Human Exposure to Brominated Flame Retardants

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Presentation Outline:

1. Background on brominated flame retardants with a focus on polybrominated diphenyl ethers (PBDEs)
   - types, use, commercial formulations
   - toxicology
   - trends in human body burdens in US

2. Exposure to PBDEs (current collaborative project)
   - dietary exposure vs. indoor exposure
   - estimating exposure to PBDEs from air and dust
   - identifying sources of PBDEs in indoor environments

3. Exposure to New/Alternate BFRs

4. Summary and Conclusions
Statistics:

- Every year in the U.S. there are over a million fires reported
- Direct losses account for billions in damages

Flame Retardants:

Definition:
“A substance added or a treatment applied to a material in order to suppress, significantly reduce or delay the combustion of the material”  
*EHC:192, WHO 1997*
How do Flame Retardants Work?

Fuel + O₂ → Heat + CO₂ + H₂O

*Formation of radical species
How do Flame Retardants Work?

• Most common method for retarding fire is to quench the radical species formed in the fire reaction.

Fuel + O₂ → Heat + CO₂ + H₂O

*Stop the formation of radical species

(www.bsef.com)
Types of Brominated Flame Retardants (BFRs)

**REACTIVE BFRs:**
- Chemically bound to the product they are flame retarding....less likely to leach out into the environment

**ADDITIVE BFRs:**
- Mixed in with the resin during extrusion process.....more likely to leach out of products over time

Examples:  
- PentaBDE  
- OctaBDE  
- DecaBDE  

Commercial Mixture Names
Types of Brominated Flame Retardants:

**ADDITIVE BFRs**
- Decabromobiphenyl
- Decabromodiphenyl ethane
- **Decabromodiphenyl ether**
- Octabromodiphenyl ether
- Pentabromodiphenyl ether
- Tetrabromobisphenol A Derivatives
  - bis-(2,3-dibromopropyl ether)
  - bis-(2-hydroxyethyl ether)
  - bis-(allyl ether)
  - dimethyl ether
- Hexabromocyclododecane
- Bis(tribromophenoxy)-ethane
- Pentabromotoluene
- Bromo-chlorinated paraffins
- **Di-(2-ethylhexyl)tetrabromophthalic ester**
- Ethylene-bis-(tetrabromophthalimide)
- Tetradecabromodi phenoxybenzene
- 1,2-Dibromo-4(1,2 dibromomethyl) cyclohexane
- Ethylene-bis(5,6-dibromo-norbornane-
  - 2,3-dicarbox imide
- 1,3,5-tris(2,3-dibromo-propanyl)-2,4,6-triazine

**REACTIVE BFRs**
- Tetrabromobis phenol A
- Tetrabromobisphenol S
- 2,4-Di-, 2,4,6-Tri- and pentabromophenol
- Tribromoneopentyl alcohol
- Vinylbromide
- Tribromophenyl allyl ether
- 2,3-Dibromo-2-butene-1,4-diol
- Tetrabromophthalic acid Na salt
- **Tetrabromophthalic anhydride**
- N,N’-Ethylene-bis-(tetrabromophthal imide)
<table>
<thead>
<tr>
<th>Resins and Polymers</th>
<th>DecaBDE</th>
<th>OctaBDE</th>
<th>PentaBDE</th>
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<tbody>
<tr>
<td>Acrylonitrile-butadiene styrene</td>
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<td>X</td>
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<td>Epoxy-resin</td>
<td>X</td>
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<td>Phenolic resins</td>
<td>X</td>
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<td>X</td>
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<tr>
<td>Polyacrylonitrile</td>
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<td>Polyamide</td>
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<td>Polybutylene terephthalate</td>
<td>X</td>
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<tr>
<td>Cross Linked Polyethylene</td>
<td>X</td>
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<td>Polyethylene terephthalate</td>
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<tr>
<td>Polypropylene</td>
<td>X</td>
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<tr>
<td><strong>Polystyrene/HIPS</strong></td>
<td>X</td>
<td>X</td>
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<td>Polyvinylchloride</td>
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<td><strong>Polyurethane</strong></td>
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<td>Unsaturated polyesters</td>
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<td><strong>Textiles</strong></td>
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</tbody>
</table>

From: EBFRIP, 1990; Rahman et al., 2001
PBDE Nomenclature

Polybrominated Diphenyl Ether (PBDE)

PentaBDE Congeners:

2,2',4,4'-tetrabromodiphenyl ether
BDE-47

2,2',4,4',5-pentabromodiphenyl ether
BDE-99

2,2',4,4',5,5'-hexabromodiphenyl ether
BDE-153

DecaBDE:

BDE 209
<table>
<thead>
<tr>
<th>Congener (# Br)</th>
<th>Percent of Total</th>
<th>Types of Products</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Penta-BDE Commercial Mixture</strong></td>
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<tr>
<td>BDE-47 (4)</td>
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<td>BDE-85 (5)</td>
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<td>BDE-99 (5)</td>
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<td>BDE-100 (5)</td>
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<td>BDE-153 (6)</td>
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<td>BDE-154 (6)</td>
<td>9.3</td>
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<td>hexa-BDE</td>
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<td><strong>Octa-BDE Commercial Mixture</strong></td>
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<td>BDE-153 (6)</td>
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<tr>
<td>BDE-154 (6)</td>
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<td>BDE-183 (7)</td>
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<td>2 hepta-BDEs</td>
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<td></td>
</tr>
<tr>
<td>3 octa-BDEs</td>
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<tr>
<td>BDE-207 (9)</td>
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<td><strong>Deca-BDE Commercial Mixture</strong></td>
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<tr>
<td>BDE-209 (10)</td>
<td>&gt;97</td>
<td></td>
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</tbody>
</table>
Commercial Word Market Demand (2003) for PBDE Commercial Mixtures (Metric Tons)

Source: www.bsef.com

**Penta- and Octa-BDE commercial mixtures currently banned or phased out in U.S.**
Potential Toxicity Observed in Laboratory Studies:

Evidence of developmental neurotoxicity (Viberg and Eriksson et al., 2002, 2003, 2005)

- PBDEs can pass the blood/brain barrier and accumulate
- Neonatal exposure can induce persistent aberrations in spontaneous behavior, and also affect learning and memory functions in rodents
- One time oral exposure during neonatal period results in effects that worsen with age

“Critical window of development associated with “Brain Growth Spurt”

**European study finds PBDE levels in mothers milk positively associated with Cryptorchidism (Main et al., 2007)

Observed thyroid toxicity:

- *In vivo* exposure in fish, rodents and birds leads to reduced levels of circulating hormones (thyroxine and triiodothyronine, T4 and T3)
- Induction of UDPGT enzyme activity (clearance of T4 and T3)
- CYP 2B enriched liver microsomes can metabolize BDEs to hydroxylated forms which are very potent competitors for transthyretin
- Activate PXR and steroid X receptors but not AhR

In general, the lower the degree of bromination, the more potentially toxic the compound……..however, debromination may lead to increased toxicity in environment
In vitro assays have shown that thyroid-hormone-like HO-BDEs can competitively bind to thyroid transport proteins.
Wildlife Exposure to PBDEs:
Historically, there has been a stronger focus on POPs in aquatic systems.

However, the terrestrial environment may be receiving higher exposure to DecaBDE.
DecaBDE does accumulate in Aquatic Organisms and recent evidence Suggests Biomagnification

Lake Winnipeg

Fig. 5. Relationship between concentration (ng/g, lipid and control corrected; mean ± standard error) of decabromodiphenylether (BDE) 209 and trophic level (TL) for the Lake Winnipeg (Canada) food web. Regression analysis: $\ln([BDE\ 209]) = 2.2804(TL) - 1.2225$ ($r^2 = 0.7359, p = 0.01$).
Environmental Levels of BDE 209 in Wildlife

Grizzly Bears Along British Columbia (Christensen et al., 2005):

ΣPentaBDEs: 0.2 to 5 ppb lipid

DecaBDEs: 0.1 to 42 ppb lipid

** Terrestrial feeding bears have higher BDE 209 concentrations in their tissues (as much as 90% of the burden was BDE 209)

Red Foxes (Voorspoels et al., 2006): (sampled ~30 individuals)

ΣPentaBDEs: 2 to 3 ppb lipid

DecaBDEs: <DL to 760 ppb lipid

**BDE 209 was the dominant congener (~80%) in almost half the foxes tested
Environmental Levels of BDE 209 in Birds

显著的生物积累会发生在一些陆地食物链中，尤其是当充足的多氯联苯（Deca-）的来源存在时。

(数据来自Chen et al., 2007)

a. Voorspoels et al., ES&T, 2006, 40, 2937
b. Voorspoels et al., Environ. Pollut. 2006, 114, 218
c. Vorkamp et al., ES&T, 2005, 39, 8199
d. Lindberg et al., ES&T, 2004, 39, 93
Human Exposure to PBDEs:
What are the Issues?
Exposure to Persistent Organic Pollutants:

- Industrial pollution
- Pesticide applications
- Diet
- Bioaccumulation
- Sediment
What about Indoor Exposure to New POPs?

- Work Environment
- Vehicles
- Homes
- House Dust
PBDEs in Human Samples From Around the World (primarily pentaBDE)
Data from Hites et al., 2004

Total PBDE concentrations in human blood, milk and tissue (in ng/g lipid) shown as a function of sampling year.

Issue 1: PBDEs were doubling about every 5 years; recent leveling observed
Issue 2: U.S. and Canadian populations have the highest accumulation of PBDEs
### What Levels of DecaBDE Have Been Measured in People?

**Measured in ppb lipid** (%<DL - less than detection limits)

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Population</th>
<th>ΣPenta</th>
<th>ΣDeca</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast Milk</td>
<td>U.S. adults (n=47)</td>
<td>6-420</td>
<td>&lt;DL to 8</td>
<td>2003 Schecter et al.</td>
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<tr>
<td></td>
<td></td>
<td>Mean 62</td>
<td></td>
<td></td>
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<tr>
<td>Serum NHANES</td>
<td>U.S. Adults (n=2062)</td>
<td>&lt;DL to 3680</td>
<td>NM</td>
<td>2008 Sjodin et al.</td>
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<tr>
<td></td>
<td></td>
<td>Median = 34</td>
<td></td>
<td></td>
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<tr>
<td>Adipose</td>
<td>U.S. Adults (n=52)</td>
<td>17 to 10,000</td>
<td>NM</td>
<td>2005 Johnson-Restrepo et al.,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean 400</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum</td>
<td>Swedish Workers</td>
<td>&lt;DL to 15</td>
<td>1 to 140</td>
<td>1999 Sjodin et al.</td>
</tr>
<tr>
<td>Serum</td>
<td>Swedish Workers</td>
<td>NM</td>
<td>3 to 230</td>
<td>2005 Thuresson et al</td>
</tr>
<tr>
<td>Serum</td>
<td>U.S. Foam Workers And Carpet Installers</td>
<td>1 to 7000</td>
<td>&lt;DL</td>
<td>2008 Stapleton et al. (In Press)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean 212</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood</td>
<td>Japanese Adults</td>
<td>0.3 to 6.6</td>
<td>1.3 to 31</td>
<td>2004 Takasuga et al.</td>
</tr>
</tbody>
</table>
What are the Levels of PBDEs in Children?  
A Case Study from Berkeley, California

Data from Fisher et al., 2006

Blood Levels Measured in ppb lipid

<table>
<thead>
<tr>
<th></th>
<th>Date Measured</th>
<th>ΣPenta</th>
<th>ΣDeca</th>
<th>ΣPBDEs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father</td>
<td>Sept. 04</td>
<td>64</td>
<td>23</td>
<td>87</td>
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<tr>
<td></td>
<td>Dec. 04</td>
<td>71</td>
<td>3</td>
<td>74</td>
</tr>
<tr>
<td>Mother</td>
<td>Sept. 04</td>
<td>106</td>
<td>14</td>
<td>120</td>
</tr>
<tr>
<td></td>
<td>Dec. 04</td>
<td>142</td>
<td>4</td>
<td>146</td>
</tr>
<tr>
<td>Daughter</td>
<td>Sept. 04</td>
<td>247</td>
<td>143</td>
<td>390</td>
</tr>
<tr>
<td></td>
<td>Dec. 04</td>
<td>244</td>
<td>11</td>
<td>255</td>
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<tr>
<td>Son</td>
<td>Sept. 04</td>
<td>418</td>
<td>233</td>
<td>651</td>
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<tr>
<td></td>
<td>Dec. 04</td>
<td>482</td>
<td>23</td>
<td>505</td>
</tr>
</tbody>
</table>

<DL -less than detection limits

Exposure Modeling Suggest Children are Receiving 10X greater exposure to PBDEs (Jones-Otazo et al., 2005)
**BDE 209 not typically
The most abundant congener
In U.S. population…

**However, Japan and China
Use Primarily DecaBDE in
Products and no PentaBDE.
Tissues in the Japanese and
Chinese populations have a
Strong contribution from
BDE 209…….

**Suggests U.S. may see shift
If DecaBDE not banned from
Use.
BDE 209 Measured in U.S. Food Items:
(Huwe et al., 2002 and Schecter et al., 2006)

**Dairy Products**
- Butter: 66 ppt
- Cream Cheese: 482 ppt
- Milk: <DL to 6 ppt
- Cheese: <DL to 18 ppt
- Eggs: 10 ppt

**Meats**
- Bacon: <DL to 28 ppt
- Ground Meats: <DL to 485 ppt
- Pork Sausage: <DL to 50 ppt
- Chicken Breast: 48 ppt
- Whole Chicken: 300 to 3400 ppt

**Fish**
- Wild Salmon: <DL
- Farmed Salmon: 20 to 681 ppt
- Canned Tuna: 5 to 9 ppt
- Fresh Tuna: 23 ppt
- Shrimp: <DL
- Tilapia: <DL
PBDEs Measured in Food Items: (Gomara et al., 2006)

Food Purchased in Spanish Supermarkets

FIGURE 1. Percentages of contribution of the 15 congeners of BDE to the total BDEs in (a) fish and shellfish, (b) meats, (c) dairy products, and (d) oils and butters.
Are Children Receiving Greater Exposure to DecaBDE from House Dust?

Children Place Their Hands, Bottles, Toys, (etc.) in Mouth

Inadvertent ingestion of PBDEs associated with Dust
## Measurement of PBDEs in Dust (ng/g dry weight, ppb)

<table>
<thead>
<tr>
<th>Type of Dust</th>
<th>Study Location</th>
<th>Range $\Sigma$PBDEs</th>
<th>Range BDE 209</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>House</td>
<td>USA</td>
<td>700 - 69,000</td>
<td>143 – 66,000</td>
<td>Schecter et al., 2005</td>
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<tr>
<td>House</td>
<td>USA (n=60)</td>
<td>780 - 31,000</td>
<td>160 - 8750</td>
<td>Stapleton et al., 2005</td>
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<tr>
<td>House</td>
<td>USA (n=17)</td>
<td>200 - 569,000</td>
<td><strong>60 - 544,000</strong></td>
<td>Allen et al., 2008*</td>
</tr>
<tr>
<td>House</td>
<td>Canada (n=68)</td>
<td>170 - 170,000</td>
<td>74 – 10,000</td>
<td>Wilford et al., 2005</td>
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<tr>
<td>House</td>
<td>Germany</td>
<td>25 - 25,000</td>
<td>20 – 19,100</td>
<td>Knoth et al., 2003</td>
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<tr>
<td>House</td>
<td>Kuwait</td>
<td>1 - 390</td>
<td>0.8 - 340</td>
<td>Gevao et al., 2006</td>
</tr>
<tr>
<td>Car</td>
<td>USA</td>
<td></td>
<td>9500</td>
<td>Gearhart et al., 2006</td>
</tr>
</tbody>
</table>

* Allen et al., 2008 – In Review
What are the Toxic Thresholds and Margins of Safety for BDE 209?

Evidence of Developmental Toxicity (Viberg et al., 2003):

- BDE 209 can pass the blood/brain barrier and accumulate
- can result in altered behavior, memory and learning in mice
- one time oral exposure during neonatal period results in effects that worsen with age

“Critical window of development associated with “Brain Growth Spurt”

- effects observed at doses of 20 mg/kg body weight
- National Academy of Sciences Reference Dose = 4 mg/kg body weight
- EPA IRIS Reference Dose for BDE 209 (2007) = 0.01 mg/kg/body weight/day

*Concentrations of BDE 209 measured in dust as high as 0.5 mg/g dust (assume ingest 100 mg/day in child = 0.05 mg/day)
Collaborative Research Project: Exposure to PBDEs in Indoor Environments

**Collaborative research project between H.M. Stapleton and colleagues at Boston University School of Public Health**

Objectives:

1. Compare indoor air and personal air levels of BDEs

2. Examine differences in BDE levels in dust collected from different rooms, over seasons and using different collection methods

3. Use XRF technology to determine sources of BDEs in the home environment

4. Quantify relative exposure via inhalation, dust ingestion and hand to mouth contact.
PBDEs in Indoor Air

We are inhaling PBDEs in indoor air

~3.5 ng/day of decaBDE
~10 ng/day of pentaBDE (assumed inhalation rate of 20 m³/day).

**Presence of personal “dust clouds” results in higher exposure than predicted by large volume air samplers.

(Data from Allen et al., 2007)

20 participants from Boston, MA

[Published in ES&T]
References:

- Standberg et al., 2001
- Wilford et al., 2004
- Harrad et al., 2006
### Findings:

- Differences in collection method
- Higher levels in living room
- No significant seasonal differences

**Concentration units: ppb (ng/g)**

(Currently in Review for Publication)
Characterizing PBDE Sources in the Home

XRF – X-Ray Fluorescence:

• Technology used to monitor lead in homes
• Analysis specific to each element

XRF Application to Identifying PBDE Sources:

1) Validate XRF method

2) Use to determine [Br] in products found within the home (e.g. TVs, electronics, furniture, carpets, mattresses, etc.)

3) Determine if dust PBDE levels correlate to [Br] measured by XRF (Currently in Review for Publication)
XRF Validation Pilot Study:

Results: XRF measured bromine was highly correlated with GC/MS measured bromine

Red points indicate TBBPA measured bromine by GC/MS
Figure 2.

(2a) \[ r = 0.12 \quad p = 0.63 \]

(2b) \[ r = 0.56 \quad p = 0.01 \]

(2c) \[ r = 0.68 \quad p = 0.001 \]
Figure 3.

(a) $r = 0.43$, $p = 0.07$

(b) $r = 0.49$, $p = 0.03$

(c) $r = 0.64$, $p = 0.003$
Therefore… items in our home are likely contributing to PBDE levels in dust…… but how does that translate to exposure???
Estimates of PBDE exposure from dust are poor and assume an ingestion rate of dust/day (e.g. 100 mg dust/day)

However, PBDEs may be adsorbing directly to surface oils of skin from contact with PBDE laden products (i.e. remote controls, keyboards, )

Better estimates are needed to quantify hand to mouth transfer of PBDEs

Objectives:

1. Determine if PBDEs were detectable on hand wipe samples

2. Examine distribution of PBDE mass present on hand surface area among 30 individuals

3. Estimate exposure to PBDEs via hand to mouth contact using hand wipe measurements.
Hand Wipe Sampling and Methods:

Wipe Sample From Top of Hand

Wipe Sample From Bottom of Hand

- 33 volunteers/participants
  - 6 children (8-11 yrs), two families of four
  - 3 individuals- repeated sampling & top/bottom comparison
-Sterile gauze pads soaked in 3 mL isopropyl alcohol
-Wipe entire surface area of hand from wrist to finger tips
-Extract with 50 mL dichloromethane (3X by sonication)
-Clean-up extract using 6% deactivated alumina resin
-Analyze by gas chromatography/electron capture negative ionization mass spectrometry (GC/ECNI-MS) for suite of 35 PBDE congeners
Results:

Range: 3 to 1980 ng total PBDE
Among all individuals

Children Only (n=6):
  Range: 59 to 560 ng total PBDE
  Median: 138 ng

BDEs 47, 99 and 100 Contribute average of 67% of total BDEs

BDE 209 Levels:
  Min: <DL
  Max: 270 ng
  Median: 25.9 ng
  Mean: 42.2 ng

In one individual, 94% of ΣBDE was from BDE 209
Concentrations Among Families:

- PBDE mass normalized to hand surface area
- Hand surface area calculated based on height, weight and gender
- Hand surface area is not a variable affecting differences in PBDE loadings
Repeated Wipe Collections from 3 individuals:

- Collected over two month period
- Some individuals consistent, others are not

<table>
<thead>
<tr>
<th>Total PBDE (ng)</th>
<th>Adult Female Age 30</th>
<th>Male Child Age 10</th>
<th>Adult Male Age 32</th>
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</tbody>
</table>

- Collected over two month period
- Some individuals consistent, others are not
Distribution of PBDEs on Hands

PBDE Levels on Top and Bottom of Hands

- Adult Female Age 30
- Male Child Age 10
- Adult Male Age 32

Distribution of PBDEs on Hands

- % on Top of Hands
- % on Bottom of Hands
### Exposure Parameters for Hand to Mouth Contact

<table>
<thead>
<tr>
<th></th>
<th>Child (1-4)</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mouthing events per hour*</td>
<td>18</td>
<td>2</td>
</tr>
<tr>
<td>Fraction of hand surface area mouthed**</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Hand to mouth transfer efficiency**</td>
<td>10 - 90%</td>
<td></td>
</tr>
<tr>
<td>Hours of Contact per day</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Median PBDE Level on Hand (ng)</td>
<td>130</td>
<td>564</td>
</tr>
<tr>
<td>95th Percentile PBDE Level (ng)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Exposure Rates via Hand to Mouth Contact (ng/day)

#### Assuming Transfer Efficiency of 50%:
- **Median Exposure**: 1380 ng/day for Child, 154 ng/day for Adult
- **95th Percentile Exposure**: 6090 ng/day for Child, 680 ng/day for Adult

#### Assuming Median PBDE Levels on Hands:
- **10% Transfer Efficiency**:
  - Child: 281 ng/day
  - Adult: 32 ng/day
- **90% Transfer Efficiency**:
  - Child: 2530 ng/day
  - Adult: 280 ng/day

* Factors taken from Tulve et al., 2002
** Factors Taken From SHED Model for CCA Treated Wood, Zartarian 2005
Estimates of Median PBDE Intake by Source In U.S. Population

- **Nursing**
  - Assuming an infant weighs 5 kg and ingests 800 mL of breast milk/day (Schecter et al., 2005).

- **Diet**
  - Assuming adult weighs 65 kg and a child weighs 13 kg (Schecter et al., 2006).

- **Inhalation**
  - Assuming an inhalation rate of 20 m³/day (Allen et al., 2007).

- **Dust Ingestion**
  - Assuming that children ingest 100 mg of dust/day and an adult 20 mg dust/day (Stapleton et al., 2005).

- **Hand to Mouth**
  - Using model parameters estimates on previous slide and median BDE levels of 130 ng on hands.
Alternative Flame Retardant Chemicals

Potential Deca-BDE Replacements
• Tetrabromobisphenol A (TBBPA)
• Hexabromocyclododecane (HBCD)
• Decabromodiphenylethane (DBDPE)
• 1,2-Bis(2,4,6-tribromophenoxy)ethane (BTBPE)
• Pentabromoethylbenzene (PBEB)
• Dechlorane Plus (DP)

Potential Penta-BDE Replacements
• Tris(1,3-dichloro-2-propyl)phosphate (TDCPP)
• Triphenylphosphate (TPP)
• Octyl tetrabromobenzoate (OTB)
Hexabromocyclododecane (HBCD)

- High production volume chemical (#3 BFR)
- Additive flame retardant
- Used in polystyrene foams for thermal insulation in buildings (expanded and extruded Polystyrene, EPS and XPS), upholstery textiles, electrical equipment housings
- Detected in human serum ranging from <DL to 850 ng/g lipid (Thomsen et al 2007).

\[ \alpha\text{-HBCD} \sim 6\% \]  
\[ \beta\text{-HBCD} \sim 8\% \]  
\[ \gamma\text{-HBCD} \sim 80\% \]
Decabromodiphenylethane (DBDPE)

Application

• Applications similar to Deca-BDE

Occurrence and Bioaccumulation

• Sewage sludge from Sweden and Canada (10-100 ng/g dry) (Kierkegaard et al. 2004; McCrindle et al. 2004)
• Great Lakes air (Hoh 2006)
• Tree bark in North America (Zhu and Hites 2006)
• Lake Winnipeg food web (Law et al. 2006)
• Not measured in CA or any urbanized estuary

Toxicity? We don’t know; aquatic acute/chronic studies not available
1,2-Bis(2,4,6-tribromophenoxy)ethane (BTBPE)

Application
- Additive flame retardant used in thermoplastics
- Replacement for Octa-BDE (Great Lakes Chemical)

Occurrence and Bioaccumulation
- U.S. air, in concentrations similar to PBDEs (Hoh et al. 2005)
- Great Lakes sediment (Hoh et al. 2005)
- Tree bark in North America (Zhu and Hites 2006)
- Lake Winnipeg food web (Law et al. 2006)
- Herring gull eggs from the Great Lakes (Gauthier et al. 2007)
- Northern Fulmar eggs from the Faroe Islands (Karlsson et al. 2006)
- Between 1979 and 1998, concentrations increased in Ontario lake trout (Tomy et al. BFR 2007)

Toxicity
- Thyroid interference minimal (if any) in juvenile rainbow trout (Tomy et al. 2007)
Brominated compounds in Firemaster 550 [BZ 54]

- 2-ethylhexyl 2,3,4,5-tetrabromobenzoate (EHBT)
- Bis(2-ethylhexyl) Tetrabromophthalate (TBPH)
Summary and Conclusions:

- PBDEs are found at greater concentrations in indoor environments relative to outdoor environments.

- XRF analyses indicates foam is a likely source of PentaBDEs and TVs are likely a source of DecaBDE to indoor dust.

- PBDEs are adsorbed to the surface of the skin and objects we come into contact with on a daily basis (e.g. remote controls, furniture, phones) may lead to increased adsorption of PBDEs to hands.

- Hand to mouth contact is likely not an insignificant route of exposure and behavior that leads to increased hand to mouth contact (e.g. smoking, thumb sucking, finger foods) likely leads to increased exposure via inadvertent ingestion.

- Alternate Brominated Flame Retardant chemicals are being detected in house dust, including BTBPE, DBDPE, HBCD and components of FM 550.

- BDE 209 can be degrade to lower PBDE congeners via photolysis and metabolism.
Dietary exposure to BDE 209 at ~1 ppm/day

Examined uptake of BDE 209 in tissues and measured debrominated metabolites
Food Exposure
1% body weight/day

Retention Time (min)
Figure 6. GC/ECNI-MS chromatograms comparing extracts from BDE 209 exposed rainbow trout (A) and BDE 209 exposed common carp (B).

Exposure to Sunlight Leads to Degradation: (primarily “debromination”)

Congeners found in OctaBDE

BDE 207

BDE 209

BDE 183

BDE 201
Does DecaBDE Degrade When Exposed to Sunlight?

NIST, Gaithersburg, MD
USA, Sept.-Oct. 2005
(Stapleton and Dodder, 2008)

0.5 grams Dust in plastic cuvettes

*Tested the ability of natural sunlight to degrade/debrominate DecaBDE

*Will DecaBDE degradation lead to formation of congeners found in OctaBDE and PentaBDE commercial mixtures (which are more Persistent and potentially more toxic)
Spiked Dust 200 Hours Exposure

Abundance

Retention Time (min)

18 20 22 24 26 28 30

0 1000 2000 3000 4000 5000 6000 7000 8000 9000 10000

I.S.

unknown heptaBDE

unknown octaBDE

183

202

201

197

196

200,203

208

207

206

*Degradation products not found in any commercial mixtures: e.g. BDE 202
Indicators of DecaBDE debromination

*BDE 202 has been measured in house dust (Allen et al., 2006)
## Half-Lives of DecaBDE Among Studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Matrix</th>
<th>Light Source</th>
<th>Half-Life (hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Söderström et al., 2004</td>
<td>Silica gel</td>
<td>UV Lamp</td>
<td>&lt;0.25</td>
</tr>
<tr>
<td></td>
<td>Sand</td>
<td>UV Lamp</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>Sand</td>
<td>Sunlight</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Sediment</td>
<td>UV Lamp</td>
<td>40-60</td>
</tr>
<tr>
<td></td>
<td>Sediment</td>
<td>Sunlight</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>Soil</td>
<td>UV Lamp</td>
<td>150-200</td>
</tr>
<tr>
<td>Eriksson et al., 2004</td>
<td>MeOH/Water</td>
<td>UV Lamp</td>
<td>0.5</td>
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<tr>
<td>Ahn et al., 2005</td>
<td>Montmorillonite</td>
<td>UV Lamp</td>
<td>866</td>
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<tr>
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<td>Montmorillonite</td>
<td>Sunlight</td>
<td>5198</td>
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<td>Kaolinite</td>
<td>UV Lamp</td>
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<td>9780</td>
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<td>UV Lamp</td>
<td>3616</td>
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<td></td>
<td>Sediment</td>
<td>Sunlight</td>
<td>23,760</td>
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<tr>
<td>Stapleton and Dodder, 2008</td>
<td>Dust</td>
<td>Sunlight</td>
<td>408</td>
</tr>
</tbody>
</table>
Is DecaDBE Debromination Environmentally Relevant?

Photolysis of DecaBDE requires wavelengths in the UV range.....some windows block UV Wavelengths.....reduces energy to degrade DecaBDE.

However......

- impossible to exclude all sunlight from homes, offices and automobiles (DecaBDE present in car dust)

-DecaBDE found in sewage sludge and biosolids (up to 5,000 ppb) which are land applied in many regions........will receive sunlight exposure

- DecaBDE found in E&E Waste...landfills receive sunlight exposure leaching from landfills (Danon-Schaeffer et al., 2006) will expose DecaBDE to sunlight.
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All study participants

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- Dr. Nicolle Tulve, EPA