Indoor Sources of and Human Exposure to Brominated Flame Retardants (BFRs)

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1. Introduction

Indoor concentrations of, and exposures to brominated flame retardants (BFRs) are influenced by poorly understood sources and, in some cases, poorly understood exposure pathways. Furthermore, the physical-chemical properties of the compounds vary greatly, influencing their indoor partitioning and residence time, and hence indoor levels, persistence and exposure. Here we report on a study of 51 participants residing in the Greater Toronto Area and Ottawa, Canada. Our goal was to characterize indoor concentrations and to estimate exposure of participants to BFRs.

2. Materials and methods

The sampling and household information collection tools are shown in Figure 1. In the bedroom and most used room we collected and analysed floor dust and air samples using two types of passive air samplers (Harner-type polyurethane foam, PUF, and polydimethylsiloxane, PDMS). We also wiped the surfaces of the most commonly used electronic products to assess their potential for contributing to indoor BFR concentrations, as well as hand wipes of participants to estimate potential hand-to-mouth exposure. An online questionnaire was developed and administered to gather personal demographic, lifestyle data, and environmental/household characteristics. Chemical analysis was done by GC-MS using published methods.

3. Results and discussion

3.1. BFRs Levels and Human Exposure

Detection frequencies of dominant compounds in each sample type were generally $>90\%$. Concentrations (median values) of BFRs in all samples are shown in Table 1. Although overall air concentrations obtained using PUF were higher than those obtained using PDMS passive air samplers, the results using the two methods were significantly correlated for both PBDEs ($r^2=0.98$, $p<0.01$) and “novel” BFRs (NBFRs; $r^2=0.74$, $p<0.01$).
Analysis of PUF and PDMS samples showed that ATE was the most abundant NBFR in indoor air followed by PBZ, PB, and TBB. BDE-47 was the major PBDE congener followed by BDE-99, BDE-28 > BDE-17, BDE-49.

As expected, dust samples showed different patterns from air. NBFRs concentrations decreased in the order of TBP > TB > DBDP > a-DP > s-DP > ATE. Following the significant dominance of BDE-209 were BDE-99 > BDE-47 > BDE-100, -153 > BDE-154 > BDE-85, -49, -183.

Hand wipes showed similar profiles of NBFRs as in dust samples. BDE-209 was also the dominant PBDE congener, followed by BDE-47, -99 > BDE-49, -66, -85, -154. Median values of the abundant compounds in hand wipes and dust were significantly correlated for NBFRs ($r^2=1, p<0.001$), and PBDEs ($r^2=0.77, p<0.01$).

These data were used to estimate participants’ external exposures via inhalation, dust ingestion and hand-to-mouth transfer using calculation methods from literature$^{1,2,3}$. Estimated median exposures to $\Sigma$PBDEs and $\Sigma$NBFRs via dust ingestion was over double and about 1.5 times of that via hand-to-mouth transfer, respectively. Both median dust ingestion and hand-to-mouth exposures exceeded median exposure via inhalation by approximately 100 times.

### Table 1: Concentrations of and Exposure to $\Sigma$NBFRs, and $\Sigma$PBDEs in Household Air (Analysed by PUF and PDMS), Dust, and Hand Wipes from 51 Participants in Ontario, Canada.

<table>
<thead>
<tr>
<th>Levels</th>
<th>NBFRs</th>
<th>range</th>
<th>median</th>
<th>average</th>
<th>Dust (ng/g)</th>
<th>range</th>
<th>median</th>
<th>average</th>
<th>Air (PUF, pg/m³)</th>
<th>range</th>
<th>median</th>
<th>average</th>
<th>Air (PDMS, pg/m³)</th>
<th>range</th>
<th>median</th>
<th>average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure</td>
<td>Hand-to-Mouth Contact (pg/day)</td>
<td>Dust Intake (pg/day)</td>
<td>Inhalation (by PUF, pg/day)</td>
<td>Inhalation (by PDMS, pg/day)</td>
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<tr>
<td>NBFRs</td>
<td>10-9,870</td>
<td>180</td>
<td>530</td>
<td>180-28,400</td>
<td>1,700</td>
<td>3,190</td>
<td>n.d.-1,790</td>
<td>40</td>
<td>88</td>
<td>n.d.-260</td>
<td>26</td>
<td>44</td>
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<td>PBDEs</td>
<td>12-5,100</td>
<td>160</td>
<td>420</td>
<td>130-39,300</td>
<td>2,730</td>
<td>4,650</td>
<td>7-820</td>
<td>46</td>
<td>95</td>
<td>3-240</td>
<td>27</td>
<td>46</td>
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<tr>
<td>Hand Wipes (pg/cm²)</td>
<td>Dust (ng/g)</td>
<td>Air (PUF, pg/m³)</td>
<td>Air (PDMS, pg/m³)</td>
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<tr>
<td>PBDEs</td>
<td>46,300</td>
<td>120,000</td>
<td>740</td>
<td>440</td>
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4. Conclusions

This study conducted in 51 homes in Ontario, Canada provides comprehensive information on the levels of NBFRs and PBDEs in household air, dust and hand surfaces of participants. Estimates of indoor air concentrations in bedrooms and most used rooms obtained using PUF were higher than, but correlated with those obtained using PDMS passive air samplers. Concentration ranges for each chemical category in each sample type crossed 1-3 orders of magnitude. Major BFRs found in air were ATE, BDE-47, BDE-99, and TBB; dust were TBP, TBB, BDE-209, BDE-99, BDE-47; and handwipes had similar patterns as dust.

The dominant exposure pathway estimated for BFRs was dust ingestion, followed by hand-to-mouth transfer. Exposure to BFRs by inhalation was estimated to be considerably less than that by hand-to-mouth transfer and dust ingestion.

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**References**

