded due to a lack of data in most media. Their results suggested first that

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100–422 g/day of PBDEs were released into the 470-km^2 modeled area; these emission rates resulted in modeled air concentrations that 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 body weight [bw]). Nearly 100 ng/day of this adult exposure is modeled to come from soil/dust exposures, mainly house 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 1,426 ppt wwt in 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 U.S. conditions.

McDonald (2005) developed a dose estimate starting from body burdens and working backward using 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 BDE Congeners BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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, resulting in half-lives of 3 years for BDE 47, 5.4 years for BDE 99, 2.9 years for BDE 100, 11.7 years for BDE 153, and 5.8 years for BDE 154. The estimated total dose of PBDEs (sum of 5 congeners) was 8.5, 16.0, and 53.6 ng/kg-day 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

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that an intake estimate of 0.4−11 ng/kg-day was derived by combining food concentrations and food intake rates in another U.S. study on food intakes only.

Hays and Pyatt (2006) conducted an evaluation similar to that of McDonald (2005) above for calculating the dose of the commercial product decaBDE, which is tantamount to an analysis of BDE 209, since this congener comprises about 98% of the commercial product. While their analysis focused on infant and child exposures to specific pathways and scenarios, and was based on a forward intake dose methodology, they also provided an estimate of "general environmental exposures" based on blood levels of BDE 209 using the simple backwards PK modeling approach. Based on a limited sampling of individuals in the United States (from Sjodin et al., 2001, reviewed in Section 5.2.1; 12 individuals, age/sex unknown), they assumed a BDE 209 concentration in blood of 0.96 ng/g lwt to represent the median exposure and 33.6 ng/g lwt to represent the upper end (UE) of exposures. They used median/high values for half-life of 6.8 and 3.0 days, and a median/high mixing volume of 25% and 50% body weight (equal to the volume of lipids in an adult). Use of these values results in an "absorbed dose." To derive their "intakes," the amount to which the individual was exposed prior to absorption, they divided their absorbed doses by 0.02 for median and 0.01 for upper end (equal to assuming that 2% and 1% were absorbed). These absorptions pertain to all routes of exposure. In summary, their analysis included different concentrations, different half-lives, different mixing volumes, and different absorption assumptions. In their analysis combining these factors, they found a median intake of 1,200 ng/kg-day and an upper end intake of 340,000 ng/kg-day for BDE 209. These values can be compared to the intakes of BDE 209 derived below for general population exposures, calculated in a forward manner. It is found that they are substantially higher than calculated in this assessment using forward dose modeling. Without providing detail on the methodologies in Section 5.4, the unabsorbed intake of BDE 209 for adults is calculated as about 14 ng/kg-day, nearly 2 orders of magnitude lower than the "median" estimate of Hays and Pyatt (2006) (note: 14 ng/kg-day derived from totals provided in Table 5-6; see section below for more detail on derivation of the intakes on this table). This is a substantial difference and could suggest that the procedures used below to estimate intakes are underestimating exposures to BDE 209 and maybe other congeners. However, other differences could bring the numbers together. The review of literature below suggested higher absorption than the 2% assumed for calculation of the median intake of 1,200 ng/kg-day. For the modeling below, the absorption from dermal exposure to

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BDE 209 was 3% and for ingestion of BDE 209 by dust ingestion was 4%. These two pathways explain over 95% of total exposure in the estimate of 14 ng/kg-day. Also, the literature review led to an assignment of 15 days for the half-life of BDE 209 rather than 6.8 days as assumed by Hays and Pyatt (2006). Changing their assumption of absorption to 3.5% (from 2%) and half-life to 15 days (from 6.8 days) would change their estimate to 316 ng/kg-day (from 1,200 ng/kg-day).

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 *Exposure Factor's Handbook* (U.S. 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 remaining 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 U.S. 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 (BDE 99/47) as a way of understanding the possible sources of exposure to humans. The study evaluated data from numerous studies that 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, BDE 99 and BDE 47 are present at about the same amount, 40% each, with BDE 100 at 7%. The ratio of BDE 99 to BDE 47 in U.S. house dust ranged from 0.5 to 2.0, with an average ratio of about 1.0, which would be consistent with the penta BDE formulation with use information. This suggests

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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, U.S. dust appears consistent with expectations. UK dust showed high levels of BDE 209, which is consistent with the deca formulation. Mother's milk, on the other hand, showed a BDE 99 to BDE 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 from animals and then to exposures from 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-day, dominated by BDE 47 at 169 ng/kg-day, followed by BDE 99 at 49 ng/kg-day, and BDE 100 at 36 ng/kg-day. 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 infants 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. Only one study included a comprehensive evaluation across pathways and age groups, as is done in this report (Johnson-Restrepo and Kannan, 2009). They found intakes of total PBDEs ranged from 2.9 ng/kg-day for adults to 86.4 ng/kg-day for infants (via breast milk).

2. On a body weight basis, adult exposures for the dietary pathway alone in the United States are estimated in the range of 0.5–2.0 ng/kg-day;

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3. Although no U.S. studies have estimated inhalation exposures, estimates for the United Kingdom, Canada, and one study in Kuwait suggest much lower exposures in the range of 0.4–>4.0 ng/day, or approximately 0.006 – >0.06 ng/kg-day ;

4. Adult exposures to PBDEs in dust were estimated to be higher than exposures from dietary or inhalation pathways. Exposure to dust ranged from 3.0 to 6.0 ng/kg-day. These estimates are made based on vacuum dust sampling combined with dust ingestion rates of 100 mg/day;

5. Estimates for individual routes suggested that exposure to dust dominated adult exposure. The majority of studies looking at multiple pathways concluded that dust exposures may dominate. However, there was 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 that looked at dermal impacts). Another study assumed house dust ingestion was <1 mg/day, so that this pathway resulted only in a BDE intake of about 3 ng/day;

6. Child and toddler exposures were higher than adult on a body weight basis (approximately 5 ng/kg-day vs. 2.0 ng/kg-day) and were dominated by dust exposures;

7. Infant exposures were dominated by breast milk ingestion, with body weight-based exposures above 80 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 EPA *Draft Dioxin Reassessment* (U.S. 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 *Draft Dioxin Reassessment*, intakes are defined as the amounts of contaminants crossing the body boundary but not yet absorbed into the bloodstream. 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 in Chapter 4, and Table 5-5 in this chapter provides the contact rates, and for dermal exposure, the full dermal contact algorithm.

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Table 5-5. Exposure pathways and factors for the PBDE intake dose estimate[a]

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 (5,700 cm^2/d for adults) × amount soil adhering to skin (0.07 mg/cm^2) × fraction absorbed through 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 U.S. 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 U.S. 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 U.S. EPA (1997) in units of g/kg bw/d, assumes bw above	49.7	21	33	48.1
Pork ingestion, g/d	Data from U.S. 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 U.S. 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 U.S. 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 U.S. EPA (2003), based on U.S. EPA (2000)	11.6	3	3.8	4.5
Freshwater/marine shellfish, g/d	Data developed in U.S. EPA (2003), based on U.S. EPA (2000)	3.8	1	1.2	1.5

[a]All exposure factors and approaches developed in EPA's *Dioxin Reassessment* (U.S. EPA, 2003), which relied on EPA's *Exposure Factors Handbook* (U.S. EPA, 1997) and USDA (1995).

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

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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 is 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 house 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 house 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 house dust measurements, as though the entire contact from "soil" was, in fact, from "house dust." In contrast to their calculations of dust exposure intakes in the hundreds 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 house 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 house dust and indoor air. The surrogate used to estimate this portion will be "time spent indoors." The *Exposure Factors Handbook* (U.S. EPA, 1997) provides tables on hours/day spent indoors, and for adults, the recommended number of hours/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(house 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. 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 14 PBDEs for which environmental

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Table 5-6. Congener-specific and total adult intake estimates of PBDEs (exposures in units of ng/day)[a]

Exposure pathways	17	28	47	66	85	99	100	138	153	154	183	197	206	209	Total	Fraction
House dust 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
House dust 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	0.00	0.01	0.42	0.00	0.00	0.31	0.05	0.00	0.04	0.02	0.03	0.01	0.12	3.01	4.01	0.01
Dairy ingestion	0.00	0.02	0.88	0.00	0.00	0.65	0.11	0.00	0.08	0.03	0.07	0.01	0.24	6.30	8.40	0.02
Egg ingestion	0.00	0.00	0.15	0.00	0.00	0.30	0.12	0.00	0.12	0.07	0.00	0.00	0.08	0.57	1.41	0.00
Beef ingestion	0.00	0.03	0.45	0.00	0.00	0.50	0.05	0.00	0.15	0.04	0.02	0.00	0.00	0.15	1.39	0.00
Pork ingestion	0.00	0.01	0.48	0.00	0.01	0.51	0.08	0.01	0.11	0.05	0.31	0.15	0.01	0.25	1.97	0.00
Poultry ingestion	0.00	0.01	0.18	0.00	0.00	0.25	0.04	0.00	0.04	0.04	0.04	0.00	0.04	0.14	0.75	0.00
Other meats	0.00	0.01	0.37	0.00	0.00	0.41	0.06	0.01	0.09	0.04	0.17	0.08	0.01	0.19	1.45	0.00
Fresh/marine finfish	0.02	0.12	2.32	0.06	0.01	0.46	0.46	0.01	0.07	0.17	0.01	0.00	0.00	0.09	3.82	0.01
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	0.13	0.54	121.89	1.25	5.49	136.61	54.02	9.85	13.99	9.05	4.20	0.35	0.52	142.19	500.08	
Fraction of total	0.00	0.00	0.24	0.00	0.01	0.27	0.11	0.02	0.03	0.02	0.01	0.00	0.00	0.28		1.00

[a]The sum of dust ingestion and dust dermal contact rounds to 0.90 rather than 0.89, and similarly, the sum of all other pathways rounds to 0.10 rather than 0.09. These rounded fractions, 0.90 and 0.10, were used in the text to describe the difference in dust versus other pathways.

media concentrations were derived in Chapter 4. The total dose to adults was 500 ng/day, or on a body weight basis assuming a 70-kg adult, 7.1 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 90% 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 5.7 below.

2. Prevalence, or Lack Thereof, of Food Exposures: After dust exposures, about 10% 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 house 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 BDE 47, BDE 99, and BDE 209 are about equal at 24%, 27%, and 28%, respectively, followed by BDE 100 at 11%, for a total of 90% among those four congeners. Dust-related exposures dominated for the individual congeners. For BDE 47, BDE 99, and BDE 209, dust ingestion and dust dermal contact explained 83%, 93%, and 91%, respectively. Exposures to BDE 138, BDE 153, and BDE 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 17, BDE 28, BDE 66, BDE 197, and BDE 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

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exemplifies how evaluating both dose and body burden can improve the understanding of 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 (see eq 5-1)

$$\delta C_{BDE}/\delta t = [\ (D_{BDE}(t) \times ABS_{BDE})/BL(t)\ ] - k \times C_{BDE}(t) \qquad (5\text{-}1)$$

where

C_{BDE}	=	the congener-specific lipid-based concentration over time(ng/g lwt)
D_{BDE}	=	the daily dose of BDE (ng/day)
ABS_{BDE}	=	the congener-specific and route-specific absorption fraction
BL(t)	=	the body lipid mass over time (g)
k	=	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:

$$C_{BDE}(t) = C_{BDE}(0) \times e^{(-kt)} + [\ (D_{BDE}\ (t) \times ABS_{BDE})\ /\ BL(t)\ ] \times [(1 - e^{-kt})/k] \qquad (5\text{-}2)$$

where

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

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

$$C_{BDE} = (D_{BDE} \times ABS_{BDE})/(k \times BL) \qquad (5\text{-}3)$$

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Equation 5-3 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 (National Institutes of Standards and Technology 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 hours 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, BDE 99, BDE 138, BDE 153, BDE 154, and BDE 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 elsewhere, these values were used in the modeling of this study for congener-specific values of acrylonitrile butadiene styrene. McDonald (2005) assumed absorption fractions of 0.78 to 0.94 for BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154 based on experiments with rats, and the values he used were assumed for these congeners for all other pathways in this study, including inhalation and water/food ingestion. There was no additional absorption assumed for dermal contact because 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 years to this same group of congeners, and these values were used here. Thuresson et al. (2006) provided estimates of 0.26 years (94 days) for BDE 183 and 0.041 year (15 days) for BDE 209, and these were used here. The first-order elimination rates, k_{BDE}, were calculated as 0.693/half-life. For lack of better information, average values of absorption and half-life, 0.90 and 6 years, respectively, were assigned to congeners BDE 28 and BDE 138, two other congeners that are present in exposure media and in measurements of blood or milk. BDE Congeners BDE 17, BDE 66, BDE 85,

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BDE 197, and BDE 206 were 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 eq 5-3 above, and then the observed concentrations of the nine PBDE congeners are shown in Table 5-7.

Overall, the total concentration was predicted at 31.0 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 five of the eight congeners (for which there are predictions and at least one observation). BDE 209 is compared only to a breast milk concentration from Table 5-7, but as noted below, the prediction is much lower than what has been found in blood in NHANES. The prediction of BDE 47 at 8.6 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 12.5 ng/g lwt, while it was found at 5.0 and 5.4 ng/g lwt in blood and milk, respectively. Otherwise, for example, BDE 100 was predicted at 3.9 ng/g lwt, while it was found at 3.9 and 5.2 ng/g lwt. BDE 153 was predicted at 3.9 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 5.7. 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 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 45 ng/day or 0.6 ng/kg-day with a body weight of 70 kg), the body burden is modeled to be 3.7 ng/g lwt, which is much less than the observed medians of 36 and 44 ng/g lwt in blood and human milk. With 27.3 ng/g lwt predicted to occur from soil/dust ingestion, dermal contact, and inhalation, the suggestion is that 88% or more of the U.S. body burden could be explained by nonfood exposures occurring in the indoor environment.

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Table 5-7. Pharmacokinetic parameters and predicted concentrations of BDEs in adults compared with measurements in blood and milk

Exposure doses/parameters/results	28	47	99	100	138	153	154	183	209	Total
I. Doses										
House dust ingestion, ng/d	0.00	81.25	102.92	39.86	7.92	10.67	6.86	2.86	104.83	357.17
House dust 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	0.22	18.98	7.98	4.39	0.03	0.70	0.46	0.66	10.75	44.17
II. Parameters										
House 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	0.1	8.6	12.5	3.9	1.3	3.9	0.6	<0.1	<0.1	31.0
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

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As currently structured and parameterized, the model may be inappropriate for the modeling of the accumulation of BDE 209. The calculated intake dose is 28% of the total, equal essentially to intakes of BDE 47 and BDE 99 (calculated at 24% and 27%, 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. This is numerically close to the observed 0.4 ng/g lwt in human milk, but it is also 1/8 as much. Some blood studies other than NHANES quantified BDE 209 in blood, also, at around 0.1 ng/g lwt. While NHANES did not quantify BDE 209 in individual blood samples, Sjodin et al. (2008) note that the concentration of BDE 209 from pooled NHANES 2001/2002 samples was 2 ng/g lwt (unpublished data; no further details supplied). PSR (2009) quantified BDE 209 in 10 of 20 samples, with a range of 3.9–9.0 ng/g lwt. The observation that this pooled blood sample BDE 209 concentration of 2 ng/g lwt, or the PSR (2009) findings, are higher than the human milk lipid concentration of 0.4 ng/g lwt is consistent with another key study (Schecter et al., 2006c) showing a similar disparity in paired samples between human milk and blood (samples of milk and blood from lactating mothers; see discussion above in Section 5.2.1). In general, this suggests that the model is underpredicting BDE 209 in the body. This could be due to an inappropriate assignment of the elimination half-life, or it could be due to an underestimation of BDE 209 intakes. In summary, the literature has shown mostly nondetects for BDE 209 when measured, and in the range of 1–10 ng/g lwt when found. In contrast, BDE 47 and BDE 99 are most often quantified in 90% or more of survey samples, with concentrations ranging higher than 20 ng/g lwt.

However, to observe that BDE 209 has not been quantified at concentrations comparable to that of BDE 47 and BDE 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, although there is no evidence that the presence of BDE 47 and BDE 99 (and other lower-brominated congeners) is the result of the debromination of BDE 209. Also, it may be true 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.

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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: (1) provides evidence for exposure to the fetus (umbilical cord blood and placenta measurements, mainly); (2) derives intake estimates and resulting body burden estimates for infants; (3) derives intake estimates for children within specific age ranges; (4) discusses intakes of BDE 209 derived for EPA's Voluntary Children's Chemical Evaluation Program (VCCEP) and by the National Academies of Science (NAS) using methods different than used in this study; (5) summarizes body burdens pertaining to occupational populations; and (6) discusses an important observation that there appears to be a proportion of individuals at the high end of the general population who experiences significantly higher exposures than the remaining general population.

5.6.1. Body Burden Data to Characterize Exposure to the Fetus

The most definitive study showing exposure of the fetus to PBDEs was a study in which early to mid-gestation fetal liver (n = 52) and placental tissue (n = 60) samples were obtained from elective abortions between 1998 and 2006 in Montreal, Canada, and measured for BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183 (Doucet et al., 2009). A clear rise in fetal liver tissue was seen over time—total PBDE concentrations (sum of the seven congeners) averaged 284 ng/g lwt in 1998 and 1,608 ng/g lwt in 2006. Placental tissue concentrations were generally lower than fetal tissue, but mean annual concentrations (4–8 samples/year) were over 1,000 ng/g lwt for 3 of the 9 years (data presented only in graphs). The total concentrations were dominated by BDE 99 (at 42–45% of total) and BDE 47 (30–36%) in both liver and placenta, leading the authors to observe that these percentages were similar to the pentaBDE formulation, DE-71.

A second study included measurements of PBDEs in tissues of infants that had been stillborn or that died shortly after birth. Schecter et al. (2007) measured levels in fetal liver tissue samples that were obtained from four stillborn infants and seven 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 = 1/2 DL, the total went up to 23.1 ng/g lwt, owing mainly to an average of 3.5 ng/g lwt (ND = 1/2 DL) for BDE 209. The congener profile was

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fairly similar to blood and human milk profiles, being dominated by BDE 47 at 55% of the total, BDE 99 at 23%, and BDE 100 and BDE 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. The 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. While there are no data on the levels of PBDEs in the mothers, these levels are consistent with body burdens in the U.S. general population as described earlier (Sjodin et al., 2008).

Several studies include measurements of PBDEs in umbilical cord blood serum. Herbstman et al. (2007) collected umbilical cord blood samples from 297 singleton births from 11/26/04 to 3/16/05 from Johns Hopkins Hospital in Baltimore, MD. Although limited geographically, this was a large number of samples, and a comparison is made here with NHANES results for 2003/2004 for the general adult population (Sjodin et al., 2008). Table 5-8 shows the comparison, which is of data provided in the literature articles and includes limits of detection, percent above limit of detection, and median concentrations for congeners where this information is reported in the two articles. There does appear to be a difference, although statistical tests were not applied to determine if the difference is statistically significant, because the detailed data were unavailable but also because statistical testing may not be appropriate since the matrices and study designs are different. But from viewing the table, it is seen that percent detected is lower for all congeners in umbilical cord blood as compared to NHANES, and the median concentrations are also all lower in umbilical cord blood. For example, BDE 47 was 19.2 ng/g lwt in NHANES but 13.6 from the umbilical cord blood, BDE 153 was 4.8 in NHANES and 2.6 ng/g lwt in umbilical cord blood. Since the same laboratory, the Centers for Disease Control and Prevention (CDC) laboratory, performed the analysis, this difference was not likely to be due to a difference in analytical capabilities. Again, while limited geographically, this umbilical cord blood data suggest that the exposure to fetuses may be less than the exposure to mothers.

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Table 5-8. Comparison of median congener concentrations from NHANES and median congener concentrations from a large study of umbilical cord blood measurements of PBDEs[a]

	BDE congener number					
	28	**47**	**85**	**99**	**100**	**153**
I. NHANES						
DL, ng/g lwt	0.8	1.0	2.4	5.0	1.4	2.2
Percent detected	79	97	23	65	93	93
Median, ng/g lwt	1.1	19.2	<LOD	<LOD	3.6	4.8
II. Umbilical Cord Blood						
DL, ng/g lwt	1.1	1.3	1.3	2.1	1.0	1.3
Percent detected	30	90	15	47	64	60
Median, ng/g lwt	0.9[b]	13.6	1.1[b]	4.3[b]	2.3	2.6

[a]The NHANES data are from Sjodin et al. (2008) and the umbilical cord blood measurements are from Herbstman et al. (2007). The detection limits (DL) are described as "maximum LOD" in Sjodin et al. (2008), and as the "median LOD" in Herbstman et al. (2007).
[b]Medians are provided in Herbstman et al. (2007) for these congeners despite the fact that over half were below LOD. No explanation for how medians were developed was provided in Herbstman et al. (2007).

Data further supporting the hypothesis that the fetus is less exposed than the mother can be found in Takasuga et al. (2006), where nine congeners (BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 183, BDE 197, BDE 207, and BDE 209) were measured in maternal blood, umbilical cord serum, placenta, and milk from 10 mothers in Japan. In mother's blood, median congener concentrations ranged from a low of 0.08 ng/g lwt for BDE 183 (found in only two samples) to a high of 1.7 ng/g lwt for BDE 209. Concentrations in mother's milk were comparable to mother's blood for BDE 28 through BDE 183, but they were lower in milk for BDE 197 (0.32 ng/g lipid in blood versus 0.17 ng/g lipid in milk), BDE 207 (0.45 ng/g lipid in blood versus 0.12 ng/g lipid in milk), and BDE 209 (1.7 ng/g lipid in blood versus 0.37 ng/g lipid in milk), leading the authors to conclude that the transfer from blood to mother's milk was lower for these higher-brominated congeners. All congeners were quantified in the placenta (with BDE 183 again found in only 2 of 10 samples) at concentrations that were about one-quarter to one-seventh that of the blood. All concentrations were lower than the detection limit (which was not provided) for umbilical cord serum. The lower concentrations in placenta, like the lower

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concentrations seen in umbilical cord blood above, may indicate lower exposure to the fetus as compared to the mother.

Further evidence showing a difference in maternal and umbilical cord blood for higher-brominated congeners was seen in a Swedish study (Guvenuis et al., 2003). Samples of maternal blood plasma, umbilical cord blood plasma, and milk were taken in 2000–2001 from 15 mothers living in Stockholm. They were opportunistic samples from women between 28 and 38 years old. Ten BDEs were measured, including BDE 17, BDE 28, BDE 47, BDE 66, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183. The median and range for total concentrations (sum of congeners measured) from the three matrices, in ng/g lwt, were as follows: maternal blood—2.1 and (0.7–8.4); umbilical 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 milk, 31–61% in maternal blood, and 45–94% in umbilical cord blood), followed by BDE 153, BDE 99, and BDE 100. BDE 47 concentrations were comparable in maternal blood and umbilical cord blood, but the levels of BDE 153, BDE 99, and BDE 100 were higher in maternal blood as compared to umbilical cord blood (e.g., BDE 153 at 0.56 ng/g lwt in maternal blood but only 0.17 ng/g lwt in umbilical cord blood). The authors suggested this might indicate that the higher-brominated congeners do not pass through the placenta to the same extent as do the lower-brominated congeners. Also noteworthy in this study is the fact that maternal blood and maternal milk samples had very similar concentrations for all congeners. The highest brominated congener, BDE 183, which is a hepta congener, possibly did not partition as much into milk as blood: the median and range in blood and milk, respectively, are 0.06 (0.01–0.44) ng/g lwt and 0.01 (<0.01–0.14). However, all other congeners showed similar median and ranges. This supports the hypothesis that the BDE congeners partition into lipids of the body, at least through the hexa congeners.

Sampling of umbilical cord and maternal blood, as well as mother's milk, in Canada suggests somewhat lower levels in umbilical cord as compared to maternal blood. Ryan and Oostdam (2004) collected small amounts of individual maternal and umbilical cord blood samples from the Northwest Territories of Canada that were part of a "Northern Contaminants Program," composited them, and measured the composites for congeners including BDE 28, BDE 47, BDE 85, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183. These samples pertained to years 1994 to 1999. In 10 maternal composite samples, the mean total PBDE

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concentration was 23.3 ng/g lwt, with a range of 13.1 to 46.5 ng/g lwt, and the umbilical 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 umbilical cord blood, BDE 47 comprised about 77%.

However, other data show umbilical cord blood concentrations of PBDEs to be similar, if not even higher, than maternal blood. Gomara et al. (2007) report on the sampling of PBDEs in human umbilical cord serum, maternal and paternal serum, placenta, and 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 that ranged narrowly between 9.7 and 12 ng total PBDE/g lwt. BDE 47 was the predominant congener in the serum samples. Gomara et al. (2007) found that there were no statistical differences between maternal and paternal blood concentrations but did find differences between maternal blood versus umbilical cord blood and placental concentrations. Unlike the discussion above on umbilical cord blood and NHANES in the United States, Gomara et al. (2007) found comparable if not slightly higher concentrations in umbilical cord blood. They found significantly lower concentrations in milk and placenta, as compared to the blood samples. For example, they found a range of medians between the two locations, between paternal, maternal, and umbilical cord blood of BDE 47 of 2.3 and 3.3 ng/g lwt, but median concentrations of 0.22 to 0.37 ng/g lwt in placenta (one location) and milk (from two locations). Interestingly, BDE 209 was highest in the breast milk samples as compared to all other matrices: the median concentrations in the two locations were 2.8 and 2.9 ng/g lwt, compared to concentrations around 1.1 ng/g lwt for maternal/paternal blood and placenta, and 2.2 ng/g lwt for umbilical cord blood. PBDE median totals were 5.5 and 6.1 ng/g lwt for the two sites and 1.8 ng/g lwt for placentas measured at one site.

Twelve paired samples of maternal and umbilical cord blood were obtained from a hospital in Indianapolis during August–December 2001 and analyzed for BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183 (Mazdai et al., 2003). Results for maternal and umbilical cord blood were essentially identical: the range and median of total PBDE for mother's blood were 15 to 480 ng/g lwt and 37 ng/g lwt, respectively, and the corresponding range and

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median for infant blood were 14 to 460 ng/g lwt and 39 ng/g lwt, respectively. BDE 47 accounted for 53−64% of total PBDEs; BDE 99, BDE 100, and BDE 153 each contributed 10−15% or total. BDE 154 and BDE 183 were found rarely and at low levels. There was no age or body mass index relationship with total BDEs.

Fukata et al. (2005) measured 27 congeners including the key ones (BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, BDE 183, and BDE 209) in umbilical cord tissue, maternal blood serum, and umbilical 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 because there was insufficient volume). Umbilical cord tissue samples were uniformly lower for all congeners as compared to umbilical cord and maternal serums, 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 tissues A and B, respectively, 35 and 18 ng/g lwt in umbilical cord serums A and B, respectively, and 20 ng/g lwt in maternal serum from Pool B. BDE 209 was not detected in umbilical cord tissue, but it was found at 23 and 10 ng/g lwt in umbilical cord serum and 10 ng/g lwt in maternal serum. PCBs and chlorinated dibenzodioxins and dibenzofurans (CDD/Fs) had a slightly different trend. While umbilical cord tissue was always lower than umbilical cord serum (like PBDEs), they were close in magnitude and in fact, maternal serum was significantly higher than either umbilical cord tissue or umbilical cord serum.

Bi et al. (2006) measured the concentrations of seven congeners (BDE 28, BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183) in 69 total samples including 21 paired maternal and fetal serum (for a total of 42 samples) and 27 milk samples (of which 11 were within the 21 individuals providing paired blood samples) in China. They showed a correlation between maternal and fetal blood samples ($r = 0.55, p < 0.05$), with a median total concentration of 3.9 ng/g lwt (range: 1.5−12) in fetal serum and 4.4 ng/g lwt (1.6−17) in maternal serum. Milk concentrations were comparable though higher than either matrix, at a median of 9.0 ng/g lwt and a range of 0.27 to 27.0 ng/g lwt.

One study showed similar concentrations in umbilical cord blood sera and 4 year-old children. The samples were obtained from a large cohort of 470 children who provided data up until the age of 4 in Menorca Island in Spain (Carrizo et al., 2007). At the time of sample measurement, 92 samples were available from a subset of this cohort who were newborn and 244 from children age 4. The congeners measured included BDE 17, BDE 28, BDE 47, BDE 66,

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BDE 71, BDE 85, BDE 99, BDE 199, BDE 138, BDE 153, BDE 154, BDE 183, and BDE 190. The average total concentration was 6.2 ng/g lwt in the umbilical cord blood sera compared to 4.3 ng/g lwt in the sera of the 4-year olds. BDE 47 was the most abundant congener found, averaging 2.8 ng/g lwt in the umbilical cord blood and 2.8 ng/g lwt in the sera of the 4-year olds, followed by BDE 99 at 1.3 and 1.2 ng/g lwt, respectively.

This section has reviewed studies that demonstrate that the fetus is exposed to polybrominated diphenyl ethers passed on by the mother. Concentrations in fetal liver and placental tissue samples from early to mid-gestation aborted fetuses showed concentrations in the hundreds to thousands of ng/g lwt total PBDEs (seven congeners up to BDE 183; Doucet et al., 2009). A second study including fetal tissues from stillborns or from infants who died shortly after birth before feedings showed concentrations comparable to national blood measurements in the United States (Schecter et al., 2007). In some paired studies including both maternal and umbilical cord serum, there is a suggestion that the fetus may be less exposed than the mother, as lower concentrations of PBDEs in umbilical cord as compared to maternal serum were found (Takasuga et al., 2006; Guvenuis et al., 2003; Ryan and Oostdam, 2004). This trend of lower concentrations was seen mostly for higher-brominated congeners in two of these studies (Takasuga et al., 2006; Guvenuis et al., 2003). However, other studies showed similar, if not slightly higher, concentrations in umbilical cord (Gomara et al., 2007; Mazdai et al., 2003; Fukata et al., 2005; Bi et al., 2006). One study showed similar concentrations in umbilical cord blood sera and sera for 4-year olds (Carrizo et al., 2007). In a large-scale study measurement of 297 umbilical cord blood samples in Baltimore, MD (Herbstman et al., 2007), all congener concentrations found were uniformly lower as compared to national U.S. blood measurements from NHANES (Sjodin et al., 2008), and the frequency of detection was also lower. Since the same U.S. laboratory, the CDC laboratory, analyzed the samples in both major studies, this would not be due to different laboratories doing the work. This may be a reflection of a general trend (i.e., lower umbilical cord blood concentrations as compared to general population concentrations), but it could reflect the fact that the Baltimore study was regional, while NHANES was national in scope.

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5.6.2. 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:

$$D = C \times f \times IR \tag{5-4}$$

where

D = ingested dose of PBDEs (ng/day)
C = concentration in milk fat (ng/g lwt)
f = fraction of fat in breast milk
IR = ingestion rate of breast milk (g whole weight/day)

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/day and 723 to 751 mL/day, 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/day (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 5-3), 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 of the 307 ng/kg-day estimate 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

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eq 5-2 above, shown in eq 5-3, 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-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) in infants was driven by the nonmetabolic process of fecal elimination. Specifically, the overall elimination half-life of 2,3,7,8-TCDD was modeled to be about 0.4 years 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 initial, 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* (U.S. 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 1 year of breast feeding, followed by child intakes from 1 year on (see Section 5.6.2 below). Table 5-9 shows the final PK parameters, including dose estimates, from birth until age 19. Figure 5-2 shows the final

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Table 5-9. 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 mo	0.50	0.16	4.3	1411
2 mo	0.60	0.18	4.6	1411
3 mo	0.70	0.20	6	1411
4 mo	0.75	0.22	6.7	1411
5 mo	0.80	0.23	7.4	1411
6 mo	0.85	0.25	7.9	1411
7 mo	0.90	0.25	8.4	1411
8 mo	0.95	0.24	8.8	1411
9 mo	1.00	0.24	9.2	1411
10 mo	1.05	0.23	9.4	1411
11 mo	1.10	0.23	9.8	1411
12 mo	1.15	0.23	11.3	1411
1 yr, 3 mo	1.30	0.22	11.7	709
1 yr, 6 mo	1.50	0.21	12.5	709
1 yr, 9 mo	1.70	0.20	12.9	709
2 yr	2.00	0.20	13.3	709
3 yr	2.50	0.18	15.6	709
4 yr	3.00	0.16	17.6	709
5 yr	3.50	0.15	19.7	709
6–11 yr[a]	4.00–6.00	0.15–0.13	24–41	389
12–19 yr[a]	6.00	0.13–0.15	41–64	484

[a]The time increments of calculation for PK modeling were 1 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.

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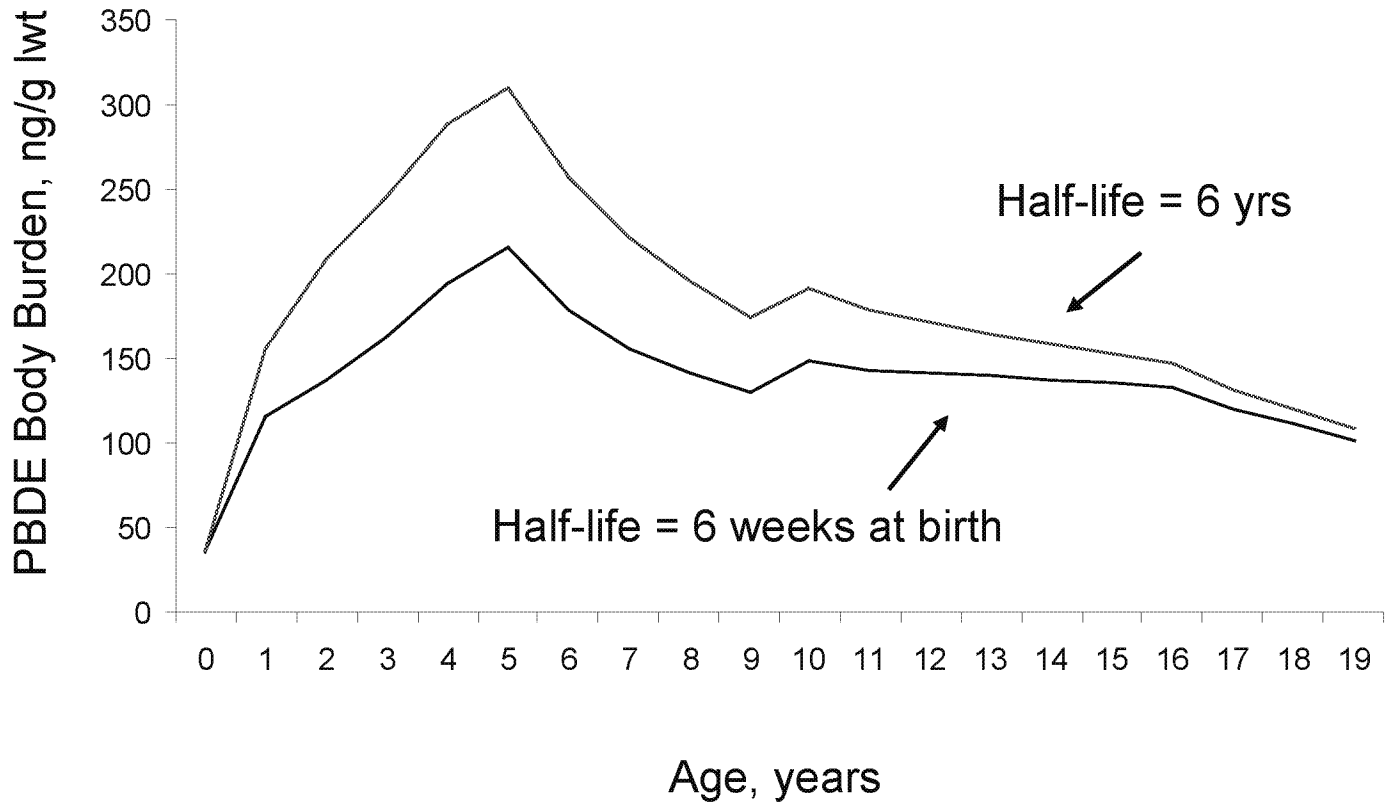

Figure 5-2. Modeled infant and childhood body burdens of PBDEs.

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 115 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 years from birth on, then the body burden would rise to near 150 ng/g lwt by age 1 and continue to rise to more than 300 ng/g lwt by age 5, only then to dissipate slowly to levels near 100 ng/g lwt by age 19.

Two other studies in the literature provide comparable approaches to modeling the breast milk exposure pathway for children. Toms et al. (2008) modeled a lifetime of exposures and resulting body burden concentrations for individuals in Australia, starting from breast milk ingestion at birth, followed by childhood and adult exposures, including food, air, and dust

pathways. Their breast milk pathway included a similar ingestion rate (788 g/day), a slightly different exposure scenario (6 months exclusive breast feeding, followed by breast plus formula feeding up to 1 year), the use of measured mother's milk concentrations (which also equaled the infant starting body burden at birth), and the use of 1-month time step to incorporate growth of the infant. They modeled infant body burdens using the adult elimination rates, while the analysis here also evaluated a more rapid elimination rate based on the mechanics of lipid elimination in the infant. Their rate of ingestion of total PBDEs was 290 ng/day, compared to 1,411 ng/day assumed in this study. Their results, like these here, show a rapid rise in infant body burden through about a year to decline thereafter. Infant body burdens were modeled to rise to about 29 ng/g lwt at Year 1, while the infant concentration in this assessment was modeled to rise to about 115 ng/g lipid.

Hays and Pyatt (2006) also modeled a breast milk pathway but for different purposes, and they did not include a module to predict infant body burden. Their study mainly looked at intakes of infants and children to decaBDE through ingestion of mother's milk and then through contact with consumer products containing decaBDE. They modeled the breast milk pathway for a mother occupationally exposed either through working in a facility that produces deca (the mother is involved in product formulation), or through working in a facility that disassembles electronics. For each of these two scenarios, they modeled a "mid-range" estimate (ME) in which they assumed a 3-month breast-feeding scenario, and for a second estimate they called "upper end" (UE), they assumed 2 years of breast-feeding. In one industrial scenario, they used limited information on decaBDE in blood from occupationally exposed workers and assumed mother's milk lipid concentration of decaBDE was a fraction of that in blood, based on information that decaBDE would not partition to mother's milk from blood in a 1:1 ratio for the highly brominated decaBDE. The ME assumption for this partitioning factor was 0.1, and the UE assumption was 0.5. In the other scenario, they modeled breast milk concentrations, starting from elevated air concentrations in the industrial setting. They used an air:serum ratio to predict serum concentrations and then the same factor less than 1.0 to predict breast milk concentrations. Their intake calculations assumed ingestion rates of breast milk of 742 g/day for the ME and 980 g/day for the UE. Therefore, they produced a wide range of intake estimates, considering different industrial settings, different blood-to-milk partitioning factors, different times of breast-feeding, and different breast milk ingestion rates. The purpose of their assessment was to

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compare their predicted intakes to a published reference dose (RfD) of 4 mg/kg-day (NAS, 2000). When modeling breast milk concentrations starting from air concentrations, the intake doses were 80,000 ng/day for the ME assumptions and 2,700,000 ng/day for UE, and when modeling based on measured occupational blood measurements of BDE 209, their intake doses were 14 and 200 ng/day. These compare with 13 ng/day BDE 209 in this study. The latter two estimates from Hays and Pyatt (2006) are comparable to the estimate in this study because of comparable breast milk ingestion rates and comparable breast milk concentrations. They started with occupational blood measurements of 9.9 (UE) and 4.8 (ME) ng/g lwt, and when extrapolated to breast milk using their partition factors of 0.5 (applied to 9.9) and 0.1 (applied to 4.8), they predicted breast milk concentrations that were similar to the background breast milk concentration of BDE 209 of 0.4 ng/g lwt used in this study (see Table 5-3).

The validity of the predictions of infant body burdens of BDE congeners presented above cannot be easily verified because of the lack of data in the literature. However, there are two studies described above in Section 5.2 on blood levels of infants and younger children. In one, data are presented on four individuals within a family in California: the two parents, 35- and 37-year olds, a 5-year old daughter, and an 18-month old son sampled in September and December of 2004. The sum of BDE 47, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 209 in the parents ranged between 64 and 147 ng/g lwt for 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 five 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 and 12 ng/g lwt (duplicate samples) 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 BDE 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

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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.

The second study was a large-scale pooled blood sampling survey in Australia (Toms et al., 2008). As described in Section 5.2, the highest concentration was found in the youngest age group pooled: 0–4 years old with a mean total PBDE concentration of 73 ng/g lwt, with subsequent lower concentrations for older groups and an average for adults (all age groups above 16 years old) about 15 ng/g lwt. Their modeling of infant and childhood impacts, which showed only about a maximum of 29 ng/g lwt at age 1, was noted by the authors to underpredict the observed body burdens of children. A third study included 244 measurements of 4-year old children of a large cohort of 470 children studied on the island of Menorca in Spain (Carrizo et al., 2007). They found higher concentrations in children who had been breast fed as compared to formula fed, and the differences were statistically significant for the congeners found at the highest concentrations, BDE 47 and BDE 99. The average total concentration (sum of 13 congeners not including BDE 209) in the 4-year olds that had been breast fed (n = 202) was 3.6 ng/g lwt, compared to 1.3 ng/g lwt for formula fed (n = 44).

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 these observations provide some evidence that body burdens in very young children are higher than adults, with the suggestion that they could be very high due to consumption of breast milk. Further, the analysis here suggests that these higher levels could persist throughout childhood into early adulthood.

5.6.3. Childhood Intakes

The total dose to adults was 500 ng/day, or 7.1 ng/kg-day (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: 709 ng/day for the 1–5 age range, 389 ng/day for the 6–11 range, and 484 ng/day for the 12–19 age range. On a body weight basis, the doses are 47.2 ng/kg-day for ages 1–5 (assuming 15 kg bw), 13.0 ng/kg-day for 6–11 (assuming 30 kg bw), and 8.3 ng/kg-day for 12–19 (assuming 58 kg bw). The much higher dose for the

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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. One study suggests that these intakes may be low for children. Stapleton et al. (2008b) evaluated PBDEs that are present on hands from 33 volunteers using wipe samples, and using a hand-to-mouth algorithm for children, they estimated a median intake for children (generally defined as 6–13 years old based on parameters selected to model hand-to-mouth intakes) to be 1,390 ng total BDE/day and 95% of 6,090 ng total BDE/day (including 13 BDE congeners up to BDE 209 that comprised roughly 1/5 of total exposures). These numbers were generated using the data from all the participants, and only six of them (ages 8 to 10) could have been characterized as children, and the authors acknowledge this to be a key uncertainty for these intakes for children.

5.6.4. Intake Estimates Derived for EPA's VCCEP and by the NAS

The intake estimates derived for EPA's VCCEP (described in Hays and Pyatt, 2006) and by the National Academies of Science (NAS, 2000) differed from the intake estimates derived in this assessment in two ways: (1) the focus was solely on the commercial product, decaBDE, which essentially is equal to assessing BDE 209 since this congener comprises about 98% of the commercial product, and (2) the purposes of both studies was to provide conservative intake estimates with which to compare to the NAS reference dose of 4 mg/kg-day for decaBDE. Because of this second difference, many of the intakes provided below were significantly higher than intakes estimated in this assessment, by upwards of 5 orders of magnitude.

The VCCEP intake assessment was presented to EPA as part of a larger assessment of decaBDE that included an assessment of toxicity, environmental levels, and many other analyses (VCCEP, 2008). Hays and Pyatt (2006) published this VCCEP assessment of exposure to children in the open literature. They modeled five exposure scenarios pertaining to children ages 0–2: (1) breast milk ingestion from a mother who is involved in the formulation of decaBDE, (2) breast milk ingestion from a mother who is dissembling electronics, (3) mouthing decaBDE-based plastic electronic products, (4) inhaling decaBDE particulates released from plastic electronic products, and (5) mouthing decaBDE-containing fabrics. They modeled a 6th scenario pertaining to all ages, which was a backward PK modeling exercise starting from a

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blood measurement of BDE 209. This exercise was described earlier in Section 5.3. The two breast milk intake estimates were described in Section 5.6.2.

The other three scenarios involving mouthing of products (fabrics and electronics) and inhaling particles released from products (electronics) involved estimating the release from the products and then the exposure by the children. Their highest exposure was from mouthing fabrics, which they used from the NAS (2000) assessment described below. The intake was 26,000 ng/kg-day of decaBDE for this pathway. The general algorithms for mouthing fabrics and mouthing electronics present an alternate to the dust ingestion and dust dermal contact algorithms in this assessment, which are the closest analogous algorithms. The general algorithm for these mouthing algorithms entails an estimate of the total mass of the PBDE in the product per square area, a rate at which it leaves the product or can leave upon mouthing, and then exposure contact parameters. Data to parameterize these approaches are sparse, and the assignments in Hays and Pyatt (2006) and NAS (2000) can lead to very high intake estimates (as will be shown in later discussions). For mouthing electronics, the total intake entails an estimate of the mass of decaBDE leaching from the surface of the product (0.15 mg/day for the "mid-range" estimate and 0.29 mg/day for the "upper" estimate, as generated from a study in the literature), combined with exposure factors including the time spent by the infant mouthing during the day (32 and 97.2 minutes/day) and the fraction of items he/she mouths that is treated (1% and 10%). Their intakes for BDE for this "mouthing" scenario were 4.3 and 250 ng/kg-day. Their inhalation scenario did not entail a release and air dilution algorithm but instead was based on measured concentrations of decaBDE in air. Their inhalation intakes were 0.3 and 0.6 ng/kg-day. In contrast, the dust ingestion, dust dermal contact (not considering absorption across the skin barrier), and inhalation intake estimates for BDE 209 for this assessment for the youngest age category, 1–5 years, were 7.0, 1.7, and 0.1 ng/kg-day (based on an average 15 kg body weight during the Years 1–5). The approach in this assessment was to use background concentrations of BDE congeners in air and house dust and combine these contact fractions to air and dust. It did not consider more specific contact or behaviors associated with treated products. The inhalation estimates in this assessment and the Hays and Pyatt (2006) assessment are similar, because the approaches were the same, both using air concentrations combined with contact fractions. Their mid-range estimate for mouthing of electronic products led to exposures

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similar to the dust pathways in this assessment, but their upper estimate was over an order of magnitude higher.

The NAS study also had different intake estimates for decaBDE as compared to the BDE 209 estimates in this study (NAS, 2000). For dermal exposure to treated fabric, their general model entails first an estimate of release from the treated fabric, followed by consideration of clothing as a barrier to skin transfer, and then an assumption that enough moisture (sweat) is present such that decaBDE can be dissolved and transferred through the skin barrier. A first iteration of their model assumed that the clothing did not prevent a barrier to transfer, and then additionally, full transfer through the skin such that the only limitation to exposure was the rate of release from the treated fabric. Under this extreme example, they estimated an exposure as 980,000 ng/kg-day. When they modeled penetration through the skin (still not considering clothing as a barrier), they estimated an absorbed intake of 0.001 ng/kg-day. As noted above, the dermal contact pathway in this assessment estimates BDE 209 exposures of 1.7 ng/kg-day to dust before absorption, and when considering a 4% absorption rate, this is reduced to 0.07 ng/kg-day. An algorithm to consider sucking of treated fabric includes an area density of treated fabric (mass decaBDE per unit area of cloth), an area of sucking, an extraction rate (a potential amount of mass extracted for a full day of sucking), and an amount of time sucking. The average dose rate using this algorithm was very high at 26,000 ng/kg-day, considerably higher than the 7 ng/kg-day estimated for dust ingestion in this assessment. An inhalation pathway models both particle and vapor phase decaBDE released from treated fabrics. The empirical model is based on area density of treated surfaces, the extent of treated surface within a room, a rate of release of the decaBDE as respirable particles, and the room volume in order to produce a concentration. Finally, a time-weighted average daily concentration considered the amount of time an individual spent in the treated room. The time-weighted average was 480,000 ng/m^3. Similar algorithms were used to estimate a vapor phase concentration, and it was found to be 380 ng/m^3. These modeled concentrations are extremely conservative, as BDE 209 has not been found to exceed 1 ng/m^3 in the nonoccupational setting, and only as high as the hundreds of ng/m^3 in occupational settings (see Section 4.5). In NAS (2000), these predicted concentrations were compared to a Reference Concentration, RfC, determined from their RfD.

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There is some concurrence in the approaches taken by Hays and Pyatt (2006) and NAS (2000) and this assessment. Such concurrence was seen in inhalation exposures based on measured air concentrations, and in the dermal contact pathway when considering the skin as a barrier to absorption. However, mostly the intake algorithms and air concentration models led to substantially higher intakes and concentrations as compared to estimates in this assessment—higher by up to 5 orders of magnitude. Certainly, the objectives of the Hays and Pyatt (2006) and NAS (2000) study were different than here. A key purpose of their assessments was to compare exposure intakes with the NAS (2000)-derived RfD of 4,000,000 ng/kg-day (4 mg/kg-day). There was never any attempt to compare predicted with measured concentrations, nor any attempt to compare intake estimates with others that may have been generated in the literature on similar pathways. Also, there was no attempt to take intakes and model a blood concentration of BDE 209, which could have been used to compare with measured blood concentrations (although there were few, if any, measurements of blood concentrations of BDE 209 for use in the NAS report completed in 2000). While the algorithms used show potential use in assessments of general population exposures, it is felt that the input parameters need to be more carefully assigned values to characterize general population exposures, and the results subjected to some reality or validation testing.

5.6.5. 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 (Lee et al., 2007). 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 (Stapleton et al., 2008a).

The study from the United States 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 composed of spouses and clerical workers from the facilities (Stapleton et al., 2008a). The median total PBDE concentrations (composed of BDE 17, BDE 28, BDE 47, BDE 66, BDE 99, BDE 100, BDE 153, BDE 154, and BDE 183) in the foam workers, carpet installers, and control were 160, 178, and 19 ng/g lipid, respectively.

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The body burdens of the workers were dominated by BDE 47, which explained 50–60% of the total concentration, followed by BDE 99 and BDE 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 (Jakobsson et al., 2002). Moreover, within these two comparison groups, refined characterizations included women who had never breast fed (NBF), hospital cleaners (HCs), and PC clerks (PCCs). Results were provided for BDE 47, BDE 153, BDE 154, BDE 183, and BDE 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 four 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 the start of vacation, 3–4 days later, and then at additional time frames during the 4–5 week vacation. The samples were analyzed for BDE 47, BDE 153, BDE 183, and BDE 209. Assuming there was a constant baseline of BDEs in these workers, which they obtained from serum measurements on nonexposed 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. Individuals not occupationally exposed 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 BDE 47, BDE 153, BDE 183, and BDE 209 averaging 2.9, 4.5, 7.9, and 4.8 ng/g lwt, respectively. While they found that BDE 209 is bioavailable based on finding high

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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 was as follows: 126 ng/g lwt (dismantling workers), 35.1 ng/g lwt (residents living within 50 km of the dismantling area), 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: BDE 28, BDE 183, BDE 208, and BDE 209 were found at 148.3, 60.2, 66.2, and 3,436 ng/g lwt, respectively.

Bi et al. (2007) similarly found elevated levels of PBDEs in residents living near and working within an e-waste recycling facility in South China. They sampled two regions, including this recycling region and a second nearby region, where the fishing industry dominates the livelihood of the region. A total of 21 individuals were sampled within the recycling region and 26 from the nearby fishing region. Sixteen BDE congeners were measured, including BDE 209. Total concentrations from the recycling region ranged from 140 - 8,500 ng/g lwt, with a median of 600 ng/g lwt, while it more narrowly ranged from 16 - 490 ng/g lwt, with a median of 170 ng/g lwt, in the fishing region. BDE 209 dominated the concentrations, with a median of 310 and maximum of 3,100 ng/g lwt in the recycling region and a median of 86 and maximum of 370 ng/g lwt in the fishing region. Octa (BDE 197 and BDE 203) and nona (BDE 206, BDE 207, and BDE 208) congeners averaged from 20 to over 100 ng/g lwt in the serum of the residents from the e-waste recycling region, suggesting possible debromination of BDE 209.

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 in Korea. 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

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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 were 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 BDE 153 (24%), BDE 183 (17%), BDE 99 (15%), and BDE 100 (7%).

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

Table 5-7 shows the analysis of the 2003/2004 NHANES blood concentration data, which was described by Sjodin et al. (2008), used as a primary comparison to the predicted concentration in the model, and did not include statistics on total concentration, just statistics on individual congeners. Table 5-7 shows the congener-specific median concentrations, 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 U.S. citizens are about 4–6 times higher than the 50th percentile and the 95th percentile 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 3,680 ng/g lipid, which included BDE 47 at 2,350 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 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. (2007) statistically evaluated the concentrations of polychlorinated dibenzo-*p*-dioxin (PCDD) and dibenzofuran (PCDF; the combination abbreviated CDD/F) concentrations from NHANES 2001/2002. Determining the TEQ concentrations of the 17 toxic CDD/F congeners for individuals in the survey, they provided population statistics for CDD/F TEQ concentrations. The median concentration in the population was 14.4 pg/g lwt, the 95th percentile was 45.2 ng/g lwt, and the maximum found was 139.2 ng/g lwt. In this case, the maximum found was 10 and not 100 times the median. To illustrate the difference in these NHANES results, Figure 5-3 shows the comparison of NHANES percentiles for CDD/F TEQ and BDE 47 findings at the 25th percentile, 50th percentile, 75th percentile, and 95th percentile, compared to the

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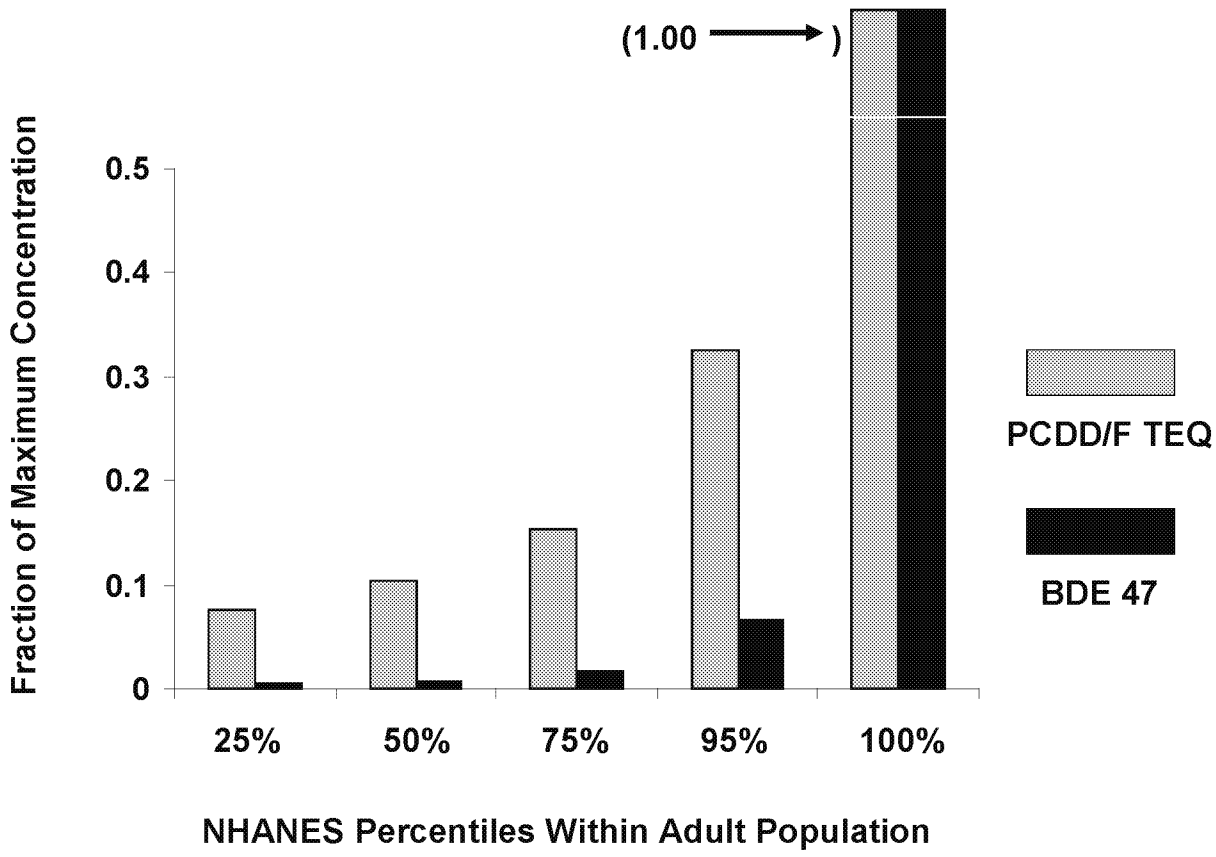

Figure 5-3. The fraction of the maximum concentrations of PCDD/F TEQ and BDE 47 concentrations found at various percentiles within NHANES surveys of these two contaminants in adults.

Note: NHANES data on PBDEs from Sjodin et al. (2008), and data on PCDD/F from Ferriby et al. (2007).

maximum found in the NHANES surveys (dioxin data from Ferriby et al., 2007 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 percentile and 0.30 at the 95th percentile for PCDD/Fs; they range much more narrowly at <0.01 at the 50th percentile 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, Morland 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

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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. Anderson et al. (2008) provides summary information for blood sampling of 350 male and 158 female participants in a study of the relationships between several organic contaminants, including PBDEs, and consumption of Great Lakes sportfish. The geometric mean total concentration (sum of 8 congeners not including BDE 209) for males was 27 ng/g lwt and for females was 25 ng/g lwt. They also showed a figure of the highest 50 concentrations for males and females. For males, the top five ranged from about 380 to 1,300 ng/g lwt (the top two at 1,100 and 1,300 ng/g lwt) and for females, the top five ranged from about 250 to 950 ng/g lwt. 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. The following is a 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 for this discussion was 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 2,000 ng on his hand. The median from this population was 128 ng/hand.

b. Allen et al. (2008a) 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 were 13,732, 6,255, and 4,269 ng/g dry weight (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. Two other 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 2 homes, they found 14,990 and 30,100 ng/g dwt, respectively. These samples were dominated by BDE 47, BDE 99, and BDE 209 in comparable amounts. In the other, Sharp and Lunder (2004) analyzed dust samples in 10 homes from nine states. Concentrations found in eight 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 BDE 47, BDE 99, and BDE 209.

d. Harrad et al. (2008a) 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 1 sample was found at 520,000 ng/g dwt, with essentially all of it (>99%) explained by BDE 209. In another study, Harrad et al. (2008b) sampled dust in 30 homes, 18 offices, and 20 cars (68 total samples), and found 3 samples at 1,400,000, 2,200,000, and 2,600,000 ng/g dwt, essentially all of it being BDE 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 fishermen consuming large amounts of Baltic Sea fish had a median BDE 47 level five times higher than that of nonconsumers. However, only a small statistically insignificant difference was found between urban anglers and nonanglers in a study of 93 individuals (79 anglers, 14 nonanglers) in the United States (Morland et al., 2005). It should also be noted that findings of PBDE body burdens in other U.S. studies are not very different from the NHANES study. As described in Section 5.2., the means or medians from essentially all U.S. 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.

A simple exercise may be able to shed light on the cause of extremely elevated exposures to a small number of individuals at the high end of measured populations in the United States. The implication from discussions above, of course, is that dust-related exposures are the likely cause of such elevated blood levels. But can that be demonstrated, and can food exposures also possibly explain these high levels? Sjodin et al. (2008) note a 95th percentile blood concentration

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of BDE 47 of 157 ng/g lwt in NHANES 2003/2004. With the use of eq 5-3 in Section 5.5 above, one can calculate the dose required under an assumption, for example, that the elevation was due entirely to dust ingestion or food ingestion. Starting with dust ingestion, the dose corresponding to this blood concentration, assuming k of 0.00063 day^{-1} (half-life of 3 years), a body lipid mixing reservoir of 17,500 g, and an absorption of 0.69 (used for BDE 47 absorbed to dust) would be 2,508 ng BDE 47/day, or for a 70-kg adult, 35 ng/kg-day. If it was from food ingestion, the absorption would be higher at 0.94, and the dose would be 1,841 ng BDE 47/day, or 26 ng/kg-day. The key uncertain parameter in this calculation is the elimination rate, which was based on a half-life of 3 years. Had the half-life been 6 years instead of 3 years, the doses would be halved at 18 ng/kg-day for dust and 13 ng/kg-day for food. These backcalculated doses can now be compared to forward calculated doses of BDE 47 from dust and from food. Allen et al. (2008a) found a maximum BDE 47 concentration in dust in Boston of 16,840 ng/g dwt, Harrad et al. (2008a) found a high BDE 47 concentration in Texas dust of 3,300 ng/g dwt, Sharp and Lunder (2004) found a high of 9,070 ng/g dwt in Montana dust, and Rudel et al. (2003) found a high of 9,860 ng/g dwt in Cape Cod dust. If one assumes an ongoing exposure to a "high" concentration of 10,000 ng/g dwt BDE 47 in dust and an average ingestion rate of 50 mg/day, then the dose on a body weight basis would be 7.1 ng/kg-day, and if one then calculates soil dermal contact in the manner done in this report, this would bring the total to 8.9 ng/kg-day. This is below the backcalculated level for hypothetical exposure via dust of 35 (or 18 at a 6-year half-life) ng/kg-day. But for food, the disparity may be larger. Schecter et al. (2006b) found high concentrations of BDE 47 of 387 pg/g wwt in meats, 2,748 pg/g wwt in fish, and 105 pg/g wwt in dairy. If these concentrations are used to calculate food exposures to BDE 47 using the contact rates in this assessment, than the total adult dose would be 115 ng BDE 47/day, or 1.6 ng/kg-day, well below the backcalculated food dose of 26 (or 13) ng/kg-day. Higher exposures would be calculated using higher contact rates, but also, the maximum BDE 47 found in NHANES 2003/2004 is 2,350 ng/g lwt (Sjodin et al., 2008). It appears as if exposures other than food and dust that are not identified or quantified properly in this assessment are affecting a small percentage of individuals at the high end (upper 5%, perhaps) of populations, at least for BDE 47.

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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 about 7 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 near this value of 7 ng/kg-day. The point estimate approach suggested that about 90% 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 ng/kg-day, dominated by dust exposures. Further, it appears that these very high body burdens cannot be explained even by exposures to the highest dust concentrations and food concentrations found.

The point estimate construct of the exercise does not guarantee that the finding of the importance of the dust pathways to mid-range (such as median) body burdens is proven. The purpose of this section is to identify and discuss known uncertainties and variabilities so that this primary finding and other findings in this chapter can be understood in their proper context.

5.7.1. Uncertainties with Estimates of Dust Intakes of PBDE

The amount of 50 mg/day assumed for adult soil/dust ingestion is a classic uncertainty. Described as an "average" value for soil ingestion in the *Exposure Factors Handbook* (U.S. EPA, 1997), the actual amount of ingestion of house dust plus 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 house dust ingestion rate of 0.56 mg/day, over 2 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 house dust was listed in EPA's *Exposure Factors Handbook* (U.S. EPA, 1997),

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which cited Hawley (1985) for using a value of 0.56 mg/day to characterize adult exposure to house dust 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 house dust ingestion, at least in the context of estimating exposure to house 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. The soil/dust ingestion rate of 100 mg/day used in the characterization of children's exposure (ages 1−5) is similarly uncertain. In the recent *Child-Specific Exposure Factors Handbook* (U.S. EPA, 2008), however, the recommended factors and approach for modeling childhood (ages 1−6) exposures to outdoor soil and indoor house dust is really not that much different than used here. Specifically, the current approach, as noted, uses a total soil/dust ingestion rate of 100 mg/day, where the concentration on that matrix is a weighted average of outdoor soil (about 20%) and indoor house dust (about 80%). Similarly, the *Child-Specific Exposure Factors Handbook* recommends a central tendency 60 mg/day of house dust ingestion using, presumably, concentrations on house dust, and 30 mg/day of outdoor soil using, presumably, concentrations in outdoor soil.

The procedure here to characterize ingestion of house dust is to multiply a total amount ingested by a fraction that comes from the house. The assumption of 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 house dust originates from outdoor soil. This may be less important for the current exercise because dust concentrations were taken from house 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 house 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.

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 (see 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.

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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 (U.S. 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 al. (2001) examined the in vitro dermal absorption of [^{14}C] decabromodiphenyl oxide. 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-hour 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 hours 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 hours was 3.13% (1.93% in the receptor solution and 1.2% in the skin). The total absorbed dose in rat skin after 24 hours 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 and Leibold., 2004). All three studies applied the chemical in a solvent that evaporated rapidly, leaving a residue of pure chemical on the skin. This would tend to increase the absorption relative to a similar dose that 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 datum, but may be high for dust exposures. U.S. EPA (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. 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, therefore containing more surface area, 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 BDE 47, BDE 99, BDE 100, BDE 153, and BDE 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 assumed that the absorption fraction of dioxin from a total intake dose was 0.80 (U.S. EPA, 2003), but dioxin intakes are dominated by food—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.

5.7.2. Uncertainties and Variabilities with Other Pathways

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

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intake quantities as developed in the *Exposure Factors Handbook* (U.S. EPA, 1997) were derived from the U.S. Department of Agriculture Continuing Survey of Intakes by Individuals, 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 UK's Food Standards Agency of high BDE 209 in all food sampled (FSA, 2006). The quality of these data are unknown and might be questioned since it differs from essentially all other studies of PBDEs in 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 range of 1−2 ng total BDE/kg-day. Inhalation and water ingestion have mostly arrived at adult exposures of 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. The discussions above on the NAS and VCCEP submissions did, in fact, discuss mouthing of products including treated fabrics, and, also, the direct contact with treated fabrics. The exercises were specific to decaBDE, were conservative by design, but showed much higher exposures than the central tendency estimates presented here (see Section 5.6.4 for more detail). Finally, Stapleton et al.

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(2008b) provides estimates for a direct hand-to-mouth pathway. They estimated the amount of PBDEs on the hands of 33 volunteers using hand wipes, and using hand-to-mouth, estimated intakes for children ranged as high as 6,090 (95%) ng total BDE/day, with a median of 1,390 ng total BDE/day.

The possibility of exposure by direct contact with treated surfaces, or surfaces that could be reservoirs of PBDEs otherwise released from their original sources, is being studied by a novel approach using X-ray fluorescence (XRF). XRF analyzers measure the bromine concentrations on the dust that resides on surfaces of household items. Allen et al. (2008b) validated their use on household items by showing that XRF readings were highly correlated with GC/MS measured bromine and PBDE levels in furnishings and electronic devices ($R = 0.93$). Imm et al. (2009) took this application a step further by measuring bromine concentrations on the surfaces of many household items for a cohort in Wisconsin and then further measuring the PBDEs in blood of residents who lived in the homes they studied. They found that the bromine content in the participant's sleeping pillow (p-value = 0.005) and primary vehicle seat cushion (p-value = 0.03) were the strongest predictors of PBDE blood concentrations.

5.7.3. Uncertainties with Pharmacokinetic Modeling

The choice of the simple one-compartment 1st order model is reasonable for PBDEs. Like dioxins, for which the model has been extensively and successfully used (U.S. EPA, 2003, and other citations not provided), most PBDEs are lipophilic and persistent in the body (there is some evidence and discussion in this report that BDE 209 is not as lipophilic as other BDE congeners, and its elimination half-life is hypothesized to be on the order of days, in contrast to years for lower-brominated BDEs). 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 (U.S. 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. Rather, the younger age ranges were found to

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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 (perhaps because of 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 BDE 47, BDE 99, BDE 100, BDE 153, and BDE 154 of 2.9 to 11.0 year. In addition, only one study was found, Thuresson et al. (2006), which provided estimates for BDE 183 of 0.26 year (94 days) and for BDE 209 of 0.041 year (15 days).

With these selections, it was found that BDE 47 appears to be underpredicted. The prediction was 8.6 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 12.5 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 BDE 47, BDE 99, BDE 100, and BDE 153 were all administered intravenously to mice, Staskal et al. (2006) found that tissue concentrations were highest for BDE 153, followed by BDE 100, BDE 99, and BDE 47. Similar to the human data, these mouse data suggest a more rapid elimination of BDE 47 as compared to BDE 99 and

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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, have 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. 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 31.0 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 that 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 human 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 47, BDE 99, BDE 100, BDE 153, and

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BDE 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-day 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 500 ng/day total, or on a body weight basis assuming a 70-kg adult, 7.1 ng/kg-day. This dose was estimated based on "representative" central tendency media concentrations and average contact rates, so the consistency of 7.1 ng/kg-day derived by a forward calculation for "average" conditions with McDonald's (2005) 8.5 ng/kg-day derived by a retrocalculation from the median body burden is very encouraging.

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, in an application of his PK framework. With his model, McDonald (2005) found quantitative consistency in those two quantities: i.e., the dose he back 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 due to house dust exposures. If not entirely due to house 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 U.S. body burdens is a 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 supports these general findings:

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1. **PBDEs bioaccumulate in lipids**: Most PBDEs bioaccumulate in lipids and typically body burden measurements are expressed in a ng/g lipid wt basis. The finding that PBDEs partition into lipids is upheld for congeners up to the hexa congeners as studies show similar concentrations measured in maternal blood and maternal milk for the commonly measured congeners, BDE 28, BDE 47, BDE 99, BDE 100, BDE 138, BDE 153, and BDE 154. There is some evidence that the hepta congener, BDE 183, and more so, the deca congener, BDE 209, are found at lower concentrations in mother's milk than mother's blood. Concentrations of BDE 209 found in mother's milk in the United States are in the range of 0.5 ng/g lwt when quantified, while it is near 5 ng/g lwt in blood when quantified.

2. **PBDE Body burdens in U.S. adults**: Total PBDE body burdens in Americans are in the range of 30 to 100 ng/g lwt. The most statistically rigorous and expansive study of general population exposures to PBDEs in the United States is an analysis of 2003/2004 NHANES data by Sjodin et al. (2008). The geometric mean congener concentrations over the entire population above the age of 12 were, in ng/g 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. The sum of these geometric means was 36.3 ng/g lwt. The 95th percentile for the total body burden of PBDEs (i.e., sum of these five congeners) was 291 ng/g lwt, and the maximum found was 3,680 ng/g lwt. The predominant congener found in nearly all body burden studies in the United States is BDE 47, explaining about 50% of the total concentration. The second most commonly found congeners are BDE 99 and BDE 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 5 ng/g lwt.

3. **PBDE Body Burdens in other Countries**: Body burdens of Americans are higher than body burdens of individuals in other countries. Body burdens in other countries are less than 10 ng/g lwt. Most non-U.S. data are from Europe.

4. **PBDE Body Burdens in U.S. Children**: Body burden data, as well as intake and body burden impact modeling, suggest that the infant and toddler have higher exposures than older children or adults. In paired sampling studies, including mother and child, the child's body burden has exceeded by the mother's by about a factor of 4, exceeding 100 ng/g lwt total PBDEs in some cases. Higher levels in toddlers were attributed to ingestion of mother's milk and high exposures to house dust.

5. **PBDE Fetal Body Burdens**: Limited data from fetal tissue and numerous studies including measurements of BDE congeners from umbilical cord blood support the conclusion that the fetus is exposed to PBDEs through the mother. In some paired studies including both maternal and umbilical cord serum, there is a suggestion that the fetus may be less exposed than the mother as lower concentrations of BDE congeners in umbilical cord as compared to maternal serum were found. This trend of lower concentrations were seen mostly for higher-brominated congeners in two of these studies,

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where this general observation could be made, but in four other studies reviewed, similar, if not slightly higher, concentrations of all congeners were seen in umbilical cord serum.

6. **High-End Exposures in U.S. Population**: 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 house dust measurement studies, suggesting that dust exposures could explain these unusually high exposures. However, modeling exercises undertaken to examine this hypothesis suggest that even the highest dust concentrations might not be able to explain the highest body burdens found. As such, these very high body burdens remain a critical uncertainty that warrants further investigation.

7. **Occupational Exposure**: Limited occupational data support the observation that individuals in occupations that would lead to higher exposures to specific congeners have higher concentrations of PBDE congeners in their blood than the general population.

8. **PBDE Intake Rates**: 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 house dust, although this has changed in recent years as researchers recognize the importance of house dust in the overall exposure paradigm for PBDEs. Total daily intake estimates derived in this study, based on exposure media concentrations derived in Chapter 4 and combined with average contact rates, were 500 ng/day for adults to 1,411 ng/day for nursing infants. Assuming an average weight of 70 kg, the estimated adult intake rate was 7.1 ng/kg-day. 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 90% of total intakes, with food and water ingestion explaining the remaining 10%.

9. **Intake Rates for Infants and Children**: Intake modeling for the breast milk pathway combining measured milk concentrations and infant ingestion of human milk led to an intake of 1,411 ng/day in this study. 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. It is lower than intake estimates for children derived in this study, which were 47.2 ng/kg-day for ages 1–5 (assuming 15 kg bw), 13.0 ng/kg-day for ages 6–11 (assuming 30 kg bw), and 8.3 ng/kg-day for ages 12–19 (assuming 58 kg bw).

10. **PBDE Dust Intake rates:** Estimates in the open literature for intake of total PBDEs (sum of congeners, with most estimates for dust including BDE 209) via house dust ingestion range from 3 ng/day to 400 ng/day. The latter assumed less than 1 mg/day dust ingestion, while the former assumed a 100 mg/day dust ingestion rate. The adult dust intake rate derived in this study (dust ingestion plus dermal absorption) was 449 ng/day.

11. **PBDE Dietary Intake Rates**: Estimates in the open literature of total PBDE intakes from food ingestion were in the range of 0.5 to 2.0 ng/kg-day for adults.

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12. **Modeling PBDE Body Burdens from Intake Rates**: Using a simple PK model parameterized with available literature values, adult lipid-based concentrations (not specific to blood or milk) were predicted, starting with the intake values derived for adults in this study (see bullet 8 above). On a total PBDE basis, the prediction was low at 31.0 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 in studies selected as representative of the general population. Specifically, congener-specific geometric means from NHANES 2003/2004 in blood were summed to get the total concentration, and congener-specific medians from opportunistic sampling in breast milk were summed to get the total concentration. Predictions were reasonably close to measurements for five of eight congeners. While these predictions encouragingly match observations for the majority of congeners, uncertainties exist in the exercise, starting from development of dose estimates based on limited environmental measurements, to indoor contact rates with house dust, to the PK parameters of absorption and elimination half-life. There is also variability in U.S. body burdens.

13. **The Importance of the Dust Pathway:** The overall weight-of-evidence of this exercise supports the finding that the bulk of U.S. exposures occur in the indoor environment through contact with house dust. The exercise suggests contact with house dust accounts for between 80 and 90% of total exposures, with the remainder due primarily to food ingestion. Circumstantial evidence supporting this hypothesis was the high concentrations found in U.S. house dust, and other researchers have also identified house dust as a key matrix of exposure concern for these compounds.

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