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Flame retardant biomarker changes with furniture replacement after flammability standard update

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Abstract

Upholstered furniture has been a major source of flame retardant (FR) exposures in the United States. However, the California update to furniture flammability standards has allowed compliance without relying on chemical additives. We investigated whether FR biomarkers would decrease in participants who replaced furniture foam or upholstered furniture with items manufactured after the policy change. Building off previous work demonstrating decreased dust FR concentrations after furniture replacement, we collected urine and blood from 25 participants prior to furniture replacement and approximately one year after and from a comparison group (n=28) over a similar time frame. Serum was analyzed for 19 polybrominated diphenyl ethers (PBDEs) and urine for 3 metabolites of organophosphate FRs (OPFRs). For BDE-47, BDE-99, and BDE-100, time to decline by half was 1.9–3.9 times longer in the comparison group than the replacement group (equivalent to 1.4 years (median) in the replacement group versus 2.6–5.2 years in the

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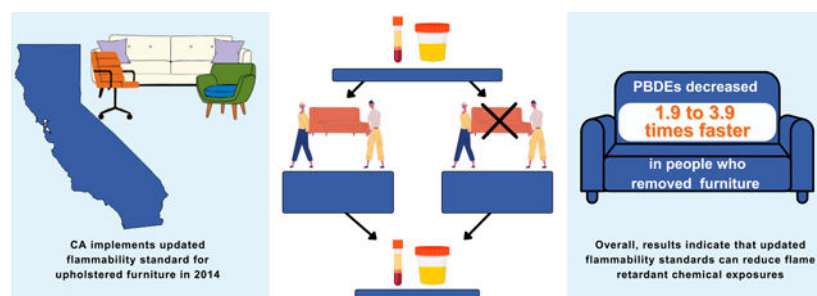
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comparison group). For BDE-153, times to decline by half were not significantly different. For OPFR metabolites, which are excreted within days after exposure, concentration changes were much more variable. Nonsignificant greater decreases were seen for bis(1,3-dichloroisopropyl) phosphate in the replacement group and for diphenyl phosphate in the comparison group, whereas concentrations remained level or increased for bis(2-chloroethyl) phosphate. Moderate positive correlations were observed for baseline PBDE concentrations with baseline dust concentrations ($\rho=0.58\text{--}0.60$); OPFRs were less correlated. Magnitudes of change were mostly positively correlated between dust and biomarker concentrations but not significant. Overall, results indicate that updated flammability standards can reduce FR exposures.

Graphical Abstract



Keywords

flame retardants; intervention study; polybrominated diphenyl ethers (PBDEs); organophosphate flame retardants (OPFRs)

1. Introduction

Chemical flame retardants (FRs) are used in materials such as furniture, textiles, building materials, and electronics to meet flammability standards intended to prevent or slow the spread of fire (Page et al., 2023; Siddiqi et al., 2003; Yang et al., 2019). Polybrominated diphenyl ethers (PBDEs) were used as FRs starting in the 1960s until restrictions began in 2004 with the voluntary manufacturing phase-out in the United States (U.S.) and the 2004 and 2006 bans in the European Union and California, respectively, of penta- and octa-PBDEs (European Union, 2004; Rodgers et al., 2021; State of California, 2005). By the end of 2013, a U.S. EPA-brokered voluntary phase out of deca-PBDE followed. However, use of organophosphate flame retardants (OPFRs) increased as substitutes for PBDEs in this same time period (Hoffman et al., 2017). To allow manufacturers to meet flammability standards without the addition of PBDEs, OPFRs, or other chemical FRs, California implemented its revised Technical Bulletin 117 in 2014 (c) (TB 117–2013), which was then adopted as the federal standard in 2020 (Consumer Product Safety Commission, 2021). Nationwide OPFR use in foam furnishings appears to be declining (Cooper et al., 2016; Gill et al., 2024). However, 19% of the world's OPFR manufacturing facilities are located in the United States and overall production trends differ among various OPFRs, as many are also used for other purposes, such as plasticizers (Gbadamosi et al., 2021; Huang et al., 2022; U.S. Environmental Protection Agency, 2020).

Both PBDEs and OPFRs have been added to polyurethane foam in significant quantities, making up to 3–7% of total weight (Blum et al., 2019). Since these FRs are not chemically bound to product materials, their migration into indoor air and dust (Frederiksen et al., 2009; Yadav et al., 2017) leads to human exposure through dermal absorption, inhalation, and ingestion (Siddiqi et al., 2003; Yang et al., 2019). PBDEs are persistent in both the environment and the human body, with congener biological half-lives ranging from 1.8–6.5 years and up to 39 years in the environment (Geyer et al., 2004; Palm et al., 2002). In contrast, OPFRs are less biologically persistent, generally with half-lives in the human body on the order of hours (Van den Eede et al., 2013). Less is known about the environmental half-lives of OPFRs, though around 120 days is estimated depending on environmental conditions (Liagkouridis et al., 2015). These compounds also appear to be readily accumulating in the environment and in many media including food (Blum et al., 2019; Gbadamosi et al., 2021).

Exposure to both classes of chemicals is widespread. In lab measurements from the 2015–2016 National Health and Nutrition Examination Survey (NHANES), representative of the U.S. population ages 12 and older, 4 PBDE congeners were detected in the serum of 100% of the pooled samples and 3 more in a majority (Centers for Disease Control and Prevention, 2022). However, levels have been showing decreases since 2005 (Sjödin et al., 2019). OPFRs are likewise commonly observed in biological samples, with 3–4 metabolites detected in a majority of participants' urine and two in >95% in the 2015–2016, 2017–2018, and 2017–March 2020 NHANES cycles (Centers for Disease Control and Prevention, 2022; National Center for Health Statistics, 2021).

PBDE exposure has been linked to many human health impacts, including cancer and neurodevelopmental delays (Gibson et al., 2018; Liu et al., 2024; Renzelli et al., 2023). Penta-BDE, specifically the pentabromodiphenyl ether mixture [DE-71 (technical grade)], has been included on California's Proposition 65 list based on liver, thyroid, and pituitary tumors observed in rodent studies (California Office of Environmental Health Hazard Assessment; National Toxicology Program, 2016). A recent study of human cancer mortality showed increased risk with the highest tertile of serum sum of 10 PBDE levels (Liu et al., 2024). Prenatal or early childhood concentrations of PBDEs have been associated with lower IQ scores, executive function, and working memory, as well as with behavioral issues such as hyperactivity, impulsivity, and inattention (Gibson et al., 2018; Linares et al., 2015). Additionally, exposure to PBDEs has also been associated with altered thyroid and other hormone levels (Linares et al., 2015). In animal studies PBDE exposure also has been linked to reproductive effects such as lower sperm motility and count, lower fetal and maternal weight gain, and delayed onset of puberty (Linares et al., 2015).

While there are fewer health studies published on OPFRs due to their more recent use as flame retardants in upholstered furniture/foam, data from toxicity testing and recent epidemiological studies are concerning (Blum et al., 2019). Toxicological evidence has been sufficient for tris(2-chloroethyl) phosphate (TCEP) and tris(1,3-dichloroisopropyl) phosphate (TDCIPP) to be classified as carcinogens under Proposition 65 in California (California Office of Environmental Health Hazard Assessment). Some OPFRs have also been associated with neurodevelopmental impacts in epidemiological studies, exhibited as

lower IQ scores, hyperactivity, and other neurobehavioral outcomes (Vuong et al., 2020). Additionally, associations have been noted with asthma and allergy exacerbation and reproductive and birth outcome impacts, including preterm birth and large-for-gestational age (Araki et al., 2018; Carignan et al., 2018; Ingle et al., 2018; Luo et al., 2021; Meeker & Stapleton, 2010; Oh et al., 2024; Varshavsky et al., 2021). Lastly, animal models also indicate OPFRs alter thyroid hormone functioning (Vuong et al., 2020).

Our team previously demonstrated significant declines in PBDE and OPFR dust concentrations after replacement of FR-containing furniture or foam with non-FR models (Rodgers et al., 2021). While the presence of FR-treated furniture in the home has been linked to higher biomarker concentrations of residents (Dodson et al., 2014; Hammel et al., 2017; Johnson et al., 2010; Meeker et al., 2013), no studies have yet examined the extent to which biological concentrations of FRs can be impacted by the replacement of FR-containing products in the home. To explore the impact of exposure to FR-treated products on body burden, we measured serum PBDE and urinary OPFR concentrations before and after replacement of participants' FR-containing furniture in a main living space with non-FR furniture manufactured after 2014 and contrasted changes with a comparison group who did not replace FR furniture.

2. Materials and methods

2.1 Study design and population

This study built on an investigation of flame retardant concentrations in dust in homes where furniture foam or entire pieces of furniture were replaced in a main living space (Rodgers et al., 2021). The current study added a biomarker component and enrolled a comparison group that did not replace furniture to help account for the overall declining serum PBDE levels in the population. We investigated the differences between the groups over an approximate 12-month period.

Participants in the furniture/foam replacement group were recruited in 2015–2016 through two different efforts as detailed in Rodgers et al. 2021. In short, the first effort recruited individuals who volunteered to replace either their older upholstered furniture or the foam in their furniture in their main living area. These were recruited through a Green Science Policy Institute (GSPI) listserv and through networks of GSPI and the University of California, Davis (UC Davis) researchers. The second effort recruited individuals to take part in a furniture replacement program, for which the replacement furniture was purchased for them. These participants were recruited from one of two affordable housing complexes through flyers and a community meeting. All enrolled homes were in Northern California. To be eligible for the study, participants' homes were required to have at least one couch or loveseat in the main living area with a manufacturing date between 1975 and 2014, with one exception for a home with an antique couch with replacement foam from the required years. All large foam-filled furniture was noted in an initial home assessment and the participant either replaced the furniture, replaced the foam, or removed the furniture within one year to qualify for the study. FR-free status was verified by checking TB117–2013 tags. After enrollment into the dust component of the furniture/foam replacement study, participants interested in being part of the biomarker investigation were consented by CDPH staff.

Participants for a comparison group were recruited in 2016 by convenience sampling through Biomonitoring California staff networks as part of the Intraprogram Pilot Study, an ongoing laboratory method development protocol. For inclusion in the comparison group, participants had to affirm that they had not removed or replaced major pieces of foam furniture from the main rooms of their homes and not changed addresses during the sampling time period.

Recruitment and data collection protocols were approved by the Committee for the Protection of Human Subjects of the California Health and Human Services Agency (biological sample collection component) or the Institutional Review Board for UC Davis (dust sample collection component). Participants provided informed consent before collection of any data.

2.3 Sample collection

Dust collection methods were documented in Rodgers et al. 2021. Briefly, trained staff vacuumed the main living spaces in participants' homes using a standardized protocol which excluded surfaces of upholstered furniture. Dust was collected with a crevice tool attachment with cellulose extraction thimbles, which were stored at -20°C until analysis. Pre-replacement dust samples were collected at the time of enrollment into the study. The participants were initially asked to replace their furniture or foam within 12 months from enrollment as procuring new furniture can take considerable time. The second dust sample was collected approximately one year after the couch was replaced.

For the foam/furniture replacement group, initial urine and blood sample collection was conducted prior to, or in two cases, soon after furniture replacement (urine median 93 days prior (range 282 days prior to 15 days after); blood median 71 days prior (range 288 days prior to 43 days after)) and the second samples were collected approximately one year after furniture replacement (Figure 1). Biological sample collections were generally timed to occur a few days after the corresponding dust collection. A phlebotomist visited homes to collect approximately 20 mL of blood in tiger top serum separator tubes. Urine was collected by participants themselves (spot sample) and frozen in their homes as soon as possible, usually immediately after collection. Participants were asked to collect a field blank with every urine sample collection. Biological samples were transported back to the California Department of Public Health for processing where serum was centrifuged for 15 min at 2000 rpm. Both media were then frozen at -20°C . Samples were then transported on ice to the California Department of Toxic Substances Control (CA DTSC), Environmental Chemistry Laboratory for analyses.

For the comparison group, participants provided urine and blood at timepoints approximately one year apart concurrent with the furniture/foam replacement study's period. Participants attended sample collection events at which a phlebotomist drew blood samples and participants self-collected and submitted urine samples. Collection and lab processing protocols were identical to the foam/furniture replacement group, except that all activities took place at the Environmental Chemistry Laboratory.

2.4 Questionnaire

Participants in the biomarker component of the foam/furniture replacement group were asked questions related to behaviors that might modify their FR exposures. Questions with sufficient data and diversity of answers, including questions regarding mattress components, meat consumption, hand washing frequency, and hours of computer use, were used for analyses. Questionnaires were conducted on the same day as urine collection for the foam/furniture replacement group participants.

2.5 Laboratory analyses

Full laboratory analyses methods and study results for dust samples are documented in Rodgers et al. 2021. Analyses were performed using gas chromatography-mass spectrometry Q/TOF running in electron ionization mode. Analytes pertinent to the current study included three PBDEs (BDE-47, BDE-99, BDE-100) and three OPFRs (TCEP, TDCIPP, and triphenyl phosphate (TPhP)).

Laboratory PBDE analyses of serum samples were performed by the Environmental Chemistry Laboratory at CA DTSC (Hurley et al., 2019; Park et al., 2015). Analytes included nineteen PBDE congeners (BDE-17, -28, -47, -66, -85, -99, -100, -153, -154, -183, -196, -197, -201, -202, -203, -206, -207, -208, and -209) and lipid measurements. Serum samples were first thawed and aliquoted for PBDE analysis. Samples were then spiked with $^{13}\text{C}_{12}$ labeled surrogate standards, extracted using Oasis HLB SPE cartridges (Waters Corp, 540 mg, 60 μm particle size) and the Biotage[®] RapidTrace system, and eluted in 1:1 dichloromethane:hexane. After concentration in a TurboVap (Biotage, Charlotte, NC), further cleaning was performed with 33% sulfuric acid silica using the Biotage[®] RapidTrace system and the matrix changed to isooctane. Final eluates were concentrated again and spiked with recovery standards. Standard reference material (SRM 1958, National Institute of Standards and Technology, Gaithersburg, MD), pre-spiked with known amounts of target analytes and duplicate blank samples of bovine calf serum were used as QA/QC samples. Samples were then analyzed using a ThermoFisher Scientific high-resolution double-focusing magnetic sector mass spectrometer (DFS MS) with gas chromatography equipped with a 15 m DB-5MS GC column (Agilent). The laboratory's proficiency with this method is evaluated up to 3 times per year by the Arctic Monitoring & Assessment Program (AMAP). A small volume of serum (0.3 mL) from each sample was sent to Boston Children's Hospital for measurement of total cholesterol and triglycerides by enzymatic methods (Allain et al., 1974; Stinson et al., 1977). Cholesterol and triglycerides were used to calculate the lipid content based on Phillips' formula (Phillips et al., 1989). PBDE levels were then standardized for lipids to produce values with units of ng/g lipid.

Laboratory OPFR analyses of urine were performed at the Environmental Chemistry Laboratory at the CA DTSC (Petropoulou et al., 2016). Analytes included three OPFR metabolites: bis(2-chloroethyl) phosphate (BCEP), bis(1,3-dichloroisopropyl) phosphate (BDCIPP), and diphenyl phosphate (DPhP), which are metabolites of three OPFRs that had been measured in the dust: tris(2-chloroethyl) phosphate (TCEP), tris(1,3-dichloroisopropyl) phosphate (TDCIPP), triphenyl phosphate (TPhP), respectively. Urine samples were thawed and aliquoted for OPFR and specific gravity measurements, the latter of which were

measured using a digital urine specific gravity refractometer (ATAGO UG-α, Tokyo, Japan). Samples were then spiked with deuterated surrogate standards and extracted using an offline solid phase extraction (SPE) procedure with a weak anionic sorbent cartridge (STRATA-X-AW, Phenomenex). After concentration in a TurboVap LV system (Biotage, Charlotte, NC) samples were reconstituted in methanol. Samples were then analyzed on an Ultra-Fast liquid chromatography system (Prominence UFLC/UFLCXR, Shimadzu Corporation, Columbia, MD, USA) with a Luna C18 column (Phenomenex, Torrance, CA, USA) coupled to a MS/MS system (Sciex QTrap 5500 MS, Sciex, Redwood City, CA) operated in negative electrospray ionization mode. QA/QC samples were created from pooled urine samples either as blanks or pre-spiked with certified analytical standards (Toronto Research Chemicals Inc. (Toronto, Ontario, Canada), Wellington Laboratories (Guelph, Ontario, Canada), and Sigma-Aldrich Laboratories (St. Louis, MO)). Field blanks received water addition and were extracted and concentrated as described above. Analysis with LC-MS-MS confirmed field blank concentrations to be below the calibration curve range. OPFR metabolite values were adjusted using specific gravity to account for urine dilution according to the following equation (Mahalingaiah et al., 2008):

$$Concentration_{SG - adj} = Concentration_{measured} \times \frac{1.017 - 1}{SG - 1}$$

The median specific gravity (SG) for adults (18+; weighted data) of 1.017 in the National Health and Nutrition Examination Survey 2007–2008 cycle (Centers for Disease Control and Prevention, 2022) was selected as the reference value to enable comparisons with other studies.

2.5 Data analyses

Of the 19 PBDEs and the three OPFR metabolites analyzed in biological samples, we prioritized chemicals for data analysis if they were above the limit of detection (LOD) in at least 65% of baseline samples. Four PBDEs (BDE-47, BDE-99, BDE-100, and BDE-153) and three OPFR metabolites (BCEP, BDCIPP, and DPhP) met this criterion. Values below the laboratory LODs were substituted with LOD/√2 before lipid adjustment for the PBDEs and SG adjustment for OPFRs. For BDEs 99, 100, and 153 which had low detection frequency at follow-up, these values are approximate and primarily used to allow slope calculation for comparison with the intervention group. We recalculated slopes using 0 and LOD instead of LOD/√2 to assess robustness of findings. We summarized FR concentrations by study visit (baseline and 12-month samples) and study group (furniture/foam replacement versus comparison) using descriptive statistics. T-tests or Wilcoxon rank sum tests (where logged data were not sufficiently normal) were used to examine differences between study groups' FR concentrations. Spearman correlations were performed within the FRs classes. To evaluate whether the intervention resulted in decreases in concentrations beyond what would be expected with biological elimination, we calculated change in log concentrations over time of PBDEs in serum. This allowed us to account for the long half-lives of these chemicals, their expected decline by first-order kinetics (Sjödin et al., 2020), and the varied periods of time between first and second samples for participants (0.8 to 1.8 years). Since several households took longer than expected to replace their foam or furniture,

this calculation led to a more conservative estimate of the change due to ongoing exposure during the pre-replacement period. Log changes were calculated as follows and converted to half-times, i.e., times it would take to decline by half as defined below, for readability. (Values presented in the tables are rounded after all calculations.) Of note, we contrast our use of half-times here with half-lives, since other sources of ongoing exposure are taken into account in the former but would not be for the latter.

$$\text{Log change in concentration over time} : = \frac{\ln[PBDE_{t2}] - \ln[PBDE_{t1}]}{\text{time}(\text{years})}$$

$$\text{Half - times} = -\ln(2) / \left(\frac{\ln[PBDE_{t2}] - \ln[PBDE_{t1}]}{\text{time}(\text{years})} \right)$$

For the OPFR metabolites, due to their short half-lives relative to the time period involved, we calculated absolute change between the timepoints. For all FRs, comparisons of concentration change between groups were performed using Wilcoxon rank sum.

To compare changes in dust concentrations to changes in biomarker levels, Spearman correlations were tested on (1) concentrations at baseline of FRs and/or their metabolites between media, (2) concentrations at 12 months of FRs and/or their metabolites between media, (3) change in log PBDE dust concentrations vs. the calculated change in log concentrations over time of PBDE biomarkers and (4) absolute change in OPFR dust concentrations vs. absolute change in OPFR metabolite levels.

Due to small numbers and similarity in percentages in sex and race/ethnicity between the furniture/foam replacement group and comparison group, adjustment by these variables was not performed. Spearman correlations and Wilcoxon rank sum tests were used to examine associations of behavioral characteristics, age, and sex with initial biomarker concentrations and change in biomarker concentrations over time in the furniture/foam replacement group.

We performed two sensitivity analyses. We tested for undue influence of participants with much higher initial PBDE values by dropping the observations and retesting. Also, due to clustering of subjects within households in the furniture/foam replacement group, we performed a clustered Wilcoxon rank sum test (R version 4.3.0, package clusrank). All other analyses were conducted in SAS version 9.4 (SAS Institute Inc, Cary, NC).

2.6. Study results reporting

Biomonitoring California is required to make individual results available to study participants, and all participants consented to receive their results for this study. Participants were mailed paper reports detailing biomarker concentrations at every time point for which they donated blood or urine samples. Results return materials also included how their results compared to other study participants, what was known about health effects of the biomarkers, strategies for reducing exposures, and contact information to speak with study staff. Dust results were also returned to participants as described in Rodgers et al. 2021.

3. Results

3.1 Study population

From the 42 households that had consented into the furniture/foam replacement dust study, 25 participants enrolled and completed the biomarker study. Two of those participants did not complete the dust study but did provide samples for the biomarker study. There were two participants per household for 8 households (16 participants) and one per household for an additional 9 participants. Baseline biomarker measurements for the furniture/foam replacement group took place over one year (September 2015 to September 2016, median in October 2015). The comparison group enrolled 28 participants (one household had two participants). Baseline measurements for the comparison group's samples were collected at arranged events taking place during August and September 2016.

All participants provided blood samples and all but one, in the comparison group, contributed a urine sample. The time between baseline and 12-month samples was very consistent for participants in the comparison group, with a median value of 1.08 years. As it took some participants a while to replace their furniture, there was more variability in the time between baseline and 12-month samples in the furniture/foam replacement group, increasing the median value to 1.27 years (Table 1).

Breakdowns by sex and race/ethnicity were similar between groups (68% female in both; 68–71% White; 16–18% Asian) (Table 1). Age ranges were also similar between the groups (34–64 years vs. 31–71 years), though the median age was slightly older in the comparison group (51.5 years vs. 45 years).

3.2 Initial flame retardant biomarker concentrations

Baseline detection frequencies for four PBDEs (BDE-47, BDE-99, BDE-100, and BDE-153) were 75–91% while other PBDEs were detected at frequencies $\leq 30\%$ (Table 2, Supplementary Table 1). Initial concentrations were significantly higher in the foam/furniture replacement group for these four PBDEs, with the highest concentrations observed for BDE-47 (BDE-47 geometric means (GMs) of 25.09 and 9.39 ng/g lipid in the foam/furniture replacement group and comparison group, respectively; BDE-99 GMs of 7.18 and 3.31 ng/g lipid; BDE-100 GMs of 4.48 and 1.73 ng/g lipid; and BDE-153 GMs of 9.89 and 4.02 ng/g lipid). Three participants in the furniture/foam replacement study had initial levels 3–13 times higher than the GM. Concentrations of three PBDEs were highly correlated (BDE-47, BDE-99, and BDE-100: Spearman $\rho = 0.85$ – 0.96) and less correlated with BDE-153 ($\rho = 0.42$ – 0.63) (Supplementary Table 5).

OPFR metabolites were detected at high frequency (96–98%) in baseline samples (Table 3). Geometric mean concentrations ranged from 1.22–2.44 $\mu\text{g/L}$. Initial concentrations of BCEP and DPhP were significantly higher in the foam/furniture replacement group. These metabolites were only weakly, and not significantly, correlated with each other ($\rho = 0.11$ – 0.30) (Supplementary Table 5).

3.3 Impact of furniture replacement on biomarker concentrations

PBDE concentrations declined in a majority of participants' serum: in 88–92% of the foam/furniture replacement group, depending on the congener, and in 72–79% of the comparison group (Figure 2, Supplementary Figures 1–3). To account for the long half-lives of PBDEs and the apparent first order elimination from the body (Sjödin et al., 2020), the differences in PBDE log change over time were tested and indicated statistically significantly greater changes within the foam/furniture replacement group than the comparison group (Table 4). Converting these to half-times (i.e., times it would take to decline by half (given ongoing other exposures to PBDEs)), we observed that concentrations of BDE-47, BDE-99, and BDE-100 were estimated to be 1.9–3.9 times longer in the comparison group than the furniture/foam replacement group (1.4 years (median) for BDE-47 and BDE-99 in the furniture/foam replacement group versus 2.7 and 2.6 years, respectively, in the comparison group and 1.4 years vs. 5.2 years for BDE-100). Longer times to decline by half for BDE-153 were additionally reflective of the longer half-life of this chemical estimated in the literature.

OPFR metabolite changes were much more variable; 50–80% of the foam/furniture replacement group had values that decreased, depending on the metabolite, while 27–67% of the comparison group had values that decreased (Figures S4–S6). Due to their short half-lives, changes in OPFRs were examined as simple differences in concentrations within participants between the two timepoints. For BCEP, the median change in concentration showed an increase in the comparison group compared to no change in the furniture/foam replacement group ($p < 0.05$ for comparison of median change between groups) (Table 4). Nonsignificant greater decreases were seen for BDCIPP in the furniture/foam replacement group, while greater decreases were seen for DPhP in the comparison group.

We performed two sensitivity tests. First, we tested whether the assumption of first order kinetics allowed the initial higher PBDE values in the foam/furniture replacement group to be overly influential. Retesting without the three people with much higher initial values in this group resulted in similar median log change over time and p-values (Supplemental Table 3). Additionally, after clustering on household to account for the 8 two-participant households, significant differences between the two groups continued to be observed for BDE-47 and –100 but not BDE-99 (Supplemental Table 4).

We investigated if various demographic and behavioral factors impacted our furniture/foam replacement group FR biomarker levels. The only significant associations observed for behavioral factors were for sleeping on a foam mattress, which was associated with higher initial BDE-47 and BDE-100 concentrations (Supplemental Table 2). In addition, age was marginally negatively correlated with baseline BDCIPP concentrations ($\rho = -0.36$, $p = 0.08$). No differences across demographic or behavioral factors were observed with magnitude of change over the study time period.

3.4 Comparisons of changes in dust and biomarker concentrations

Both dust and biomarker measurements for the two timepoints were available for most analytes for 23 furniture/foam replacement participants (TDCIPP/BDCIPP data

was available for 22 participants). Initial concentrations of dust and biomarker PBDE concentrations were moderately positively correlated with correlation coefficients of 0.58 to 0.60 ($p < 0.01$) (Table 5). Initial concentrations of OPFR metabolites in urine and their parent compounds in dust were not correlated ($\rho = 0.05$ – 0.27 , $p > 0.05$). At the 12-month timepoint, PBDE concentrations were less positively correlated than they were at the initial timepoint. Two OPFR metabolites remained uncorrelated, while TCEP/BCEP measurements were moderately positively correlated ($\rho = 0.58$, $p < 0.01$). Correlations between the magnitudes of change were mostly positive between biomarkers and dust, but no significant findings were observed.

4. Discussion

In this study examining whether biomarker flame retardant levels will decrease with removal and replacement of pre TB117–2013 standard furniture or foam, greater decreases in PBDEs were seen in the furniture replacement group. As expected, PBDE concentrations decreased in the comparison group as well, though to a lesser degree. Both groups also experienced a decrease in two of three OPFR biomarkers, though the decreases were not significantly different between the groups. However, one OPFR increased in the comparison participants but showed no change in the replacement group. Additionally, while biomarker concentrations of PBDEs were initially correlated with dust concentrations ($\rho = 0.58$ – 0.60 , p -values < 0.001), correlations were not observed at the 12-month time point or between change in dust concentrations and the degree of biomarker change over time.

The PBDE congeners with the greatest difference in change between the two groups corresponded to the BDEs most prominent in the technical mix whose primary usage has been as an additive to polyurethane foams, penta-BDE (DE-17) (Birnbaum & Staskal, 2004). This mix generally has consisted of 24–38% tetrabromodiphenyls (which includes predominantly BDE-47) and 50–60% penta-BDE (dominated by BDE-99 and BDE-100). Only 4–8% of the mixture is estimated to consist of hexabromodiphenyls (including BDE-153), which is reflected in the lower serum concentrations observed in our study. Other commercial mixtures of PBDEs also contain hexabromodiphenyls but have been populated mostly by higher brominated congeners (≥ 7 bromines) which were observed in less than 30% of our serum samples. These mixtures have historically been used for other purposes, such as polymer additives for use in plastic housings and smaller components, such as electronics and office equipment (Birnbaum & Staskal, 2004). Since BDE-153 is a less prominent component in the mixtures used in foam versus other formulations (14% in commercial OctaBDE products) (Agency for Toxic Substances and Disease Registry, 2004) the BDE-153 serum levels may have been less affected by the furniture change versus other sources, including through diet which particularly influences this congener (Li et al 2020). Additionally, slower rates of metabolism and elimination observed for this more highly brominated congener in human and animal studies likely influenced study levels and our ability to detect a significant difference in the change over time.

The declining levels of PBDEs in both groups in our study are likely reflective of the various manufacturing and regulatory changes, including the 2014 change in California flammability standards which was subsequently adopted for the entire United States (Consumer Product

Safety Commission, 2021). These efforts have impacted PBDE use in the country, and overall population trends show declining levels of PBDEs in serum (Sjödén et al., 2019). The follow-up period in this study was long enough to capture these secular trends in exposure reduction of PBDEs. The decline in correlations between the dust and serum measurements between the pre-replacement measurements and at the 12-month time period also point to the lack of a continuing major source of PBDEs in the home (namely the furniture). While serum levels slowly declined after removal of a previous major source, 12-month dust levels may have been influenced by other sources, such as other plastics and electronics in the home. Diet is also a known source for PBDEs, which could have affected participants' levels, though prior studies in the U.S. suggest biomonitoring concentrations are much more highly associated with dust than other sources for many PBDEs (Pohl et al., 2017).

Our measurements of OPFR biomarkers appeared less related to the intervention, likely due to their short half-lives and high intraindividual variability (Meeker et al., 2013; Wang et al., 2021). Additionally, other prominent sources may also have played a role in OPFR levels, such as food, food packaging, or car furnishings (Blum et al., 2010; Gbadamosi et al., 2021; Hoehn et al., 2024). Additionally, OPFRs have been used as plasticizers, hydraulic fluids, solvents, adhesives, and coatings for electronic devices (Agency for Toxic Substances and Disease Registry, 2012). In the more extended tracking of dust concentrations in these homes, while the largest reduction of TCIPP and TDCIPP appeared at six months after furniture replacement, reductions were attenuated at 12 and 18 months, showing a potential re-equilibration in the homes from other sources (Rodgers et al., 2021).

Among the few studies examining effects of interventions on flame retardant exposure, all have found decreases after interventions. A study of 32 people in 2016 found decreases in most measured OPFRs after one week of instructed handwashing and/or house cleaning (Gibson et al., 2018). Two studies that replaced FR-containing foam with non FR-containing foam in gymnastics practice spaces found decreases in all measured OPFRs and most PBDEs measured on handwipes (Ceballos et al., 2018; Dembsey et al., 2019). Additionally, two studies that collected dust samples from university campus areas that self-described as following TB 117–2013 guidance compared with campus areas that did not found lower dust concentrations of all measured PBDEs and most measured OPFRs (Rodgers et al., 2020; Sjödén et al., 2020; Young et al., 2021). In the current study, the magnitudes of decrease in PBDE concentrations were similar but somewhat lower than in a study of US residents followed after moving to Australia, where PBDE serum concentrations have been tracked at one tenth the levels of the American populace (Sjödén et al., 2020). The estimated half-lives of that study were 1.19 years for BDE-47, 1.03 years for BDE-99, 2.16 years for BDE-100, and 4.12 years for BDE-153. In contrast, magnitudes of decrease in this study were greater than in an earlier non-intervention observational study that followed US participants over a year, which could be an indicator of a lower amount of ongoing PBDE exposure sources in our study (Makey et al., 2014). Because PBDE and OPFR use in upholstered furniture appears to be decreasing (Cooper et al., 2016; Gill et al., 2024), interventions with TB 117–2013 compliant furniture evidently can play a significant role in reducing exposure. Replacement of older foam-containing furniture should therefore be encouraged.

Our study had several limitations. Firstly, we did not collect biological samples at the exact time of the foam/furniture replacement due to participants' need for flexibility in completing the intervention. The variable time between their baseline sample collection and the intervention was therefore factored into the change over time calculations, likely causing an underestimate of the true change relative to the comparison group. If serum levels had yet to reach steady state after this source elimination, our estimates from the 12-month measurements would likely also be an underestimate. Secondly, while the follow-up time was long enough to capture changes in PBDE concentrations with a single specimen per visit, spot OPFR samples have been shown to have poor reproducibility, such as in a study of repeated measurements in adults where intraclass correlation coefficients (ICCs) for DPhP and BDCIPP were below 0.35 (Meeker et al., 2013; Wang et al., 2021). Multiple measurements or 24-hour collections may have allowed a change over time to be better captured. Thus, our biospecimen measurements of OPFRs should be interpreted as indicative only of a given point in time. However, high variability may be the norm. For example, in the Wang et al. 2021 study, even with 24-hour collections the ICCs for DPhP only reached 0.22 (while BDCIPP reached 0.65) over the 7-consecutive day study period. (ICCs over 0.75 are considered to have excellent reproducibility, and fair to good if between 0.4 and 0.75) (Rosner, 2016). Additionally, because detection frequencies were low for BDEs 99, 100, and 153 at follow-up among the comparison group, these slope estimates are approximate and should be interpreted cautiously. Substituting values <LOD with 0 and the LOD instead of $\text{LOD}/\sqrt{2}$ produced similar results and did not change the conclusion that PBDEs declined more rapidly in the intervention group. This study also did not capture differences that may have existed between the groups on other potential exposure sources (such as diet or exposures to electronics or other plastic components) and was limited by the low sample size, which has an impact on the statistical power to detect associations. Lastly, while the current study determined that removal of some FR sources from the home had beneficial effects, the study results would underestimate the impacts that could be expected from a full removal of all FRs from the home.

5. Conclusions

This is the first study to evaluate the impact of in-home furniture replacement on FR biomarker levels. Replacing foam-containing furniture purchased before the implementation of TB-117–2013 with furniture purchased after and without added flame retardants was an effective way to reduce serum PBDE concentrations. While concentrations also decreased over time without the intervention, likely due to overall reduced population exposure, removal of old foam/furniture from the home likely accelerated the rate of the reduction. OPFR urine concentrations did not demonstrate a relationship with the intervention. Given the well-established associations with adverse health outcomes, efforts should be made to reduce exposures to PBDEs and other organohalogen and organophosphate FRs, while considering the potential health effects of substitutes. Updating flammability standards can be an effective way to reduce these harmful exposures at a regulatory level.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Updated California flammability standards allow compliance without added chemicals
- Changing furniture was associated with faster times to reduce PBDEs by half
- Changing furniture was associated with less consistent changes for OPFRs
- Updating flammability standards may aid in reducing flame retardant body burdens

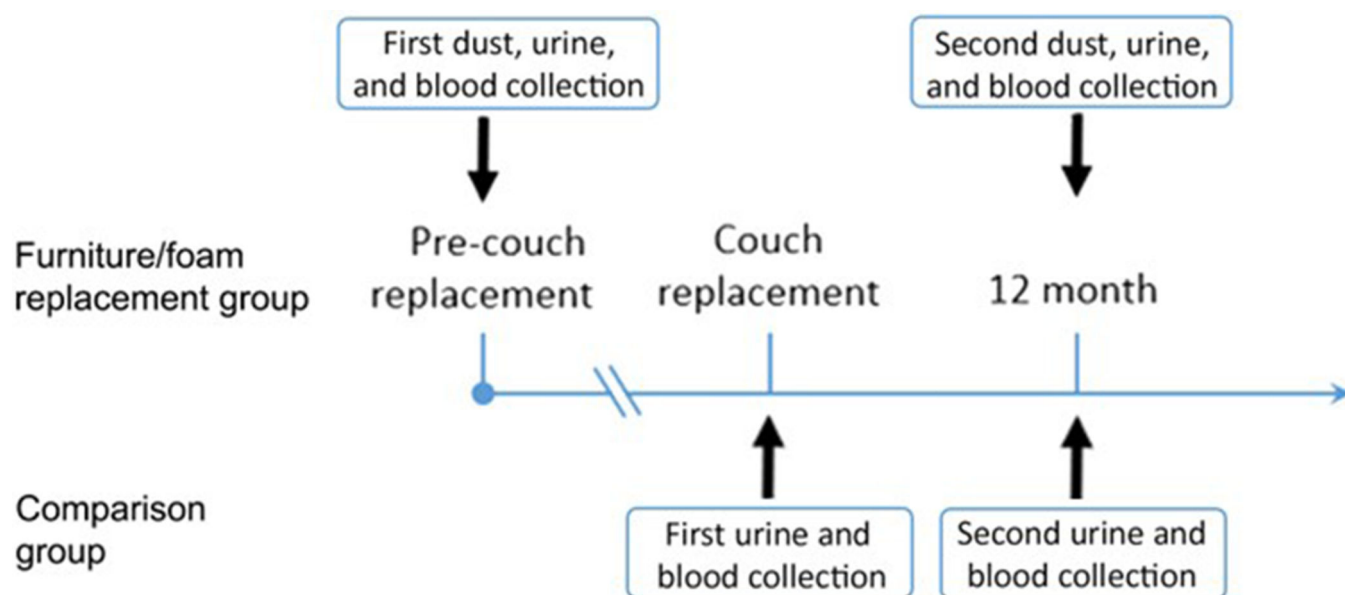
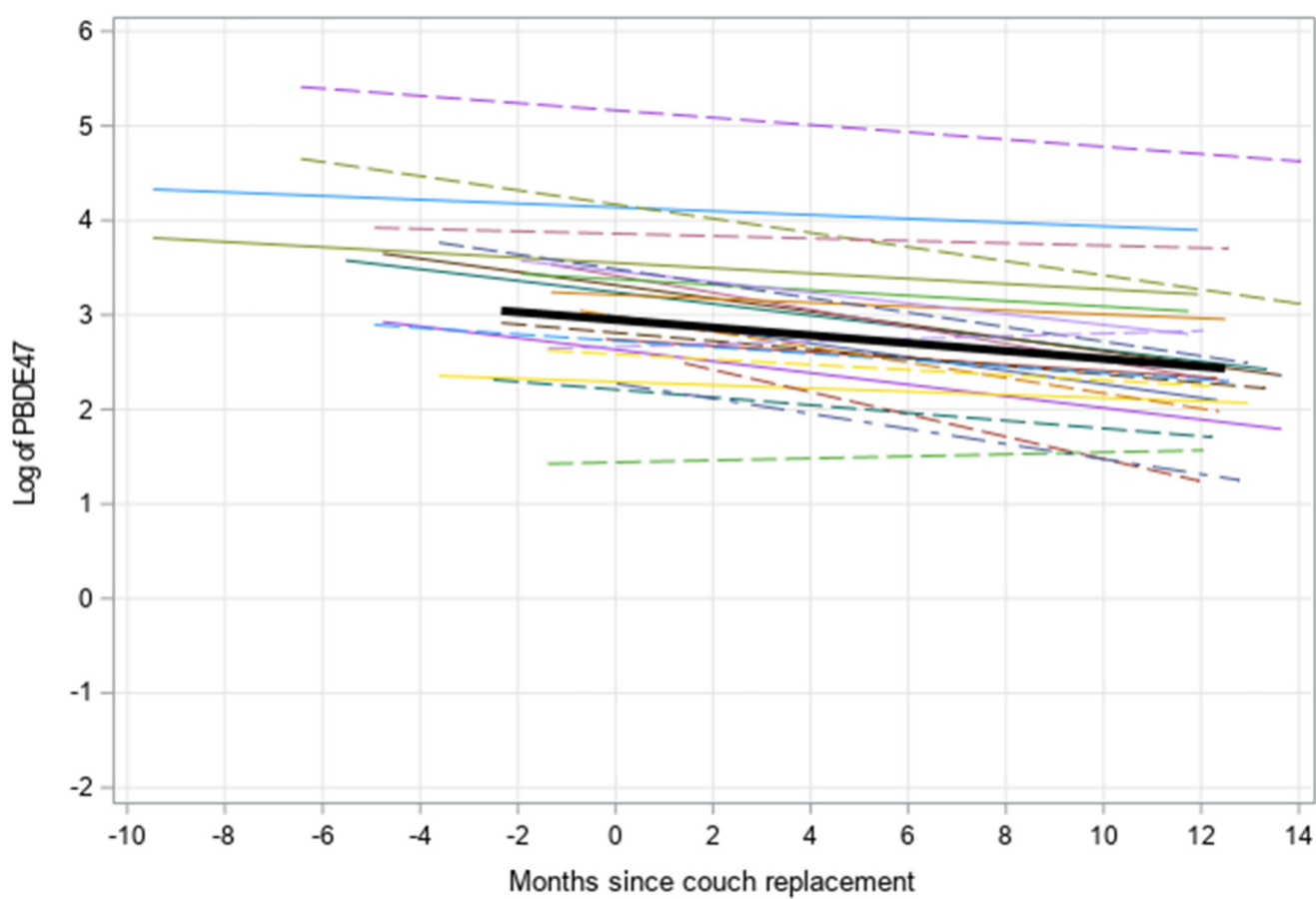


Figure 1.
Timeline schematic



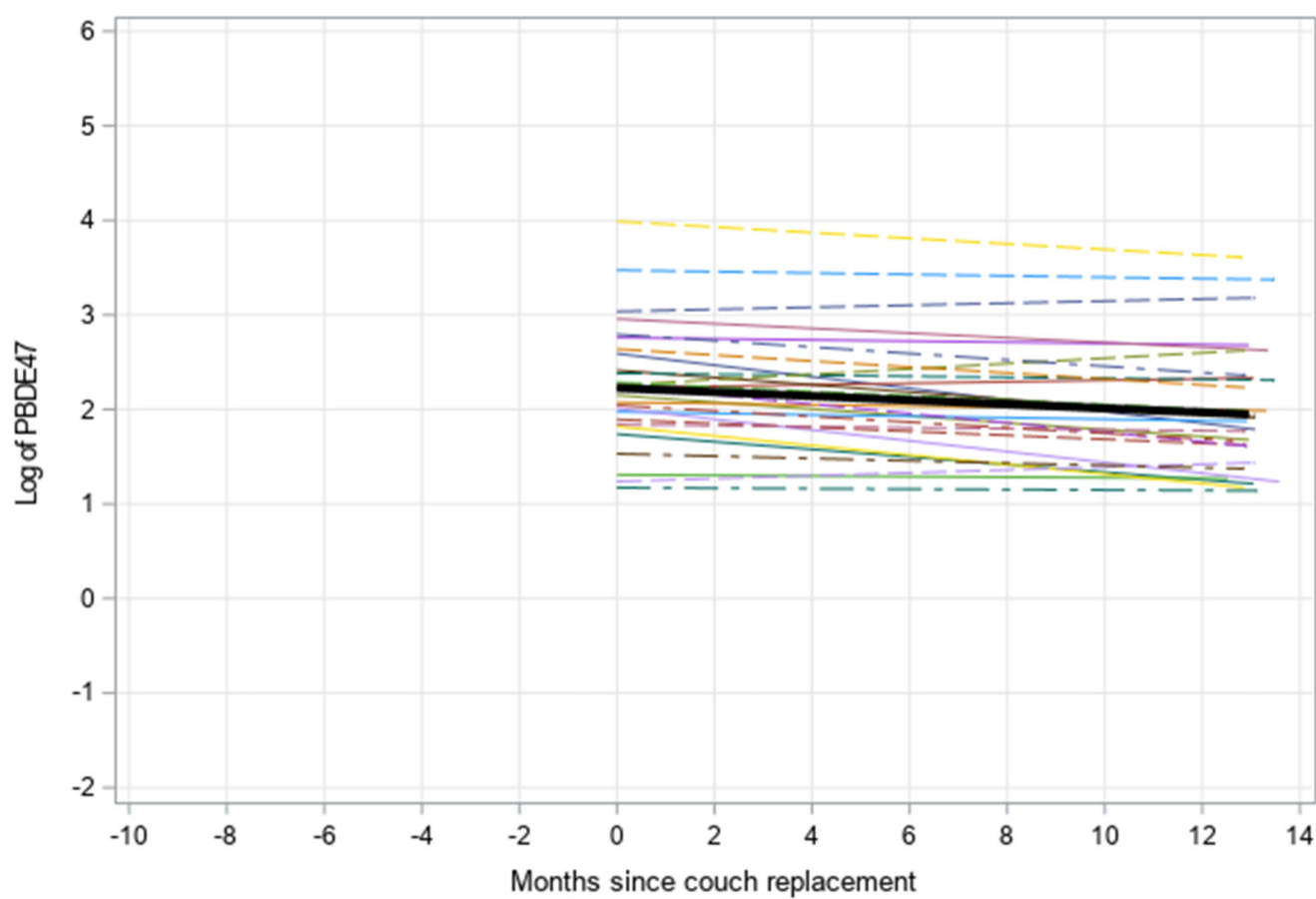


Figure 2.
Individual values of BDE-47 (ng/g lipids) over study period by group. Median change indicated with bolded black line.
A. Furniture/foam replacement group
B. Comparison group

Table 1.
Demographic and specimen collection characteristics of the comparison group and furniture/foam replacement group participants

	<u>Comparison group (N=28)</u>	<u>Furniture/foam replacement group (N=25)</u>
	<u>N (%)</u>	<u>N (%)</u>
Sex		
Female	19 (68%)	17 (68%)
Male	9 (32%)	8 (32%)
Race/ethnicity		
White	20 (71%)	17 (68%)
Asian	5 (18%)	4 (16%)
Hispanic	1 (3%)	2 (8%)
Black	0 (0%)	1 (4%)
Other/Multi-racial	2 (7%)	1 (4%)
	<u>Median (Range)</u>	<u>Median (Range)</u>
Age (years)	51.5 (31–71)	45 (34–64)
Baseline sample date *	8/26/2016	10/21/2015
	(8/2016 – 9/2016)	(9/2015 – 9/2016)
12 month sample date	9/27/2017	4/28/2017
	(9/2017 – 10/2017)	(10/2016 – 10/2017)
Comparison Interval		
(years) *	1.08	1.27
	(1.04 – 1.13)	(0.95 – 1.78)

* Dates and intervals correspond to blood sample collection dates. For 16 participants in the furniture/foam replacement group, baseline urine samples were collected an average of 14.8 days earlier (range: 42 days earlier to 6 days later) than the baseline blood samples. For all participants, blood and urine were collected on the same day for the 12-month sample.

Table 2.

PBDE concentrations (ng/g lipids) at baseline and 12 months for comparison group participants (N=28) and furniture/foam replacement group (N=25)

	Baseline												T test p-value
	Comparison group						Furniture/foam replacement group						
	Detection frequency (%)	GM	95% CI	25th pctl	Median	75th pctl	Detection frequency (%)	GM	95% CI	25th pctl	Median	75th pctl	
BDE-47	86	9.39	(7.27, 12.13)	6.48	9.29	13.68	96	25.09	(17.67, 35.62)	14.10	21.03	38.41	<0.01
BDE-99	64	3.31	(2.70, 4.05)	<LOD	3.18	4.00	88	7.18	(5.27, 9.77)	4.78	6.13	11.46	<0.01
BDE-100	64	1.73	(1.31, 2.28)	<LOD	1.45	3.16	96	4.48	(3.15, 6.39)	2.39	4.35	6.51	<0.01
BDE-153	68	4.02	(3.06, 5.27)	<LOD	4.09	6.35	92	9.89	(6.74, 14.50)	6.27	7.64	13.93	<0.01
12 month													
	Comparison group						Furniture/foam replacement group						Wilcoxon p-value
	Detection frequency (%)	GM	95% CI	25th pctl	Median	75th pctl	Detection frequency (%)	GM	95% CI	25th pctl	Median	75th pctl	
BDE-47	75	7.33	(5.61, 9.59)	4.62	6.65	10.45	88	12.22	(8.77, 17.04)	7.93	10.30	19.20	0.02
BDE-99	25	NC	NC	<LOD	<LOD	2.59	64	3.59	(2.86, 4.50)	2.27	3.47	4.89	<0.01
BDE-100	46	NC	NC	<LOD	<LOD	1.93	76	2.54	(1.80, 3.60)	1.24	2.26	3.55	0.01
BDE-153	57	NC	NC	2.02	<LOD	5.37	88	7.86	(5.56, 11.13)	5.60	6.70	11.30	<0.01

NC = GMs not calculated for detection frequencies under 60%

T-tests are of logged FR concentrations

Table 3.

OPFR concentrations (µg/L) at baseline and 12 months for comparison group participants (N=28) and furniture/foam replacement group (N=25)

	Baseline														T test p-value		
	Comparison group							Furniture/foam replacement group									
	Detection frequency (%)		N	GM	95% CI	25th pctl	Median	75th pctl	Detection frequency (%)		N	GM	95% CI	25th pctl		Median	75th pctl
	12 month																
BCEP	92.59	27	1.22	(0.87, 1.71)	0.53	1.29	2.62	100	24	2.00	(1.53, 2.62)	1.36	1.99	3.03	0.02		
BDCIPP	100	27	1.96	(1.48, 2.60)	1.20	2.05	3.74	100	25	1.95	(1.48, 2.57)	1.24	1.86	3.57	0.97		
DPhP	96.30	27	1.41	(1.08, 1.84)	0.87	1.27	2.23	100	25	2.44	(1.86, 3.22)	1.43	1.93	3.24	<0.01		
	12 month														T test p-value		
	Comparison group							Furniture/foam replacement group									
	Detection frequency (%)		N	GM	95% CI	25th pctl	Median	75th pctl	Detection frequency (%)		N	GM	95% CI	25th pctl		Median	75th pctl
BCEP	100	26	2.29	(1.75, 3.01)	1.40	2.28	3.64	100	25	1.74	(1.27, 2.37)	1.10	1.51	2.79	0.17		
BDCIPP	100	27	1.60	(1.18, 2.17)	0.97	1.68	2.47	100	25	0.92	(0.68, 1.26)	0.61	0.91	1.40	0.01		
DPhP	88.89	27	0.98	(0.77, 1.26)	0.61	0.86	1.50	100	25	2.39	(1.72, 3.33)	1.45	1.79	2.99	<0.01		

Specific gravity adjusted (µg/L), using reference of 1.017

T-tests are of logged FR concentrations

Table 4.

Magnitudes of change over time depicted as log change over time and half-times* for PBDEs and absolute change in concentrations for OPFRs between baseline and 12 months for comparison group participants (N=28) and furniture/foam replacement group participants (N=25)

	Log change in concentrations over time (yr ⁻¹)							Median half-times (years)	
	Comparison group			Furniture/foam replacement group			Wilcoxon p-value	Comparison group	Furniture/foam replacement group
	25th pctl	Median	75th pctl	25th pctl	Median	75th pctl			
BDE 47	-0.42	-0.26	-0.06	-0.87	-0.49	-0.33	<0.01	2.7	1.4
BDE 99	-0.50	-0.26	0.04	-0.81	-0.50	-0.19	0.02	2.6	1.4
BDE100	-0.29	-0.13	0.04	-0.67	-0.51	-0.23	<0.01	5.2	1.4
BDE153	-0.36	-0.08	0.04	-0.26	-0.16	-0.11	0.28	9.1	4.3
	Absolute change in concentrations (µg/L)							Wilcoxon p-value	
	Comparison group			Furniture/foam replacement group					
	25th pctl	Median	75th pctl	25th pctl	Median	75th pctl			
BCEP	-0.14	0.96	1.93	-1.37	0.01	0.98	0.046		
BDCIPP	-1.34	-0.43	0.61	-1.82	-1.08	-0.32	0.11		
DPhP	-0.76	-0.26	0.17	-1.53	-0.06	0.73	0.90		

* Half-times defined here as $\ln(2)/((\ln[\text{PBDE}_{t2}] - \ln[\text{PBDE}_{t1}])/\text{time}(\text{years}))$ which take into account other sources of ongoing exposures.

Table 5. Correlations of biomarker and dust concentrations at baseline and 12 months and for change in concentrations for furniture/foam replacement group participants with dust data (N=22–23)

Dust compound	Biomarker	Initial concentrations		12 month concentrations		Change in concentrations*	
		Spearman coefficient	p-value	Spearman coefficient	p-value	Spearman coefficient	p-value
BDE 47	BDE 47	0.60	<0.01	0.40	0.06	0.17	0.43
BDE 99	BDE 99	0.60	<0.01	0.31	0.15	0.11	0.62
BDE100	BDE 100	0.58	<0.01	0.12	0.59	0.12	0.58
TCEP	BCEP	0.27	0.20	0.58	<0.01	0.12	0.59
TDCIPP	BDCIPP	0.05	0.80	-0.12	0.59	-0.14	0.53
TPhP	DPhP	0.20	0.33	0.11	0.63	0.05	0.81

* For PBDEs, change in concentration = the slope of change in natural logged biomarker concentrations from baseline to 12 months vs. change in natural logged dust concentrations from baseline to 12 months (indicated as rate constants in Table 4). For OPFRs, change in concentration = change in biomarker concentration from baseline to 12 months vs. change in dust concentrations from baseline to 12 months.