Human Exposure to Decabromodiphenyl Ether, Tetrabromobisphenol A, and Decabromodiphenyl Ethane in Indoor Dust

Catherine PETITO BOYCE, Sonja N. SAX, David G. DODGE, Margaret C. POLLOCK, and Julie E. GOODMAN

1 Gradient Corporation, 600 Stewart Street, Suite 803, Seattle, WA 98101, USA
2 Gradient Corporation, 20 University Road, Cambridge, MA 02138

Abstract

This paper explores efforts that have been made to quantify the intake of selected flame retardants (FRs) from indoor dust, focusing on decabromodiphenyl ether (BDE-209), tetrabromobisphenol A (TBBPA), and decabromodiphenyl ethane (DeBDethane). After reviewing approaches used to evaluate human exposures to FRs via indoor dust and available exposure estimates, we present quantitative estimates of human exposures to indoor dust via incidental ingestion and dermal contact in a residential setting based on a probabilistic exposure assessment framework. Average daily intake estimates for BDE-209 ranged from 2.2 to 451 ng/kg-day for young children and from 0.26 to 54 ng/kg-day for adults. Based on data from a single study each for TBBPA and DeBDethane, the average daily intake for TBBPA was estimated to be 0.26 ng/kg-day in children and 0.03 ng/kg-day in adults, while the corresponding estimates for DeBDethane were 0.99 ng/kg-day for children and 0.11 ng/kg-day in adults. These results support previous evaluations pointing to incidental soil/dust ingestion as the primary contributor to the total indoor dust exposures associated with the combined exposure pathways (constituting 60 to almost 90% of the combined exposures). This paper also reviews important sources of uncertainty in the exposure estimates and additional research needed to develop more rigorous exposure estimates, such as more accurate characterization of exposure parameters for indoor dust exposures, studies to address data gaps for specific FR compounds and exposure settings, and development of more standardized sampling and analysis protocols.

Keywords: PBDE, BDE-209, TBBPA, DeBDethane, flame retardants, exposure assessment, dust

1. Introduction

Flame retardants (FRs) are chemicals that are used in a variety of products to delay or prevent the ignition or spread of a fire in combustible materials. Flame retardant chemicals differ in their chemical nature and in the ways that they act to reduce the potential for fire-related harm and damage [1]. In recent years, numerous studies and reports have examined the presence of FR chemicals in various environmental media (e.g., air and dust) and biological media (including human tissues), the sources of these chemicals, and the likely magnitude and relevance of potential pathways for human exposures (e.g., inhalation or incidental ingestion in food or dust). These studies vary in the sampling methodologies used to measure FRs and in the specific chemicals, environmental media, exposure pathways, and exposure locations included in these analyses. As a result, only preliminary conclusions can be drawn from these studies regarding the exposure sources, pathways, and chemicals that are the primary contributors to human exposure.

In a companion paper, Dodge et al. [2], critically reviewed and analyzed available data regarding the presence of six selected FRs in indoor dust collected using variable sample collection methods (i.e., vacuum, wipe, and tweezer techniques), in different exposure settings [i.e., residences, workplaces (reflecting use of FR-containing products, not manufacturing processes), schools, automobiles, and hotels], and different geographical locations (i.e., North America, Europe, and elsewhere). The selected FRs examined by Dodge et al.
[2] included compounds reflecting a range of FR characteristics, including their current role in FR uses (e.g., compounds representing a high proportion of current FR uses vs. compounds that have more limited, but potentially emerging, use), their chemical composition (e.g., halogenated vs. non-halogenated compounds), and their method of application in products (e.g., bound to vs. applied to product materials).

Based on a detailed literature search, Dodge et al. [2] found that the most extensive information regarding indoor dust concentrations and related exposure evaluations was available for decabromodiphenyl ether (decaBDE) – an FR compound that currently makes up a major portion of the brominated FR market. The commercial decaBDE belongs to the family of polybrominated diphenyl ethers (PBDEs) that have received intense scrutiny because several studies have reported increasing concentrations in the environment and in humans. DecaBDE is composed almost entirely of the PBDE congener decabromodiphenyl ether (BDE-209), and is used primarily as an additive in a range of hard plastics (i.e., TV casings) and secondarily in some upholstery textiles. More limited information was identified regarding tetrabromobisphenol A (TBBPA) – a high production FR mostly used as a reactive intermediate in printed circuit boards – and decabromodiphenyl ethane (DeBDethane) – another brominated FR being produced and marketed as a general-purpose, direct substitute for BDE-209. For three of the six FRs considered by Dodge et al. [2], no indoor dust data or exposure analyses were located in the scientific literature – resorcinol diphenyl phosphate (RDP), bisphenol A diphenyl phosphate (BDP), and 6H-dibenzo(c,e)(1,2)oxaphosphorin, 6-oxide (DOPO). All three of these non-halogenated compounds have been used as alternatives to either decaBDE or TBBPA.

In this paper, we build upon the Dodge et al. [2] analysis to develop estimates of human intake associated with residential exposures to BDE-209, TBBPA, and DeBDethane in indoor dust. First, we review the approaches that have been used to estimate human intake of FRs via indoor dust and present the available intake estimates that have been derived. Then, we present an assessment of human exposures to FRs in indoor dust based on a probabilistic exposure assessment framework. Data compiled by Dodge et al. [2] are applied in this framework to derive intake estimates for BDE-209, TBBPA, and DeBDethane. In contrast to most previous evaluations of potential exposures to FRs via indoor dust, which have been conducted using simplified, deterministic calculations, the use of a probabilistic approach allows for more explicit consideration and examination of the distribution of available dust concentration data and the inherent variability and uncertainty in the exposure parameters used in the analysis. Finally, we review important sources of uncertainty in the exposure estimates and additional research that is needed to support development of more rigorous exposure estimates.

2. Exposure Assessment Literature Review

Dodge et al. [2] conducted an extensive literature search to identify published articles and other reports presenting indoor dust data for selected FRs. This literature search also identified materials discussing approaches for developing quantitative exposure estimates for FRs in indoor dust and closely-related issues, which were the focus of the analysis presented in this paper. Table 1 briefly summarizes the articles identified in the literature search that present approaches for quantifying human intake of FRs.

Almost all of the identified reports that provided exposure estimates for FRs focused on PBDEs, with most of the reports providing specific information regarding BDE-209. One recent article provided exposure estimates for DeBDethane [3], while another report presented quantitative exposure estimates for TBBPA [4]. This review also identified one study which examined exposure issues for phosphororganic FR compounds [5].

Almost all of the reports that provided quantitative exposure estimates – including the report identified for DeBDethane – examined intake associated with incidental ingestion of indoor dust, although one report only included air data and inhalation exposures [6] and several reports evaluating PBDEs did not provide intake estimates specifically for BDE-209 [7, 8]. The report that presented intake estimates for TBBPA assessed exposures via dust ingestion, inhalation, and dietary sources [4]. Of the studies that provided estimates for BDE-209, only a few provided intake estimates for exposure pathways other than soil and dust ingestion. For example, several studies also provided quantitative estimates of intake via inhalation and/or diet [9-11]. Hays and Pyatt [12] evaluated potential exposures to decaBDE in a variety of scenarios, including ingestion of breast milk from mothers with workplace exposures, mouthing of decaBDE-containing products, inhalation of decaBDE particulates released from products, and overall intake (estimated based on measured body burden levels). The Hays and Pyatt study, however, did not develop estimates of decaBDE intake associated specifically with incidental ingestion of soil and dust. Lorber [13] conducted an extensive multipathway assessment of potential exposures to PBDEs (including BDE-209), including consideration of exposures via inhalation; dermal contact with soil and dust; and ingestion of water, soil, dust, and a variety of food types.

In most cases, only highly simplified exposure algorithms were used to estimate FR intake associated with dust exposures. For example, the primary focus of many of the studies was collection of concentration data from
<table>
<thead>
<tr>
<th>Study</th>
<th>PBDEs/ chemicals</th>
<th>Exposure Setting</th>
<th>Exposure Data</th>
<th>Exposed Population</th>
<th>Exposure Routes</th>
<th>Exposure Assessment Type</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdullah et al., 2008</td>
<td>TBBPA and</td>
<td>R, W, A.</td>
<td>Collected medium measurements - ID, 1A, OA; used estimated dietary intakes from another study</td>
<td>Receptors in ~160 homes, offices and public microenvironments (e.g., restaurants and pools), 5 outdoor sampling locations; and 5 cars.</td>
<td>ING - S/D, ING - FD, INH</td>
<td>MOD - INTK (simple algorithms)</td>
<td>UK (West Midlands)</td>
</tr>
<tr>
<td>Harrad et al., 2008b</td>
<td>PBDEs 6-10 congeners, including BDE-209b, Dl(DF)/Hb, and 12 other brominated PBDEs</td>
<td>R, W, A.</td>
<td>Collected medium measurements - ID</td>
<td>Adults and toddlers contacting 30 homes, 18 offices, and 29 cars</td>
<td>ING - S/D</td>
<td>MOD - INTK (simple algorithms)</td>
<td>UK (West Midlands)</td>
</tr>
<tr>
<td>Harrad et al., 2008b</td>
<td>PBDEs 6-10 congeners, including BDE-209b</td>
<td>R</td>
<td>Collected medium measurements - ID</td>
<td>Adults and toddlers contacting ~80 homes</td>
<td>ING - S/D</td>
<td>MOD - INTK (simple algorithms)</td>
<td>NA (TX: Houston; W (NZ))</td>
</tr>
<tr>
<td>Loehr, 2008</td>
<td>PBDEs primary focus on 9 congeners</td>
<td>R, W</td>
<td>Used measured medium data from various individual studies - WRK, GE, ID, 1A, OA, fish, meat, dairy, soy BLD, MLK</td>
<td>General population analyses for adults; children (1-7, 4-11, 12-19 years old)</td>
<td>ING - S/D, WTR, FD (dairy, meat, fish), DERM - S/D, INH</td>
<td>MOD - INTK (simple algorithms)</td>
<td>primarily NA; some references for E, O</td>
</tr>
<tr>
<td>Sjodin et al., 2008</td>
<td>PBDEs 2 congeners, including BDE-209b</td>
<td>R</td>
<td>Collected medium measurements - ID</td>
<td>Adults in 40 residences in 4 countries</td>
<td>ING - S/D</td>
<td>MOD - INTK (simple algorithms)</td>
<td>NA (USA; E (Germany, Great Britain; O (Australia))</td>
</tr>
<tr>
<td>Stapleton et al., 2008</td>
<td>PBDEs C-2 congeners, including BDE-209b</td>
<td>R</td>
<td>Collected medium measurements - OTHER (hand wipes)</td>
<td>52 receptors in Durham, NC</td>
<td>ING - S/D</td>
<td>MOD - INTK (simple algorithms)</td>
<td>NA (USA)</td>
</tr>
<tr>
<td>Allen et al., 2007</td>
<td>PBDEs R.</td>
<td>R</td>
<td>Collected medium measurements (using personal and air sampler) - 1A; used measured FD and S/D data from other studies</td>
<td>20 Boston-area residents</td>
<td>ING - S/D, FD (dairy, meat, fish); INH</td>
<td>MOD - INTK (simple algorithms)</td>
<td>NA (MA)</td>
</tr>
<tr>
<td>Fan et al., 2007</td>
<td>PBDEs 6 congeners</td>
<td>R</td>
<td>Collected medium measurements - ID (from fans and air conditioner filters); used FD and INH intake estimates from other studies</td>
<td>Residents of 31 Singapore homes</td>
<td>ING - S/D, FD (dairy, meat, fish); INH</td>
<td>MOD - INTK (simple algorithms)</td>
<td>primarily O (Singapore); some info for NA, E</td>
</tr>
<tr>
<td>Fischer et al., 2006</td>
<td>PBDEs 6 congeners</td>
<td>R</td>
<td>Collected medium measurements - BLD; used measured S/D and MLK data from other studies</td>
<td>4 members of a CA family</td>
<td>ING - S/D, MLK</td>
<td>MOD - INTK (simple algorithms)</td>
<td>NA (CA)</td>
</tr>
<tr>
<td>Harrad et al., 2006</td>
<td>PBDEs R, W, O</td>
<td>R, W, A.</td>
<td>Collected medium measurements - 1A, soro 1D; used FD intake estimates from other studies</td>
<td>Receptors at 31 homes, 33 offices, 23 cars, and 3 public microenvironments in Birmingham, UK</td>
<td>ING - S/D, INH</td>
<td>MOD - INTK (simple algorithms)</td>
<td>E (UK)</td>
</tr>
<tr>
<td>Reference</td>
<td>Exposure Sources</td>
<td>Time Period</td>
<td>Notes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------------------------</td>
<td>------------------</td>
<td>-------------</td>
<td>----------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hays and Pyatt, 2009&lt;sup&gt;65&lt;/sup&gt;</td>
<td>BDE-209</td>
<td>R</td>
<td>Used data collected and compiled in scientific literature, agency reports, and manufacturer information. General population - analyses focused on children (0-2 years old); also some analyses of adults. ING - S/D, FT, INH, also MLK, OTHER. MOD - INTEK (simple algorithm); MOD-HUBAPK (for general environmental exposures). NA.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wenning et al., 2008&lt;sup&gt;66&lt;/sup&gt;</td>
<td>PBDEs (17 congeners, including BDE-209)</td>
<td>R, W</td>
<td>Collected medium measurements - ID (from vacuum cleaners and air conditioners filters). Receptors at approximately 10 homes and 1 business. ING - S/D. MOD - INTEK (simple algorithm). NA (Northern CA, O (New Zealand)).</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jones, Otacek, et al., 2005&lt;sup&gt;67&lt;/sup&gt;</td>
<td>PBDEs (excluding BDE-209)</td>
<td>R</td>
<td>Used measured and modeled concentrations from various sources - IA, OA, ID, OS, FD (including fish, vegetables, grain, fruit). General population - adults 20+ years old; children 0-0.5, 0.5-4, 4-11, and 12-19 years old; also workers in computer recycling facility. ING - S/D, FT, INH; also MLK. MOD - INTEK (using Canadian multimedia risk assessment model - MUM-FAME). OTHER. NA (Toronto, Canada).</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Singleton, et al., 2005&lt;sup&gt;68&lt;/sup&gt;</td>
<td>PBDEs (22 congeners, including BDE-209)</td>
<td>R</td>
<td>Used measured data from several other studies. Young children (1-3 years old). ING - S/D, FT, INH. MOD - INTEK (simple algorithm). E (including Germany); NA (including US, Canada).</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Luksemburg et al., 2005&lt;sup&gt;69&lt;/sup&gt;</td>
<td>PBDEs (17 congeners, including BDE-209, and more through non homes)</td>
<td>R, W</td>
<td>Collected medium measurements - ID. General population in 17 residences (young children [1-4 years old], adults). ING - S/D, FT, INH; also MLK. MOD - INTEK (simple algorithm). NA (MI, CA, NC, WA). O (New Zealand).</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sharp and Londer, 2004&lt;sup&gt;70&lt;/sup&gt;</td>
<td>PBDEs (13 congeners, including BDE-209)</td>
<td>R</td>
<td>Collected medium measurements - ID. General population in 17 residences (young children [1-4 years old]). ING - S/D, FT, INH; also MLK. MOD - INTEK (simple algorithm). NA (10 locations throughout the US).</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wilford et al., 2004&lt;sup&gt;71&lt;/sup&gt;</td>
<td>PBDEs (10 congeners detected, not including BDE-209)</td>
<td>R, O</td>
<td>Collected medium measurements - IA, OA. General population - adults in 74 residences. INH. MOD - INTEK (simple algorithm). NA (Ontario, Canada).</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes:
- **Time Period**
  - BLD = blood
  - IA = indoor air
  - ID = indoor dust
  - MLK = human milk
  - OA = outdoor air
  - OS = outdoor soil
  - PTW = water
  - OTHER

- **Exposure Dose**
  - INC - incidental ingestion of soil and/or food
  - IDG - ingestion of water
  - IIG - ingestion of food
  - BBD = body burden
  - BBD - body burden
  - INK - intake
  - MOD - modeled (estimated) exposure
  - MBM - measured exposure
  - SMTG - measured exposure
  - PFF - pharmacokinetic model

- **Exposure Assessment Type**
  - PBDE - polybrominated diphenyl ethers
  - PBDE - polybrominated diphenyl ethers
  - PBDE - polybrominated diphenyl ethers
  - PBDE - polybrominated diphenyl ethers

- **Location**
  - NA = North America
  - E = Europe
  - O = Other
  - Source Ottone et al., 2003 includes modeling of emissions and fate.
environmental media (e.g., indoor dust), and the exposure assessment component of the study consisted of multiplying the measured concentrations by an intake rate (e.g., an incidental soil/dust ingestion rate) to derive a rudimentary intake estimate. In most of these cases, other factors that would be required to obtain more refined or accurate exposure estimates (e.g., factors influencing absorption or uptake, or activity patterns influencing the nature or magnitude of exposure) were not considered or addressed. In contrast, Lorber [13] addressed the necessary input parameters for a wide range of intake pathways, while Jones-Otazo et al. [7] applied a Canadian multimedia risk assessment model that uses physical and chemical properties to predict the environmental fate of chemicals and then estimates exposures via a number of pathways. Hays and Pyatt [12] also considered a variety of exposure assumptions (many for non-standard exposure scenarios) and incorporated a simplified body burden/pharmacokinetic model to estimate overall intakes based on biological monitoring data. Stapleton et al. [14] estimated dust intake based on assumptions regarding dust intake occurring during hand-to-mouth contacts.

Another approach for examining potential exposures to chemicals is to measure or estimate concentrations of the chemicals of interest in biological media, such as blood or human milk. Two of the identified studies used this approach to explore FR exposures [12, 13]. In the Lorber study [13], measured concentrations in a variety of exposure media were used to estimate potential exposures via a number of exposure pathways, including soil and dust, air, water, and a number of dietary sources. A simple pharmacokinetic model was then used to estimate the body burdens in blood and human milk that would be associated with the intake estimates. The estimated body burdens were compared with measured body burden concentrations to assess the validity of the exposure estimates. Lorber [13] concluded that the measured and estimated body burden concentrations were in relatively good agreement for many of the studied PBDE congeners, "suggesting general validity of the approach."

Similarly, Hays and Pyatt [12] estimated the overall intake of decaPBDE using a simple pharmacokinetic model and measured concentrations of decaPBDE in human serum. This approach was selected to avoid the uncertainties inherent in attempting to identify all possible exposure pathways, to locate suitable concentration data for the potential exposure media, and to estimate total intakes via the sum of the identified pathways. This study did not include any analyses to compare intakes estimated based on serum data with intakes estimated using alternative methods.

Table 2 summarizes the soil/dust intake estimates for BDE-209 that were identified in the literature. Underlying key assumptions (i.e., incidental soil/dust ingestion rates and bioavailability from soil) are also summarized in this table. The estimated incidental soil/dust ingestion rates are generally drawn from a similar set of available interpretations of the scientific literature. As a result, the assumed values are similar in many of the studies. For children, estimated soil/dust intake rates range from 3 mg/day (a low-end value assumed for older children) [15] to 400 mg/day (a high-end value assumed for young children) [16], with most estimates in the 50 to 200 mg/day range. Estimated soil/dust intake rates for adults were lower, ranging from 0.56 mg/day to 110 mg/day, with most estimates in the 20 to 100 mg/day range.

Applying mid-range ingestion estimates and BDE-209 concentrations, the estimated BDE-209 intakes via dust ingestion ranged from approximately 33 to 14,000 ng/day for young children (with most estimates in the 5 to 250 ng/day range), and from approximately 1 to 4,300 ng/day in adults (with most estimates in the 1 to 100 ng/day range). Overall, intake estimates for young children ranged from a low of < 1 ng/day (when minimum detected concentrations were used) to a high of 54,000 ng/day (when high-end soil concentrations or high-end soil ingestion rates were used). All of the available exposure estimates used soil/dust ingestion values corresponding with chronic incidental ingestion of soil and dust in a residential setting.

For TBBPA and DeBDethane, only one study each was identified that presented estimates of FR intake via dust ingestion. Abdallah et al. [4] estimated TBBPA intake via inhalation, dust ingestion, and dietary sources for toddlers (between the ages of 6 and 24 months old) and adults in the United Kingdom (UK). TBBPA in ingested dust was assumed to be completely absorbed. Assuming a mean toddler body weight of 10 kg, the average TBBPA intake from dust estimated for toddlers in this study was 0.44 ng/day (using a mean dust intake of 50 mg/day) with a 95th percentile estimate of mean intake of 0.85 ng/kg-day. Using a higher estimated dust intake rate for toddlers of 200 mg/day, the average TBBPA intake from dust was estimated as 1.8 ng/kg-day and the 95th percentile estimate of the high end intake was estimated as 3.5 ng/kg-day. Assuming a mean adult body weight of 70 kg, the average TBBPA intake from dust estimated for adults in this study was 0.023 ng/kg-day (using a mean dust intake of 20 mg/day) with a 95th percentile estimate of mean intake of 0.046 ng/kg-day. Using a higher estimated dust intake rate of 50 mg/day for adults, the average TBBPA intake from dust was estimated as 0.057 ng/kg-day and the 95th percentile estimate of the high end intake was estimated as 0.12 ng/kg-day.

When comparing exposures for all three pathways (air, dust, and diet), Abdallah et al. [4] reported that dust ingestion comprised approximately 90 to 99% of the total TBBPA intake for toddlers, but represented a smaller portion of total TBBPA intake in adults (ranging from 11.8 to 71.7% of total intake depending on the summary statistic and dust intake rate examined). Thus, in some instances, dietary sources were estimated to be a greater
contributor than dust to total TBBPA exposures for adults. For both toddlers and adults, air was a minor contributor to total intake, comprising < 0.1 to 2.6% of total intake for toddlers and 3.5 to 6.8% of total intake for adults.

In the one study providing intake estimates for DeBDethane, Harrad et al. [3] estimated incidental intake of DeBDethane based on indoor dust data collected in several exposure settings in the UK. Only exposures associated with incidental dust ingestion were quantified in this study, and the total soil/dust ingestion rate was apportioned among several exposure settings as follows: 72% home, 23.8% office, and 4.2% car for adults; and 95.8% home and 4.2% car for 6- to 24-month-old toddlers. Based on these assumptions (and assuming an average body weight of 71 kg for adults and 10 kg for toddlers), the intake of DeBDethane from dust was estimated to be 0.070 ng/kg-day (average) and 0.24 ng/kg-day (95th percentile) for adults using a mean dust ingestion rate of 10 mg/day, and 0.17 ng/kg-day (average) and 0.62 ng/kg-day (95th percentile) using a high dust ingestion rate of 50 mg/day. For toddlers, the intake of DeBDethane from dust was estimated to be 1.4 ng/kg-day (average) and 4.8 ng/kg-day (95th percentile) using a mean dust ingestion rate of 50 mg/day and 5.4 ng/kg-day (average) and 19 ng/kg-day (95th percentile) using a high dust ingestion rate of 200 mg/day.

### 3. Exposure Assessment Approach

A number of investigators have suggested that the indoor environment is the primary source of exposures to FRs as a result of incidental ingestion of and dermal contact with FRs present in house dust following release from commercial products (e.g., televisions, mattresses) [13]. Yet the characteristics and magnitude of FR releases from indoor sources are not well understood [17, 18]. The potential significant contribution of indoor sources to overall FR exposures has been of particular interest.
because people spend most of their time indoors [10, 19] and because house dust can be an important exposure pathway for young children [20]. Thus, a probabilistic model was developed to quantitatively estimate exposures to FRs via contacts with indoor dust. The approach presented in this paper incorporates and discusses a number of important exposure factors that were omitted from the simplified, deterministic exposure calculations that underlie many of the exposure estimates previously presented in the literature. In addition, in contrast with deterministic approaches, which apply only a single value for each input parameter, the probabilistic framework presented here also incorporates and allows for more explicit evaluation of the distribution of available dust concentration data and the inherent variability and uncertainty in the exposure parameters.

Two potential exposure pathways were included in these analyses: incidental ingestion of indoor dust and dermal contact with indoor dust. Incidental ingestion was included because it clearly represents a primary pathway whereby dust exposures could occur, and is a pathway that has been quantified (at least in a screening level way) in a number of studies. Although many FR chemicals have high molecular weights and other physicochemical properties that are unfavorable to dermal absorption [21-23], the dermal contact pathway was included in these evaluations because it may contribute a non-negligible amount to overall FR exposures. For example, the Lorber [13] analysis quantified adult exposures to nine PBDE congeners via inhalation, dermal contact with soil/dust, and ingestion of water, soil/dust, and nine dietary sources. For BDE-209, soil/dust ingestion accounted for 71% of the total estimated exposure and soil/dust dermal contact accounted for 16% of the total exposure. In contrast, inhalation exposures accounted for approximately 1% of total exposures. Similar results were observed for other congeners and for the total PBDE exposure estimates. Thus, the current analysis was designed to allow exploration of the relative contributions of ingestion and dermal exposure pathways and the associated uncertainties in conducting such analyses.

The exposure calculations are structured to reflect exposures to adults and young children within a residential setting. The residential exposure scenario was selected for several reasons. Most importantly, standard assumptions for this scenario typically represent the most regular, extensive exposure conditions, i.e., individuals in settings such as schools or workplaces are typically assumed to have exposures in such settings less frequently or for shorter durations. Therefore, calculations based on standard residential assumptions commonly yield high-end estimates of potential exposures, and exposures associated with other scenarios can be assumed to be less, e.g., a school exposure scenario might assume exposure for only six hours/day and five days/week, and thus would yield lower exposure estimates than a residential scenario that assumes daily exposures for 24 hours/day. Moreover, for the specific FR chemicals included in this analysis, available data were most frequently collected in residential settings and the available limited data showed no statistically significant differences between residential concentrations and those measured in most other settings [24]. In the only exception, BDE-209 concentrations measured in vehicles were found to be significantly greater than those found in the other studied settings. Thus, exposure estimates based on assumptions for a residential setting were derived in this analysis; however, the implications of these results for assessing potential exposures in other settings (e.g., workplaces using FR-containing products, schools, vehicles, or other settings) are described in the Discussion section.

As discussed above, the literature search conducted by Dodge et al. [2] yielded studies of dust concentration data for three of the FRs of interest: decaBDE (composed almost entirely of BDE-209), TBBPA, and DeBDethane. Thus, these three compounds are included in the quantitative calculations presented below. Dust data collected for these compounds in various locations in North America, the UK, and other European countries were used to estimate average daily intakes for adults and young children residing in these areas.

Indoor exposures to FRs can vary among members of United States (US) and European populations owing to differences in sources of these compounds in indoor environments, laws and regulations governing the use of these FRs, building ventilation, and the habits of residence occupants (e.g., frequency of cleaning). Measured levels of FRs in dust may also vary as a result of differences in the way samples are collected, processed, and analyzed [24]. Therefore, a probabilistic Monte Carlo approach was used to estimate a range of potential exposure levels in US and European populations. A Monte Carlo analysis accommodates a distribution of values for a given input parameter (e.g., a range of FR concentrations in dust) and allows different combinations of input parameter values to be modeled based on the specified distributions. In this case, the Monte Carlo analysis was conducted using Crystal Ball® software and incorporated 10,000 iterations of the exposure estimate calculations.

To estimate exposures of children and adults via incidental ingestion of and dermal contact with BDE-209, TBBPA, and DeBDethane in indoor dust, the following basic exposure algorithm was developed.

\[
\text{ADD} = \left( C \times \left( \sum IR \times FS \right) + \left( SA \times DA \times DAF \times EF \right) \times CF \right) \times \frac{1}{BW}
\]

where:
ADD: Average daily dose of FR for a child or an adult (ng/kg-day)
C: Concentration of FR in dust (ng/g)
IR: Incidental soil/dust ingestion rate for a child or an adult (mg/day)
FS: Fraction of soil/dust ingestion that consists of dust (unitless)
SA: Skin surface area exposed for a child or an adult (cm²)
DA: Dermal absorption fraction (unitless)
DAF: Soil/dust adherence factor for a child or an adult (mg/cm²-event)
EF: Event frequency (number of events per day)
CF: Conversion factor (1 g/1,000 mg)
BW: Body weight of a child or an adult (kg)

This algorithm reflects standard components and approaches for estimating exposures as presented in risk assessment guidance [e.g., United States Environmental Protection Agency (US EPA)] [25]. Values for each of the input parameters that best reflect currently available scientific data were selected based on a review of information available in the scientific literature and other sources. The input assumptions for FR intake via incidental ingestion of dust and dermal contact with dust are summarized in Table 3 and are described below. The selected distribution types presented in this table reflect the availability of data for each parameter (e.g., point estimates were used for certain parameters for which only limited data were available). Where more extensive data were available, the selected distributions represent the distribution type determined to best fit the available data by researchers, as well as agencies and other entities, who have applied those data in exposure analyses. For the FR concentrations in indoor dust, the raw data were available for each of the FRs and regions included in the analysis; as a result, the raw data were used as inputs into the Monte Carlo analyses. The distributions selected for each FR data set reflect the best fit of the raw data.

### 3.1 Concentrations of BDE-209, TBBPA, and DeBDethane in indoor dust (C)

The concentrations of BDE-209, TBBPA, and DeBDethane used in these analyses are based on the dust data compiled by Dodge et al. [2]. Dodge et al. [2] obtained data from studies in North America, Europe, and other parts of the world. The dust data were found to differ by region; therefore, separate calculations were conducted for North America, the UK, and for other parts of Europe, reflecting measured concentrations of these FRs in these regions. Dodge et al. [2] also identified limited data for several other countries. These data were not included in the Monte Carlo analyses, but are described in the Discussion section.

Table 3 presents the summary statistics used in the Monte Carlo analyses for measured concentrations of BDE-209, TBBPA, and DeBDethane in residential indoor dust for the three regions included in these analyses. Note that the data summaries presented in Dodge et al. [2] reflect indoor dust concentrations measured in a variety of different exposure settings (including residences, workplaces, a school, a hotel, and vehicles) and areas of the world based on raw data from the original studies. Only those data collected from residential exposure settings in North America, the UK, or the non-UK countries in Europe were included in the exposure estimate calculations. As a result, the summary statistics reflected in these exposure analyses differ slightly from those presented in Dodge et al. [2]. It should also be noted that several studies identified by Dodge et al. [2] for which raw data were not available or which had a low detection frequency were excluded from the statistical analyses presented in Dodge et al. [2] and the summary statistics applied in the exposure calculations.

### 3.2 Assumptions for incidental dust ingestion

Standard calculations addressing exposures via incidental ingestion of dust require assumptions regarding the incidental dust ingestion rate (in both young children and adults when evaluating a residential scenario), as well as the relative bioavailability of the chemical of interest when ingested in a dust matrix. The current analysis focuses on issues critical to development of exposure estimates and does not include an evaluation of available toxicity information to determine toxicity values for deriving risk estimates. Because determination of "relative" bioavailability is dependent on the choice of a specific toxicity factor (and consideration of the underlying toxicity study – or studies – used to derive that toxicity factor), a relative bioavailability adjustment factor was not included in the Monte Carlo exposure calculations. Available information regarding bioavailability of the chemicals of interest, and the implications of this information for exposure and risk estimates for these chemicals, are described in the Discussion section.

#### 3.2.1 Incidental soil/dust ingestion rate (IR)

A number of studies have been undertaken to estimate children's incidental ingestion of soil and dust [26, 27-29, 30-32]. These studies have estimated incidental ingestion rates (in units of mg of soil/dust intake per day) using a mass balance approach comparing concentrations of various trace elements in residential soil and dust with concentrations of the same trace elements in food and fecal samples for the studied children. A range of potential values for this parameter exists because of inter-individual variability among different children and intra-individual variability in ingestion rates on different
### Table 3
Monte Carlo Parameter Assumptions and Inputs

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Distribution Type</th>
<th>Assumptions</th>
<th>Values</th>
<th>Source</th>
<th>Basis for Selection</th>
</tr>
</thead>
</table>
| Flame retardant concentrations BDE-209         | Lognormal         | NA: GM = 1,325 GSD = 3.1
| concentrations in dust (ng/g)                 |                   | 95th percentile = 8,495                          |                             | Dodge et al. (2009)                         | Distribution reflects compilation of concentrations measured in residential settings based on a comprehensive literature evaluation. The statistics were generated from the combined raw data of 6 US studies (78 samples; Costner et al., 2005; Harrad et al., 2008b; Seelhorst et al., 2005; Sharp and Lunder, 2004; Stapleton et al., 2004; Wu et al., 2007b), 3 European studies (40 samples; Karlsson et al., 2007; Knoth et al., 2002; Santillo et al., 2009), and 3 UK studies (43 samples; Harrad et al., 2008b; Pless-Mulloli et al., 2009) |
| TBBPA concentrations in dust (ng/g)            | Lognormal         | UK: 95th percentile = 12
|                                            |                   | GM = 56 GSD = 4.5                                 |                             |                                            | Distribution reflects compilation of concentrations measured in residential settings based on a comprehensive literature evaluation. The statistics were generated from the raw data of 1 UK study (35 samples; Abdallah et al., 2008). |
| DeBDEbromane concentrations in dust (ng/g)     | Lognormal         | UK: 95th percentile = 9.1
|                                            |                   | GM = 56 GSD = 4.5                                 |                             |                                            | Distribution reflects compilation of concentrations measured in residential settings based on a comprehensive literature evaluation. The statistics were generated from the raw data of 1 UK study (35 samples; Abdallah et al., 2008). |
| Dust Ingestion                                 |                   |                                                  |                             |                                            |                                                                                                                                                    |
| Soil/dust intake (mg/day) – child              | Lognormal         | GM = 45                                          |                             | Stanek and Calabrese, 1995a,b, 2006          | a) Based on well-conducted mass balance study (Amherst, MA);
b) Appears to be conservative based on values observed in other analyses, e.g., Anacostia, MT study (Stanek and Calabrese, 1999) and Washington study (Davis and Mirick, 2000); |
|                                            |                   | GSD = 1.85                                        |                             |                                            |                                                                                                                                                    |
|                                              |                   | 95th percentile = 124                             |                             |                                            |                                                                                                                                                    |
| Soil/dust intake (mg/day) – adult              | Lognormal         | One-half of that for children                     |                             | Calabrese et al., 1996; Davis and Mirick, 2006; Stanek et al., 1997 US EPA, 1996 | Consistent with US EPA standard deterministic RMG recommendations for soil/dust ingestion rates and available study data |
| Fraction of soil/dust ingestion that consists of dust (unless) | Point estimate | 0.55                                             |                             | US EPA, 1996                              | Consistent with US EPA standard guidance regarding proportion of intake derived from soil vs. dust |
| Dermal Contact                                |                   |                                                  |                             |                                            |                                                                                                                                                    |
| Skin surface area (cm²) – child                | Point estimate    | 4.849                                            |                             | US EPA, 2006                              | Average skin surface area for a child < 1 to < 6, for forearms, hands, lower legs, and feet; included body parts are consistent with those assumed in deriving area-weighted soil/dust adherence factor presented below |
|                                              |                   |                                                  |                             |                                            |                                                                                                                                                    |
| Skin surface area (cm²) – adult                | Point estimate    | 1.852                                            |                             | US EPA, 2006                              | Average surface area for an adult, for face, forearms, hands, and lower legs; included body parts are consistent with those assumed in deriving area-weighted soil/dust adherence factor presented below |
| Soil adherence factor (mg/cm²-event) – child   | Lognormal         | GM = 0.04                                        |                             | US EPA, 2006                              | Represents a conservative estimate of likely indoor dust adherence:
a) Based on weighted average for daytime child scenario (including time spent playing outdoors)
b) Weighting based on contact with forearms, hands, lower legs, and feet |
analyses of the available study data have examined the relative reliability of the trace elements used in the studies, the inter- and intraindividual variability in daily soil ingestion rates observed in the studied children, and the implications of using data from short-term studies to estimate long-term patterns of soil/dust ingestion.

A distribution based on one of the most comprehensive and detailed studies of children’s incidental soil ingestion was selected for use in these calculations and is shown in Table 3 [27, 29]. This distribution is based on a study of 64 children between the ages of one and four years old residing in the town of Amherst, Massachusetts. This distribution was selected because of the high quality of the underlying study and because it incorporates an effort to predict long-term average intake rates based on the short-term study data. In addition, this distribution appears to be conservative (i.e., health-protective) based on values observed in other studies [26, 29], i.e., the results from this study generally yield higher soil/dust ingestion rates than indicated by these other recent studies. Moreover, because children between the ages of one and four years old tend to have higher incidental soil/dust ingestion rates than other age groups [33-35], use of this distribution to estimate soil ingestion rates for the broader age range examined in this analysis (i.e., under one- to six-year-old children) will tend to overestimate likely actual soil/dust ingestion rates.

Data regarding incidental soil ingestion rates in adults are far more limited [26, 36, 37]. In the absence of such information, the incidental soil ingestion rate for adults was assumed to be one-half of that for children. This assumption is consistent with US EPA standard risk assessment guidance, which recommends a mean soil ingestion rate for adults that is one-half of the recommended value for children under six years old [34]. This approach is also consistent with approaches used in other analyses [11, 13, 38, 39] and with other reviews of available data that have generally estimated incidental soil ingestion rates for adults as one-half or less of rates estimated for young children [40].

### 3.2.2 Fraction of incidental soil/dust ingestion that consists of dust (FS)

The incidental soil/dust ingestion rates described above include ingestion of both soil and dust, but the Monte Carlo exposure model is intended to estimate exposures associated with incidental ingestion of FRs in indoor dust only. Thus, the soil/dust ingestion rates must be adjusted to reflect the component of total soil/dust ingestion that consists of dust. Although some exposure to FRs may occur via contacts with outdoor soil, available studies have suggested that indoor exposures predominate and that outdoor soil concentrations may be substantially less than indoor dust concentrations; therefore, separate consideration of outdoor dust exposures is reasonable. For example, Lorber [13] summarizes exposure media concentrations measured in a variety of studies,
including studies of indoor dust and outdoor soil. These data illustrate the lower concentrations reported in outdoor soil samples vs. indoor dust samples (e.g., the average concentration of BDE-209 in outdoor soil observed in the studies reviewed by Lorber was 15.3 ng/g while the average measured in indoor dust was 2,394 ng/g).

Some analyses of FR exposures have assumed that the amount of dust ingestion is directly proportional to the amount of time spent indoors or in specific environments [7-9, 12, 13]. Because most individuals spend a substantial proportion of their time indoors, these analyses have assumed that almost all soil/dust ingestion is derived from indoor dust, i.e., they have assumed that the fraction of soil/dust intake that consists of dust is approximately 0.8 to 0.9. As recognized by US EPA [33], however, such an assumption is unlikely to be valid. Instead, the proportion of total soil/dust intake that is derived from contacts with soil is likely to be much greater than the proportion suggested based on the percentage of waking hours that are spent outdoors, especially for children. Factors identified by the US EPA that enhance intake of soil include the potential for outdoor activities to include contacts with bare soil areas and areas with "large amounts of accessible loose particles," as well as the lower likelihood of hand washing while outdoors. Based on these considerations, US EPA determined that more appropriate assumptions for apportioning soil/dust ingestion are that the fraction of soil/dust intake derived from soil is 0.45, and that derived from dust is 0.55 [33]. This assumption was applied in the US EPA Integrated Exposure Uptake Biokinetic (IEUBK) model for estimating children's exposure to lead, a model that has been at least partially validated using environmental and biomonitoring measurements from certain communities. The assumption of 0.55 of soil/dust ingestion being derived from dust has also been used in some FR analyses [41] and was applied in the Monte Carlo analyses presented here.

3.3 Assumptions for dermal contact with dust
Exposure assessment calculations addressing exposures via dermal contact with dust require pathway-specific assumptions regarding the exposed skin surface area, adherence of soil/dust to the skin, the dermal absorption fraction for the chemical of interest when present in dust, and the exposure frequency for dermal contact with dust.

3.3.1 Exposed skin surface area (SA)
This exposure parameter reflects the exposed skin surface area that is assumed to come into contact with dust. As will be discussed in more detail below, the default assumptions for this parameter typically reflect the skin surface area that is exposed, and could thus potentially come into contact with dust. The amount of the exposed skin surface area that actually contacts dust during a dust contact event is likely to vary widely from event to event due to differences in such factors as clothing, activity types, and intensity of dust contacts.

The skin surface area assumption applied in this analysis was selected to be consistent with the area-weighted soil/dust adherence factor (DAF) described below (that reflects differences in adherence associated with different body parts). Such consistency is necessary to ensure that the combined parameters used to estimate dermal exposures accurately reflect the underlying data and assumptions used to derive the input parameters. For young children, the area-weighted DAF used in this calculation considers exposures to the forearms, hands, lower legs and feet, while that used in the calculations for adults includes face, forearms, hands, and lower legs. Therefore, the total exposed body surface areas for young children and adults that were applied in these calculations were based on average surface areas of the specific body parts included in the DAF weightings (as presented in US EPA [42], Exhibit C-1).

For children, the surface area values for individuals between the ages of < 1 and < 6 years old were used, resulting in an area-weighted value of 1,852 cm². For adults, the average values for males and females combined were used, resulting in an area-weighted value of 4,849 cm².

It should be noted that these estimates do not account for variability in exposed skin surface areas that would result from different meteorological conditions in different seasons (i.e., exposed skin surface areas are likely to be greater in summer than in winter), but the values applied in this analysis appear to be conservative when compared with other typical US EPA default values. For example, US EPA uses two clothing scenarios to derive recommended default skin surface area assumptions for adults when estimating dermal exposure to soil. In one scenario, an adult is assumed to be wearing a long-sleeved shirt, pants, and shoes – limiting exposure to the head and hands (resulting in a central tendency estimate of exposed skin surface area of 2,000 cm²). In the second scenario, an adult is assumed to be wearing a short-sleeved shirt, shorts, and shoes – and the exposed skin surface area is assumed to include the head, hands, forearms, and lower legs (resulting in an upper percentile estimate of exposed skin surface area of 5,300 cm²) [34]. The adult skin surface area estimate used in these calculations (4,849 cm²) is closer to US EPA's upper percentile estimate. The skin surface area estimate used for young children in these calculations represents approximately 31% of the estimated total skin surface area for a child between the ages of two and three years old (i.e., approximately 6,000 cm²) [35].
3.3.2 Soil/dust adherence factor (DAF)

The soil/dust adherence factor estimates the amount of soil or dust that adheres to the surface of the skin. The entire mass of chemical present in this soil/dust is then assumed to be available for absorption, to the degree determined by the dermal absorption fraction (discussed below). Currently available information regarding this parameter is derived primarily based on data from studies of activities likely to represent frequent and substantial contact with soil, especially for adults (e.g., for gardeners, landscape workers, or groundskeepers).

The degree to which indoor dust will adhere to skin during typical indoor activities will likely be very different from (and less than) the degree of soil adherence observed in most of the soil exposure scenarios that have been studied. Within the studies and in the scenarios evaluated in these analyses, soil/dust adherence will also differ depending on the exposed body parts where skin contact occurs, owing to differences in such factors as clothing, activity types, and intensity of dust contacts. For example, some areas (such as hands or knees) are likely to have a greater degree of soil/dust contact and adherence than others (such as upper arms). While the approach used to develop the parameter assumptions discussed below partially addresses these issues, it cannot completely account for the variability in exposures that will exist under real-world exposure conditions. Lacking any data specific to dust exposures only, the available data were used to estimate potential dust adherence to skin. These data included results from activity studies of soil contacts only, or studies that may have included dust exposures in addition to soil exposures. As a result, the adherence values used in these calculations are likely to be conservative, i.e., to overestimate dust adherence to skin. Using these parameters within the context of a distribution incorporates some of the variability that may be associated with dust exposures.

Based on a series of studies examining soil adherence to skin following a variety of soil contact activities, US EPA has developed recommended soil adherence factors. For children, adherence data are available from studies of children indoors, at daycare centers, and playing in dry or wet soil. For adults, the studied activities include those undertaken by groundskeepers, landscapers, and gardeners. To the extent allowed by the available data, US EPA has developed adherence factors that are weighted to reflect the skin surface areas and measured adherence values associated with various body parts that contacted soil during the studied activities [42].

For children, the US EPA-recommended default adherence factor is 0.2 mg/cm², based on the 95th percentile weighted adherence factor for young children (between the ages of one and six and one-half years old) playing at a daycare center (i.e., a high-end estimate of soil adherence from a central tendency soil contact activity) and the 50th percentile adherence factor for children (between the ages of eight and 12 years old) playing in wet soil (i.e., a central tendency estimate of soil adherence from a high-end soil contact activity). Because adherence of indoor dust to skin during typical indoor activities is likely to be less than soil adherence in any of the studied activities, an adherence factor distribution based on a daycare scenario was selected as a conservative distribution for use in calculating dermal exposures for young children in this analysis. The underlying data used to generate the selected value reflect children's soil/dust exposures during both indoor and outdoor activities. Although the mean value of the selected distribution is less than the US EPA-recommended default point estimate, the selected distribution is likely to better represent indoor exposures than a distribution that includes a greater amount of soil contact or contact with wet soil (see Table 3).

The US EPA default adherence factor value for an adult resident is 0.07 mg/cm². This value is based on the 50th percentile value observed for individuals participating in gardening, and is considered representative of high-end soil intensive activities. In contrast, 50th percentile adherence factors for adult residents for other activities are less (e.g., 0.04 mg/cm² for landscapers, and 0.01 mg/cm² for groundskeepers). Again, because adherence of indoor dust to skin during typical indoor activities is likely to be less than soil adherence in any of the studied activities, an adherence factor distribution based on a scenario for groundskeeper contact with soil was selected as a conservative distribution for use in calculating dermal exposures for adults in this analysis (see Table 3).

3.3.3 Dermal absorption fraction (DA)

The dermal absorption fraction reflects the fraction of the chemical present in soil that is assumed to penetrate the skin following soil adherence to the skin. Default generic assumptions for dermal absorption efficiency reflect data indicating that volatile organic chemicals are absorbed through the skin to a greater extent than are non-volatile organic chemicals. Within standard risk assessment frameworks, default absorption efficiencies are typically recommended when chemical-specific data are not available.

There are limited data regarding the dermal absorption of FRs – however, as noted above, several FR chemicals have high molecular weights (i.e., > 500 g/mole) and other physicochemical properties (e.g., low water solubility and high octanol/water partition coefficients) that are unfavorable to dermal absorption [21-23]. For example, as molecular weight increases, the ability of a chemical to diffuse through the skin will be reduced [43]. Similarly, several studies have observed...
decreasing dermal absorption as a chemical's lipophilicity and octanol/water partition coefficient increase [43]. One in vitro study identified in the scientific literature tested the dermal absorption of two FRs, including decaBDE [43]. In this study, skin from hairless mice was used in a flow-through diffusion cell system and three different concentrations of decaBDE were applied to the skin in a tetrahydrofuran solvent vehicle. The amount of decaBDE was then measured in receptor fluid that was pumped below the skin and collected over a 24-hour period following application. In addition, the skin was washed and concentrations of the compound were measured in the skin and in the wash fluid.

This study found that the 24-hour cumulative percentage of decaBDE in the receptor fluid was very low for all dose levels, and ranged from 0.07 to 0.34% (or a fraction of 0.0007 to 0.0034). This finding indicates that very little of this compound was absorbed through the skin. Depending on the applied dose level, 1.8, 3.2, and 20.2% of the dose was found in the skin for the high, middle, and lowest dose, respectively. Most of the remainder of the applied compound was recovered in the skin wash. In contrast, 39 to 57% of an FR chemical with a lower molecular weight and octanol/water partition coefficient [(1,3-dichloro-2-propyl)phosphate, or TDCP] was found in the receptor fluid during the 24-hour test period, and another 30 to 35% of the dose was found in the skin. The authors of this study noted that mouse skin has been observed to be more permeable than skin from other species, including humans. Thus, results based on use of mouse skin in this test system may overestimate chemical absorption through human skin. Moreover, absorption of decaBDE from dust adhering to skin is likely to be less than that observed following dermal administration of decaBDE in a solvent vehicle.

Other exposure assessments for FRs – including decaBDE – have also concluded that dermal absorption of these compounds is likely to be very low. For example, based on consideration of physical and chemical properties and analogy with data for PCB compounds, a maximal value of 1% dermal absorption (or a DA of 0.01) was recommended in the most recent European Union (EU) risk assessment for decaBDE [21, 44]. In its risk assessment for TBBPA, the EU assumed a dermal absorption fraction of 0.1 (10%), based on physical/chemical property data and the absence of any chemical-specific studies [22]. Similarly, based on the physical and chemical properties of DeBDethane, a worst-case dermal absorption fraction of 0.1% has been recommended for this compound [45]. Lorber [13] used a dermal absorption fraction of 0.03 (3%) for PBDEs in his exposure analysis. This value is the same as that recommended by US EPA for use in analyses of TCDD and other dioxins, and is in a similar range to the US EPA-recommended values for other poorly absorbed compounds [42]. Based on these findings, a DA value of 0.03 was selected as a reasonably conservative value for BDE-209, and a DA value of 0.10 was used as a conservative estimate for TBBPA and DeBDethane in this assessment. The implications of alternative values for this parameter are also described in the Discussion section.

3.3.4 Event frequency (EF)

The event frequency describes the number of events resulting in dermal soil/dust contact that are assumed to occur each day. For the analysis presented here, the event frequency was assumed to be 1 event/day. When combined with the other assumptions used in calculating exposures via dermal contact, this value reflects the assumption that, during each day, a receptor contacts soil over the entire assumed exposed skin surface area, soil adherence is equal to the value assumed for that parameter, and that the soil remains on the skin surface long enough for the mass of chemical present in the adhered soil to be absorbed in accordance with the assumed dermal absorption fraction. This value is consistent with the default value assumed by US EPA [42].

3.4 Other input parameters

3.4.1 Body weight (BW)

Toxicity values are typically calculated based on the amount of substance intake per unit of body weight. Thus, assumptions regarding body weight are needed to calculate exposure estimates in equivalent units. For these calculations reflecting quantitative differences in exposures for young children and adults, assumptions regarding typical body weights for these two groups were applied.

The distributions of body weights used in the Monte Carlo analysis were derived based on the second National Health and Nutrition Examination Survey (NHANES II), conducted by the US Public Health Services. In this survey conducted between 1976 and 1980, information was collected regarding the height and weight of several thousand men and women residing in the US. The data are statistically weighted to represent the entire US population based on age, sex, and race. The normal distributions applied in this Monte Carlo analysis (shown in Table 3) reflect the Brainard and Burmaster [46] re-analyses of these data, which are still the most reliable and comprehensive data currently available for estimating body weight distribution, as summarized by Finley et al. [47]. For the child age range of zero to six years old examined in the Monte Carlo analysis, a weight distribution for three-year-old children (the midpoint of the range) was used. The average body weights from these distributions are consistent with the
standard US EPA-recommended deterministic estimates for adults and young children [34].

3.4.2 Averaging time
Because the focus of this assessment is on estimating exposures to FRs, and not on developing risk estimates, no assumption regarding averaging time was included in these calculations. Instead, the derived exposure estimates reflect average daily intake levels for young children and adults in the US and Europe under the assumed exposure conditions. To derive exposure estimates appropriate for combining with standard toxicity factors for carcinogenic or non-cancer health effects, the average daily intake levels would need to be adjusted to derive time-weighted estimates reflecting the appropriate averaging time. Specifically, to derive standard estimates of cancer risk, lifetime-average daily exposure estimates would be required, while for estimating non-cancer health effects, exposure estimates averaged over an appropriate exposure duration would be required.

4. Results of Exposure Assessment Calculations
Using the approaches and assumptions described above, a Monte Carlo model was run to estimate average daily intakes of decaBDE (based on dust measurements of BDE-209), TBBPA, and DeBDethane via ingestion of and dermal contact with indoor dust. The results of these calculations are presented in Table 4 by region and for adults and children separately.

The estimated average daily intake of BDE-209 was approximately 10-fold greater for children than for adults in all regions included in these calculations. For children, the highest average daily intake for the combined ingestion and dermal pathways was estimated based on indoor dust data collected in the UK (451 ng/kg-day of BDE-209). This value was approximately 75 times greater than the estimated average daily intake for the combined two pathways calculated based on data collected in North America (6.0 ng/kg-day). The indoor dust data collected in other European countries yielded an even lower average daily intake estimate of 2.2 ng/kg-day for children for the combined two pathways. Estimates of average daily intakes for the combined two pathways for adults were similarly higher based on UK data than on North American data, or data from other European countries (i.e., 54, 0.66, and 0.26 ng/kg-day, respectively).

The substantially greater intake estimates generated based on UK data are largely driven by two elevated dust measurements collected in two of the 30 homes sampled by Harrad et al. [3]. Because these researchers resampled these homes approximately nine months after the initial sampling and found similarly elevated concentrations in those samples, these concentrations were retained in the exposure analysis. As can be seen in Table 3, the North American dust data reflected results from six studies and reflected approximately half as many studies (three) and samples (43). While the geometric mean concentrations in North America and the UK differed by approximately a factor of five, the 95th percentile values of the two distributions differed by a factor of 60.

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Estimated Daily Intake of BDE-209, TBBPA, and DeBDethane</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average Daily Intake (ng/kg-day)</td>
<td></td>
</tr>
<tr>
<td>Ingestion</td>
<td>Dermal Contact</td>
</tr>
<tr>
<td>Mean</td>
<td>95th Percentile</td>
</tr>
<tr>
<td>Adult</td>
<td>Child</td>
</tr>
<tr>
<td>North America</td>
<td>BDE-209</td>
</tr>
<tr>
<td></td>
<td>Europe (except UK)</td>
</tr>
<tr>
<td></td>
<td>UK</td>
</tr>
<tr>
<td></td>
<td>TBBPA</td>
</tr>
</tbody>
</table>
Reflecting the lower measured dust concentrations, lower intake estimates were calculated for TBBPA and DeBDethane. For each of these two FRs, only one study was identified providing adequate residential dust data for use in the exposure calculations. Both of these studies were conducted in the UK. For both FRs, the average daily intake estimates for the combined ingestion and dermal pathways were approximately 10 times greater for children than for adults based on dust measurements. The intake estimates for DeBDethane were higher than those for TBBPA. Specifically, the average daily intake of DeBDethane estimated for young children was 0.99 ng/kg-day, while that for TBBPA was 0.26 ng/kg-day. 95th percentile intake estimates for young children were 3.1 ng/kg-day for DeBDethane and 0.80 ng/kg-day for TBBPA. Estimates of adult intake were approximately 10-fold lower in each case.

As shown in Table 4, incidental ingestion of indoor dust was the predominant contributor to the estimated average daily intake of BDE-209, TBBPA, and DeBDethane. Based on the mean intake estimates, approximately 85% of the combined average daily intake values for BDE-209 were associated with incidental dust ingestion, with the remaining 15% associated with dermal contact with dust, reflecting in part the likely low degree of absorption of this chemical through the skin. For TBBPA and DeBDethane, incidental dust ingestion was also the predominant contributor to the combined average daily intake estimates. Because the dermal absorption of these chemicals was assumed to be slightly higher (0.10) than that assumed for BDE-209 (based on more limited available data), the percentage of the combined average daily intake that was associated with incidental dust ingestion was slightly lower (i.e., approximately 60 to 65%).

5. Discussion

Average daily intake estimates generated in the Monte Carlo analysis for indoor dust exposures to BDE-209 ranged from 2.2 to 451 ng/kg-day for young children, and from 0.26 to 54 ng/kg-day for adults. Ninety-fifth percentile combined intake estimates calculated using the Monte Carlo model ranged from 8.5 to 1,154 ng/kg-day for young children, and from 0.96 to 140 ng/kg-day for adults. As summarized in Table 2, intake estimates for BDE-209 presented in the literature ranged from < 0.06 to 3,400 ng/kg-day for young children (with all but two of the mean estimates being < 65 ng/kg-day, and five of the high end estimates being > 100 ng/kg-day) and < 0.01 to 155 ng/kg-day for adults (with most estimates well below 10 ng/kg-day). The literature values were derived primarily based on North American data and, for many of the studies, largely reflected exposures via incidental ingestion of dust – although other exposure pathways were included in some studies. Variations in the literature estimates are due to differences in the exposure pathways examined in the studies, assumptions made for key exposure parameters (such as incidental dust ingestion rates), and assumptions regarding dust concentrations applied in the calculations (e.g., use of mean or upper-bound concentration values). In addition, in contrast to the approach applied in this analysis, many of the intake estimates available in the scientific literature reflect more rudimentary exposure calculations omitting a number of the exposure parameters that would be required for more refined exposure estimates.

As noted above, only one study was found in the scientific literature that presented estimates of intakes for TBBPA [4]. These researchers estimated the mean intake via dust ingestion by toddlers as 0.44 ng/kg-day using a mean dust intake, and as 1.8 ng/kg-day using a high estimated dust intake [4]. Ninety-fifth percentile intake estimates for toddlers were 0.85 ng/kg-day for the mean dust intake and 3.5 ng/kg-day for the high dust intake. For adults, the mean TBBPA intake via dust ingestion was estimated as 0.023 ng/kg-day using a mean dust intake, and as 0.057 ng/kg-day using a high estimated dust intake. Ninety-fifth percentile intake estimates for adults were 0.046 ng/kg-day for the mean dust intake and 0.12 ng/kg-day for the high dust intake. These values are comparable to the intake estimates calculated for TBBPA using the Monte Carlo model, as presented in Table 4.

Similarly, as discussed above, only one study [48] was identified that provided intake estimates for DeBDethane. In this study, the mean intake via dust ingestion by toddlers was estimated as 1.4 ng/kg-day using a mean dust intake estimate and 5.4 ng/kg-day using a high estimated dust intake [3]. Ninety-fifth percentile intake estimates for toddlers were 4.8 ng/kg-day for the mean dust intake and 19 ng/kg-day for the high dust intake. For adults, the mean intake via dust ingestion was estimated as 0.070 ng/kg-day using a mean dust intake, and as 0.17 ng/kg-day using a high estimated dust intake. Ninety-fifth percentile intake estimates for adults were 0.24 ng/kg-day for the mean dust intake and 0.62 ng/kg-day for the high dust intake. Again, these estimates are comparable to those derived for DeBDethane in the Monte Carlo analysis, as summarized in Table 4.

The Monte Carlo analyses presented here focused on a residential exposure scenario as a conservative screening approach for evaluating potential exposures in other settings. The studies identified in the literature provided more limited data for settings other than a residential setting. Specifically, almost 80% of the studies reviewed by Dodge et al. [2] presented residential data, while only eight studies provided data regarding dust concentrations in workplaces (reflecting use of FR-containing products, not manufacturing processes), four studies provided data collected from vehicles, two
studies presented data from schools, and one study provided data from a hotel setting. Based on these data, the concentrations of the FRs of interest in residences were not found to differ significantly from concentrations in other indoor settings, with one exception, i.e., that BDE-209 concentrations in dust collected from cars were found to differ significantly from concentrations in other settings.

When considering human activity patterns and duration of exposure in the non-residential settings assessed in this analysis, exposure estimates would likely be less than those estimated based on residential scenarios. For example, in this Monte Carlo analysis, individuals were assumed to spend essentially all of their time within a residential setting. For the other settings considered in this analysis (e.g., schools/day care centers, workplaces, and vehicles), only a part of each day would be spent in these settings. In particular, for most individuals, time spent in vehicles would only be a small fraction of the day. Thus, based on the currently available data, time-averaged exposure estimates for these scenarios would be expected to be less than those calculated in these analyses for the residential scenario.

The analyses presented here focused on three regions in which several studies of indoor dust concentrations of the FRs of interest had been conducted (i.e., North America, the UK, and other European countries). Only a limited amount of data from other areas was identified by Dodge et al. [2], i.e., a single study each providing BDE-209 data from residential settings in Antarctica [49], Australia [15], Kuwait [50], and Singapore [10], and one providing BDE-209 data from a hotel setting in Japan [51]. The mean and range of BDE-209 concentrations observed in these studies were generally well within the range of concentrations observed in the three regions included in the Monte Carlo analyses, with the concentrations observed in Kuwait being less than those included in the Monte Carlo calculations. Thus, application of data from these other regions in the exposure model would be expected to yield intake estimates comparable to or less than those generated in the current analyses.

The impacts of sources of uncertainty in these analyses were also considered. A number of the exposure parameters included in this analysis reflect uncertainty. For example, substantial differences have been observed in measured concentrations of the FRs of interest in indoor dust samples. These differences may arise as a result of actual differences in dust concentrations at different locations (due to different sources or other conditions affecting dust generation) and/or they may be due to variations in sampling methods [19, 24]. Similarly, only limited data are available regarding incidental soil/dust ingestion rates for adults, and little information is available to assess the proportion of total soil/dust intake that is composed of dust. Although not applied in this Monte Carlo analysis, the alternative approach used by Stapleton et al. [14] to quantify dust intake via hand-to-mouth contacts also includes a number of uncertain exposure parameters. Only limited data are available for estimating such parameters as hand-to-mouth contact frequency, transfer efficiency from hand-to-mouth, and fraction of hand that is contacted during each contact event.

Uncertainties also exist regarding exposure parameters needed to estimate dermal exposures to indoor dust, e.g., uncertainties regarding the amount of the FRs of interest that would be absorbed through the skin following dust adherence to the skin. In some cases, uncertainty in the exposure calculations arises because indoor exposures via only dust are estimated by using input parameters derived based on studies of exposures to both soil and dust. For example, in the absence of dust-specific information regarding incidental ingestion or dermal adherence, values based on a combination of exposures to both soil and dust in both indoor and outdoor settings were used.

In light of these uncertainties, several conservative assumptions were applied in the Monte Carlo calculations (i.e., certain assumptions were selected that would be more likely to overestimate than underestimate exposures). For example, the assumptions regarding incidental soil/dust ingestion by young children are based on data from children between the ages of one and four years old, the age range during which the maximum incidental intake of soil/dust is thought to occur. Typical actual ingestion rates in the broader age range included in the Monte Carlo analysis (i.e., children between the ages of zero and six years old) would be less. Because the adult soil ingestion rate estimates were directly based on the children’s soil ingestion rates, the conservative aspects of the children’s estimates also apply to the adult estimates. Moreover, the assumption that adult soil ingestion rates are one-half of those observed in young children between the ages of one and four years old is also likely to overestimate typical soil ingestion by adults. Similarly, the soil/dust adherence factors used in these calculations reflect data collected in studies of young children who had contact with both soil and dust in both indoor and outdoor settings. The values selected for adults reflect data collected in studies of activities likely to include frequent contacts with outdoor soil. These adherence values are likely to be greater than those that would be observed in individuals exposed only to dust in an indoor setting.

An evaluation of the quantitative implications of these uncertainties on the exposure assessment results yields the following observations. Because the underlying exposure algorithm used in the Monte Carlo calculations is a simple linear equation, changes in the assumed values for any specific input parameter result in a corresponding change in the intake estimate for the
affected exposure pathway. For example, if the assumed mean value of the incidental soil/dust ingestion rate is increased (or decreased) by 50%, the mean estimated intake of the FR of interest via incidental ingestion of indoor dust would also increase (or decrease) by 50%. Similarly, if the mean value of an input parameter for the dermal contact pathway (e.g., the dermal absorption fraction) is increased (or decreased) by 50%, then the mean estimated intake of the FR of interest via dermal contact with indoor dust would also increase (or decrease) by 50%. Because the incidental soil/dust ingestion pathway is the primary contributor to the total intake estimates generated for the combined exposure pathways (particularly for BDE-209), changes to input parameters for this pathway would have a greater impact on the intake estimates for the combined pathways. For example, if the mean incidental soil/dust ingestion rate assumption is increased (or decreased) by 50%, the mean estimated intake via both exposure pathways is increased (or decreased) by approximately 30 to 40%. In contrast, if the mean assumption for the dermal absorption fraction is increased (or decreased) by 50%, the mean estimated intake via both exposure pathways is increased (or decreased) by an amount ranging from less than 10 to approximately 20%. These results indicate that uncertainties in the soil/dust ingestion pathway have more significant implications for the overall exposure estimates, and that resolution of these uncertainties will have a more significant impact on deriving more accurate exposure estimates.

One common component of estimating incidental soil/dust ingestion that was not included in the exposure calculations presented here was an assumption regarding the relative bioavailability of the chemicals of interest from ingested dust, i.e., the amount of ingested FR in dust that is actually absorbed into the body. A chemical’s bioavailability, or ingestion absorption efficiency, is influenced by such factors as the species of the chemical, the matrix in which it is present, the amount of time that a chemical is in a matrix, and the route by which exposure occurs. When chemicals are ingested, bioavailability is determined by the amount of a chemical that is dissolved in gastrointestinal fluids and absorbed across the gastrointestinal tract into the bloodstream. An ingested chemical that is adsorbed to soil or some other solid medium may be absorbed less completely than the same ingested dose of the chemical in a more accessible form, such as when dissolved in water or an experimental dosing vehicle such as corn oil.

It is widely recognized that the bioavailability of many organic chemicals in soil/dust tends to be considerably lower than bioavailability from food or water [52-54]. Bioavailability from soil/dust can be affected by a number of factors, including the form of the chemical, its solubility, the size distribution of the ingested soil/dust particles, the type of soil/dust, the degree of encapsulation of the chemical within an insoluble matrix, and the nutritional status of the exposed individual. The need to make adjustments for the reduced bioavailability of compounds in soil/dust has been long recognized in the scientific literature and in regulatory guidance. For example, as early as 1989, US EPA’s Risk Assessment Guidance for Superfund [25] included a discussion of “adjustments for absorption efficiency,” addressing the need to account for differential absorption in instances where “the medium of exposure in the site exposure assessment differs from the medium of exposure assumed by the toxicity value.”

There are limited data regarding the bioavailability of FRs adsorbed onto dust or soil particles; however, the estimated organic-carbon sorption coefficients for BDE-209 ($K_{oc} = 2.76 \times 10^5$ to $4.78 \times 10^7$ L/kg), TBBPA ($K_{oc} = 2.70 \times 10^0$ to $1.74 \times 10^5$ L/kg), and DeBDethane ($K_{oc} = 2.40 \times 10^5$ to $6.88 \times 10^8$ L/kg) suggest that these FRs will strongly adsorb to organic matter in soil or dust, and will have reduced bioavailability [55]. One recent study conducted by Huwe et al. [56] provides some data estimating the absorption of various PBDEs (including BDE-209) in rats following exposures via spiked samples of household dust (NIST Standard Reference Material 2585) or a corn oil solution [56]. The concentrations of 15 PBDE congeners were measured in adipose tissue, the liver, feces, and urine after 21 days of exposure via diet (1 or $6\mu g/kg$). The absorption of PBDEs in the tissues was congener-dependent and was found to be lowest for BDE-209 (i.e., < 5%). Furthermore, the researchers found that the degree of absorption did not differ with either dose level or dose vehicle (i.e., administering the compound in dust or corn oil). The authors reported that the absorption of BDE-209 was consistent with two previous studies. In addition, several earlier studies in rats have also determined that BDE-209 is very poorly absorbed following oral ingestion (i.e., < 1%) [57].

As noted above, the choice of a relative bioavailability factor is important when risk estimates will be derived, and will depend on the specific toxicity data used to develop the toxicity value used in calculating the risk estimates. If the bioavailability of the material of interest in the exposure calculations is similar to that of the exposure material used to generate the selected toxicity data, then a relative bioavailability factor is not necessary. Because the exposure estimates calculated in this Monte Carlo analysis were not applied in risk calculations, no toxicity data were selected and the need for a bioavailability factor was not assessed. Based on the available data indicating limited gastrointestinal absorption of several FRs, however, it should be noted that the absorbed dose following incidental ingestion of these compounds will be far less than the amounts suggested by the intake estimates derived in the absence of any bioavailability assumption.
When considering the necessary information for deriving a risk estimate, the data from the Huwe et al. [56] study suggest that a relative bioavailability factor may not be necessary for BDE-209. Huwe et al. [56] found no difference in the absorption of BDE-209 based on the dose vehicle used (i.e., dust vs. corn oil). Much of the available toxicity data for BDE-209 is derived from studies in which the FR was administered in an oil vehicle. The Huwe et al. [56] study data suggest that absorption under the experimental conditions of the toxicity studies would be expected to be similar to absorption following incidental ingestion of dust. As a result, the bioavailable dose for a person ingesting the studied FRs in dust would be similar to the bioavailable dose for an animal in a toxicity study in which the FR was administered in a dosing vehicle (e.g., an oil emulsion).

6. Conclusion

Building upon a review of efforts to date to characterize human exposures to FRs in indoor dust, this paper presents a probabilistic model for quantifying potential intake of FRs in indoor dust, providing both a methodology for developing exposure estimates and a framework for exploring data needs for improving such estimates. Using data compiled in Dodge et al. [2], exposure estimates were derived for incidental ingestion of and dermal contact with three FR compounds in a residential setting, based on data collected in residences in North America, the UK, and other European countries.

These exposure analyses indicate that estimates of young children's exposures to decaBDE, TBBPA, and DeBDethane in residential indoor dust are approximately 10 times greater than those estimated for adults. Paralleling the underlying dust data, exposure estimates based on data collected in the UK are greater than those based on data from North America or other European countries. In addition, exposure estimates for decaBDE are greater than those for TBBPA and DeBDethane.

The results from the exposure analyses also support previous evaluations pointing to incidental soil/dust ingestion as the primary exposure pathway contributing to overall exposures to FRs in indoor dust. Based on mean intake estimates, this pathway contributed approximately 85% of the total intake for BDE-209, while exposures via dermal contact with dust contributed only 15%. For TBBPA and DeBDethane, incidental soil/dust ingestion contributed approximately 60 to 65% of mean total intake, while dermal contact contributed the remainder. This finding indicates that further resolution and refinement of the input parameters required to assess the incidental dust ingestion pathway will be important for developing more accurate FR exposure estimates. In particular, if indoor FR sources and consequent exposure pathways continue to be implicated as significant sources of human exposure, it will be important to more accurately understand the portion of total soil/dust ingestion that is derived from incidental ingestion of indoor dust. A related issue is the collection of coordinated indoor dust and outdoor soil concentration data to develop a more rigorous understanding of the relative concentrations (and exposure potential) of these two media.

Based on its lesser contribution to overall intake estimates, advances in the understanding of input parameters required for assessing FR exposures via dermal contact with indoor dust will have a smaller impact on refining exposure estimates. To the extent that some FRs may be more extensively absorbed through the skin than is likely for the FRs examined in this analysis, however, additional data for certain input parameters for this pathway may be more important in refining exposure estimates. In particular, data are currently sparse regarding the degree to which some FR compounds are absorbed through the skin. In addition, available estimates of soil/dust adherence to the skin are derived from data collected in observational studies of activities involving exposures to both dust and soil in indoor and outdoor settings. As a result, exposure calculations could also benefit from a better understanding of dermal adherence of indoor dust to the skin.

Only a few of the currently available analyses in the scientific literature have attempted to assess the potential health risks associated with the exposure estimates that have been derived. Such evaluations are critical for understanding the degree of potential health hazard associated with the FR concentrations that have been measured in indoor environments. As efforts to characterize human exposures to FRs evolve to focus more extensively on risk characterization, additional evaluations may be necessary to better evaluate the bioavailability of FRs adsorbed to dust to better understand FR intake and to derive more refined risk estimates.

Finally, as discussed in Dodge et al. [2], significant gaps exist in the information available for specific FR compounds or exposure settings. For example, no indoor dust data were found for three of six FR compounds of interest. In addition, inconsistencies in currently used sampling protocols hamper exposure assessments based on the available data. Use of standardized sampling protocols and data quality standards would enhance the comparability of collected data as well as the exposure and risk analyses generated based on those data. The assessment of potential human exposures to FRs in indoor dust would also benefit from consideration of whether other exposure scenarios exist that might have unusual exposure characteristics meriting more detailed evaluation.
7. Acknowledgment

This work was primarily funded by Albemarle Corporation. Albemarle Corporation is a global specialty chemical manufacturer whose product line includes flame retardants. This review represents the individual professional views of the authors and not necessarily the views of Albemarle Corporation.

8. References


