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Associations between brominated flame retardants in house dust and hormone levels in men

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Abstract

Brominated flame retardants (BFRs) are used in the manufacture of a variety of materials and consumer products in order to meet fire safety standards. BFRs may persist in the environment and have been detected in wildlife, humans and indoor dust and air. Some BFRs have demonstrated endocrine and reproductive effects in animals, but human studies are limited. In this exploratory study, we measured serum hormone levels and flame retardant concentrations [31 polybrominated diphenyl ether (PBDE) congeners and 6 alternate flame retardants] in house dust from men recruited through a US infertility clinic. PBDE congeners in dust were grouped by commercial mixtures (i.e. penta-, octaand deca-BDE). In multivariable linear regression models adjusted by age and body mass index (BMI), significant positive associations were found between house dust concentrations of pentaBDEs and serum levels of free T4, total T3, estradiol, and sex hormone binding globulin (SHBG), along with an inverse association with follicle stimulating hormone (FSH). There were also positive associations of octaBDE concentrations with serum free T4, thyroid stimulating hormone (TSH), luteinizing hormone (LH) and testosterone and an inverse association of decaBDE concentrations with testosterone. Hexabromocyclododecane (HBCD) was associated with decreased SHBG and increased free androgen index. Dust concentrations of bis-tribromophenoxyethane (BTBPE) and tetrabromo-diethylhexylphthalate (TBPH) were positively associated with total T3. These findings are consistent with our previous report of associations between PBDEs (BDE 47, 99 and 100) in house dust and hormone levels in men, and further suggest that exposure to contaminants in indoor dust may be leading to endocrine disruption in men.

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1. Introduction

Brominated flame retardants (BFRs) are a group of chemicals used in the production of consumer goods to meet fire safety standards. Polybrominated diphenyl ethers (PBDEs) have been one of the most widely used groups of BFRs. Commercial formulations of PBDEs consist of a mixture of congeners and are commonly referred to as penta-, octa- and deca-brominated diphenyl ethers (BDEs). The European Union banned the use of penta- and octa-BDEs in 2004 due to their persistence and bioaccumulation (WHO, 2007). These formulations were voluntarily phased out of production in the United States in 2004; the U.S. has no federal regulation on the use of PBDEs, but several states have issued their own restrictions (http://www.BSEF.com, accessed Nov. 3, 2011). DecaBDE is currently still in use, although there are plans to begin phasing out in December, 2012 (EPA, 2009)..

Despite use restrictions, the general population continues to be exposed to PBDEs due to their persistence and through the continued use of existing products containing PBDEs (e.g. furniture and TVs). Few studies on the human health effects of PBDEs exist, despite evidence of widespread exposure through contact or ingestion of house dust or from dietary sources. Additionally, there are alternate flame retardants entering the marketplace or increasing in use as others are phased out. As alternative flame retardants replace discontinued compounds and production volumes increase, concern over potential exposure to these alternates and possible health effects is rising (DiGangi et al., 2010; Shaw et al., 2010).

Hexabromocyclododecane (HBCD) is another large volume BFR used primarily in polystyrene foam insulation but also in certain textiles and electronics (EPA, 2008). 1,2-bis (2,4,6-tribromophenoxy)ethane (BTBPE) is a new BFR that is being used as a replacement for octa-BDE, while 2,3,4,5-ethylhexyltetrabromobenzoate (TBB) and 2,3,4,5-tetrabromo di (2-ethylhexyl) phthalate (TBPH) are two components of a mixture that is being marketed as a replacement formulation for pentaBDE (Chemtura, 2006, 2007). Because these BFRs are additive chemicals, they can leach into the environment in the same manner as PBDEs and have also been measured in house dust (Stapleton et al., 2008; Zhu et al., 2007). However, few or no studies exist on the toxicity and potential human health effects of these replacement compounds. The use of TBPH is a concern because it is a brominated analogue of di(ethylhexyl)phthalate (DEHP), which is listed under California's Proposition 65 as a chemical known to cause cancer and reproductive and developmental toxicity (OEHHA, 2008). Dechlorane Plus (DP) (bis(hexachlorocyclopentadieno)cyclooctane) is a large volume highly chlorinated flame retardant recently identified in lake sediments (Hoh et al., 2006), house dust (Zhu et al., 2007), and humans (Ren et al., 2009; Siddique et al., 2012) with growing concern for human exposure.

Animal studies have established that PBDEs are endocrine disruptors, altering reproductive and thyroid hormone homeostasis. A number of animal studies report reduced serum thyroxine (T4) levels after administration of pentaBDE mixtures (Fowles et al., 1994; Hallgren et al., 2001; Zhou et al., 2001, 2002; Stoker et al., 2004; Ellis-Hutchings et al., 2006). Displacement of thyroid hormones from the hormone receptor or the transport protein by PBDE metabolites, which was shown *in vitro* (Meerts et al., 2000), has been postulated as a possible mechanism by which thyroid hormone homeostasis is disrupted (reviewed by

Darnerud, 2008). Additionally,Butt et al. (2011) conducted an *in vitro* study and found that OH-PBDEs inhibited deiodinase enzymes that convert T4 to T3, and this may also affect thyroid hormone regulation.

Experimental studies have demonstrated that the endocrine disrupting properties of PBDEs are congener dependent. For example, several less brominated congeners, such as the tetra- and penta-brominated congeners, acted as estrogen receptor agonists, while some hexa- and hepta-brominated congeners had antiestrogenic effects *in vitro* (Meerts et al., 2001). Animal studies have shown that exposure to PBDEs resulted in reproductive effects such as delayed puberty (Stoker et al., 2004), reduced sperm counts (Kuriyama et al., 2005) and reduced reproductive success (Fernie et al., 2009; Henny et al., 2009), which may be a consequence of endocrine disruption.

HBCD has been much less studied in relation to altered endocrine function. Similar to PBDEs, exposure of rats to HBCD resulted in decreased T4 and increased TSH levels (Ema et al., 2008). To our knowledge, there are no studies to date on endocrine function in relation to exposure to the other non-PBDE flame retardants measured in the present study. Studies of potential human health effects of BFRs are limited and focus primarily on thyroid hormone disruption in relation to PBDE exposure. Contrary to most of the animal experiments, several human epidemiological studies reported increases in T4 and T3 levels associated with exposure to PBDEs (Bloom et al., 2008; Meeker et al., 2009; Turyk et al., 2008; Wang et al., 2010; Daillare et al., 2009; Gascon et al., 2011; Stapleton et al., 2011).

Although consumption of contaminated foods is an important exposure route for PBDEs (Fraser et al., 2009), PBDE exposure in North America is estimated to be mainly from the ingestion of indoor dust (Lorber, 2008; Jones-Otazo et al., 2009; Webster et al., 2005; Johnson-Restrepo et al., 2009). These estimates are supported by studies that link body burdens to indoor dust concentrations (Wu et al., 2007; Johnson et al., 2010; Stapleton et al., 2012). Dust concentrations of HBCD (Roosens et al., 2009) and DP (Zheng et al., 2010) have also been correlated to body burdens.

We recently demonstrated strong correlations between serum and house dust concentrations of PBDEs (BDE 47, 99, and 100) for 12 men and their female partners (Johnson et al., 2010). Serum hormone data were not available for those 12 men and they were not included in the present analysis. We also recently reported that dust concentrations of BDE 47, 99, and 100 were positively associated with serum levels of free T4 in 24 men. These congeners were also associated with alterations in levels of luteinizing hormone (LH), follicle stimulating hormone (FSH), Inhibin B, sex hormone binding globulin (SHBG) and free androgen index (FAI) (Meeker et al., 2009). The present study expands our previous work to an additional 38 men (n=62 for PentaBDE congeners) and analytes, including 31 BDE congeners and 6 alternate flame retardants. The objective of the present study was to explore concentrations of a variety of BFRs in house dust and to determine whether BFR exposure is associated with serum hormone levels in men.

2. Methods

2.1 Subject Recruitment

The present study utilizes serum hormone data collected from 62 participants in an ongoing study on environmental exposures and male reproductive health. Characteristics and descriptions of this population have been presented elsewhere (Meeker et al., 2008; Meeker et al., 2007; Hauser et al., 2003). Briefly, men between 18 and 54 years of age were recruited from the Vincent Memorial Andrology lab at Massachusetts General Hospital (MGH). The participation rate was approximately 65%. Male participants were from couples

seeking infertility treatment due to a male factor, a female factor, or a combination of both male and female factors. Exclusionary criteria included prior vasectomy or current use of exogenous hormones. Research ethics committees at participating institutions approved the study protocols, and all participants signed an informed consent.

2.2 Dust sample collection and analysis

A subset of male participants recruited in years 2002 and 2003 donated existing vacuum bags in the home. Upon enrollment, at which time serum samples were collected, men were asked to send their used vacuum bags from home using a pre-addressed mailer given to them by the research nurse at their clinic visit. Participants wrapped the used vacuum bag in aluminum foil, sealed it in a labeled plastic bag and mailed it within 1 month, typically within 2 weeks. Dust samples were stored at −20C until analysis. Dust was sieved using a 150 μm mesh sieve to obtain the fine fraction. Determination of the target analytes was performed by a gas chromatograph (Agilent 6890) coupled to an Agilent 5975 mass spectrometer (Agilent Technologies, Santa Clara, CA) operated in negative chemical ionization mode (GC/ECNI-MS) using the method by Stapleton et al. (2008). Laboratory blanks were low enough (<1%) for most analytes that blank correction was not needed except as follows. The average concentration of four laboratory blanks (sodium sulfate) was subtracted from each sample for TBB and TBPH. Method detection limits were calculated as three times the standard deviation of the laboratory blanks. The separate stereoisomers of HBCD were not distinguished, and HBCD is presented as the total of all three isomers. The two stereoisomers of Dechlorane Plus, syn-DP (sDP) and anti-DP (aDP) were quantified separately.

2.3 Serum hormones

Upon enrollment to the study, one non-fasting blood sample was drawn and centrifuged, and the serum was stored at −80C until analysis, which was conducted up to 12 months later at the MGH Reproductive Endocrine Clinic laboratory. The hormone analytical methods and QA/QC were described previously (Meeker et al., 2008). The methods employed were as follows: Follicle stimulating hormone (FSH), serum luteinizing hormone (LH), estradiol, prolactin, free T4, total T3, and thyrotropin (TSH) concentrations were determined by microparticle enzyme immunoassay using an automated Abbott AxSYM system (Abbott Laboratories, Chicago, IL,USA); Inhibin B was measured using a double-antibody, enzyme-linked immunosorbent assay (Oxford Bioinnovation, Oxford, UK); a Coat-A-Count RIA kit (Diagnostics Products, Los Angeles, CA, USA) was used to measure testosterone; sex hormone binding globulin (SHBG) was measured using an Immulite fully automated chemiluminescent immunometric assay (DPC, Inc., Los Angeles, CA, USA). The free androgen index (FAI) was calculated as the ratio of testosterone to SHBG. Additionally, free unbound testosterone was estimated using the equation byVermeulen et al. (1999).

2.4 Data analysis

Descriptive statistics were calculated for dust concentrations of the flame retardants (FRs). One half the limit of detection (LOD) was assigned to non-detect levels. Further statistical analysis was conducted for the alternate flame retardants and for PBDE congeners that were detected in over 85% of the dust samples. Spearman's correlation coefficients were calculated, using SAS version 9.2 (SAS Institute, Inc., Cary, NC, USA), to assess bivariate relationships between different FRs and between FR concentrations in house dust and serum hormone levels. These relationships were assessed using multivariable linear regression to control for potential confounding variables in R version 2.8.1 (R Foundation for Statistical Computing, Vienna, Austria). Regression models for PBDEs were run using individual PBDE congener concentrations and using summed concentrations of PentaBDEs (BDE 47, 99, 100), OctaBDEs (BDE 183 and 201), and DecaBDEs (BDE 206, 207, 208 and 209).The

congener grouping was based on three factors: the congener prevalence in commercial mixtures (ATSDR, 2004; La Guardia et al., 2006), the Spearman's correlation coefficients between congeners within a group in dust ($r > 0.80$, $p < 0.05$) and the Spearmans' correlation coefficients between congeners we detected in serum and matched dust samples ($r > 0.60$, $p < 0.05$; Johnson et al., 2010). Note that because fewer congeners were detected in serum, this data could only inform the pentaBDE congener grouping. Dust concentrations of BDE 47, 99 and 100 were combined with concentrations from a previous analysis of 24 men from the same study cohort (Meeker et al., 2009) so that the PentaBDE congener group has a greater total number (n=62) than the other analyses.

As a supplementary analysis, regression models were also run using independent factor variables generated by a factor analysis of all detectable congeners performed using SAS. The factor analysis utilized data on all congeners detected in dust, providing a data adaptive reduction in the number of predictors to be used in the subsequent regression model and identifying important congeners in each leading factor. The use of leading factors that are designed to be mutually independent eliminated the potential for collinearity arising from any correlation between variables in the same model. Leading factors that represented 90% of the variability among the congeners were included in the regression model for this method of analysis.

All variables were analyzed as continuous variables. The distributions of several hormone levels (estradiol, testosterone, inhibin B, free T4 and total T3) approximated normality and were not transformed in statistical models. Several other hormones (prolactin, FSH, LH, SHBG, FAI, and TSH) were skewed right and were transformed to the natural log (ln) for statistical analyses. FR concentrations in house dust were also transformed to the natural log. All multivariable models were adjusted for age and body mass index (BMI) because these factors are known to be associated with changes in hormone levels. Age and BMI may also be associated with differences in FR levels in the body, although the ranges of these parameters were not large in this cohort of men.

3. Results

The distributions of selected PBDE groupings and alternate FRs measured in house dust were highly skewed (log-normal distribution) (Table 1). The distribution and detection limits of all individual PBDE congeners measured were previously reported (Johnson et al., 2010). BTBPE was detected in 100% of the dust samples, while HBCD, TBPH and TBB were detected in 97, 63 and 47% of samples, respectively. All samples contained aDP and 96% contained sDP, although concentrations of these chlorinated flame retardants were lower than the brominated flame retardants. The medians and ranges of all hormone measurements are presented in a supplementary table alongside MGH reference ranges (Table S1).

Concentrations of PBDE congeners with similar degrees of bromination were strongly correlated (Spearman $r \geq 0.80$, $p<0.05$) (Johnson et al., 2010). The correlation coefficients for the grouped PBDE congeners included in the data analysis are shown in Table 2, along with coefficients for the alternate BFRs. Concentrations of tetrabromobenzoate (TBB) and tetrabromo phthalate (TBPH), which are both found in the same commercial products that replaced pentaBDE, were strongly correlated with one another (Spearman $r = 0.79$, $p<0.0001$). As expected, there were strong correlations between concentrations of the two stereoisomers of Dechlorane Plus, sDP and aDP (Spearman $r = 0.83$, $p<0.0001$). There was also moderate correlation between alternate BFRs and lower-brominated PBDEs ($r = 0.31–0.49$), octaBDE formulation congeners ($r = 0.34–0.60$), and decaBDE formulation congeners ($r = 0.31–0.43$).

Several bivariate relationships between alternate BFRs and hormone levels are presented in scatterplots (Figures 1 through 3). There was a positive correlation between BTBPE and total T3 ($r = 0.33$, $p = 0.04$). TBPH, which was detected in 63% percent of the samples, was also positively associated with total T3 ($r = 0.30$, $p = 0.07$). HBCD was positively correlated with free androgen index (FAI) ($r = 0.46$, $p = 0.004$) and inversely correlated with sex hormone binding globulin (SHBG) ($r = -0.35$, $p = 0.03$). Table 3 presents results from multivariable linear regression models, expressed as percent difference from the median hormone level associated with an interquartile range (IQR) increase in BFR dust concentration. After adjustment for age and BMI, the results remained similar to bivariate relationships. An IQR increase in pentaBDEs was associated with a 20% decrease in FSH as well as increases in SHBG, estradiol, free T4 and total T3. Additionally, there were significant positive associations between dust concentrations of octaBDEs and serum T4, TSH, LH and testosterone and a significant inverse association between dust concentrations of decaBDEs and testosterone (Table 3).

When analyzed as individual congeners (data not shown) in multivariate linear regression models adjusted for age and BMI, IQR increases in BDE 153 and 154 were associated with statistically significant 24% and 22% increases, respectively, in SHBG, and with 31% and 38% increases in prolactin. The sum of all congeners was also associated with a 6% increase in total T3.

Table 4 describes the factor pattern of 8 independent variables generated by the factor analysis. The cumulative proportions of the variability explained by each factor, as determined by eigenvalues of the correlation matrix from the factor analysis, were used in our decision to choose 8 factors to use in the regression models. These eight factors account for 90 percent of the variability of all the detectable congeners. Table 4 indicates that most of the variability of the congeners is accounted for in the first 2 factors, and the weightings indicate that factor 1 is most heavily weighted by pentaBDE congeners and factor 2 is heavily weighted by decaBDE congeners. The congeners that we grouped into our octaBDE group (BDE 183 and BDE 201) were not as clearly delineated by the factor pattern. Linear regression models were run for each hormone outcome with all 8 factors in the models. Results from these models (not shown) corroborate the findings from models using the congener groupings by commercial mixture presented in Table 3. For example, factors 2 and 3, which are mainly weighted by decaBDE congeners, were inversely and significantly associated with testosterone levels. Factor 1, which is heavily weighted by pentaBDE congeners, had a significant positive association with prolactin and total T3. These results are consistent with the associations shown for the congener groupings presented in Table 3.

4. Discussion

The FR concentrations in house dust in the present study were similar to those found in other studies in the United States (Stapleton et al., 2005, 2008; Allen et al., 2008; Sjodin et al., 2008). Similar to other studies, DecaBDE was the dominant mixture found in house dust, followed by pentaBDEs. Due to the decline in use of penta- and octaBDE mixtures since 2002–2003 when the dust samples were collected, it is reasonable to expect that indoor levels of these congeners may have declined. However, because of continued use of older products, it is uncertain. It is also possible that concentrations of alternate FRs may be increasing due to the increased use of alternate FRs as substitutes for PBDEs. As expected, there were strong correlations found among FRs that comprise the same commercial formulations. There was also some degree of correlation between different formulations, suggesting that these FRs may have originated from similar sources within the home.

Although several animal studies show decreased T3 and T4 levels following dosing with PBDEs, our findings of increased T4 levels associated with PBDE exposure are consistent with most other human epidemiological studies (Bloom et al., 2008; Turyk et al., 2008; Wang et al., 2010; Stapleton et al., 2011). A more recent study, however, found that lower levels of a pentaBDE mixture increased T4 levels in perinatally exposed rats (Blake et al., 2011), which is consistent with the human studies to date. The authors note that the relatively higher doses of PBDEs that decrease T4 levels in animal studies are also associated with increased liver weights (Zhou et al., 2001, 2002; Stoker et al., 2004) and may be the result of a different mechanism of action. However,Kuriyama et al. (2007) reported decreased T4 in rats exposed to BDE 99 at similarly low levels. It is difficult to draw conclusions about potential animal and human differences in thyroid hormone effects based on a few studies, particularly due to differences in study design such as specific congener or congener mixture tested, time of dosing, and timing of effects measurement.

The positive association between PBDEs and T3 levels we found is consistent with a study byDallaire et al. (2009) which found higher T3 levels associated with serum BDE 47 among a population of 623 Inuit adults. However, another large study of 308 men found lower T3 and TSH levels associated with serum PBDEs (sum of BDE 47, 99, 100 and 153) (Turyk et al., 2008). Other studies have reported both increases and declines in TSH levels in relation to PBDE exposure, which may be dependent on exposure level, population, specific congener measured or other study characteristics (Chevrier et al., 2010; Hagmar et al., 2001; Yuan et al., 2008; Zota et al., 2011). We observed higher TSH levels with increased PBDE exposure, and this association was statistically significant for the OctaBDE group.

There are very few human studies examining associations between BFR exposure and reproductive hormone levels.Meijer et al. (2008) reported positive associations between prenatal BDE 154 exposure and testosterone, SHBG, inhibin B and estradiol measured in male infants at 3 months of age.. We also observed a significant positive association between BDE 154 and SHBG. We also found positive associations between the summed pentaBDEs and inhibin B, SHBG and estradiol, although the relationship with inhibin B was not statistically significant. These findings may be consistent with *in vitro* studies where pentaBDEs showed estrogenic activity (Meerts et al., 2001; Hamers et al., 2006), but further research on this topic is needed to establish stronger evidence.

Animal studies on BFRs that measured reproductive hormone responses are limited. Gestational exposure to BDE 47 resulted in reduced FSH in males (Andrade et al., 2004). Our findings are consistent with the Andrade et al. findings of reduced FSH related to pentaBDE exposure. It is again difficult to compare results between studies, however. For example, many animal studies measured effects of gestational exposure while the present study estimates current exposure to adult men.

There may be certain limitations when comparing our findings to other studies using biomarkers of BFR exposure, considering we estimated exposure to BFRs by measuring concentrations in dust. However, we expect good agreement between serum and dust concentrations for at least the pentaBDE congener grouping, as we previously demonstrated (Johnson et al., 2010). Additionally, our previous report also showed that dust concentrations of higher-brominated congeners tended to be correlated to serum concentrations of lower-brominated congeners, which may have implications for exposure assessment in terms of debromination and congener-specific body burdens. Because PBDEs can debrominate within an organism (Huwe and Smith, 2007; Stapleton et al., 2004; Noyes et al., 2011), it is reasonable to predict that exposure to higher-brominated congeners in dust may result in body burdens of lower-brominated congeners. Because there was some degree of correlation between some PBDEs and BTBPE and TBPH, and these BFRs were

associated with the same hormone effects, further study is needed to confirm the associations involving these two alternate BFRs. Another potential limitation of this study is the population of men from couples seeking infertility treatment, which, if they respond differentially to PBDE exposure, may limit generalizability to the general population.

This work expands upon our previous study (includes a larger number of samples and analytes) where we found hormone level alterations associated with exposure to BDE 47, 99 and 100 as measured in house dust. The results in the present study are consistent with our earlier report among the original 24 samples (Meeker et al., 2009). The use of vacuum bag dust as a marker of exposure had the advantages of low cost and efficiency. Furthermore, as compared to spot sampling, vacuum bag dust may be a measure of longer-term integrative exposure representing the total home environment. Future studies should address validation of house dust as a marker of exposure for flame retardants other than PBDEs. Although we previously found a strong correlation between concentrations of the major penta formulation congeners in dust and serum (Johnson et al., 2010), BDE 153 was not correlated. Other flame retardants may also have alternative sources (e.g., diet) or characteristics that could affect the relationship between body burdens and environmental measures such as dust. Differences in vapor pressure may influence the contribution of compounds to other routes of exposure such as inhalation. However, in the absence of biomarkers of exposure for these compounds, dust may be an adequate surrogate estimate of exposure that likely underestimates body burden to some degree.

This study had a relatively small sample size, and a large number of relationships were investigated due to its exploratory nature. Further studies of these exposures and outcomes should include larger sample sizes to increase confidence and reduce the possibility that some of the findings are due to chance. We used a factor analysis to generate independent exposure variables and eliminate the potential problem of collinearity when including multiple variables in the same regression model. These results supported those of our *a priori* congener groupings, suggesting that it was appropriate to group the congeners using our criterion for at least the pentaBDEs and decaBDEs. Additionally, because house dust may contain a variety of chemical compounds to which people are potentially exposed, we cannot rule out the possibility that our reported findings may be due to unmeasured co-exposures or confounders. Although the present study was cross-sectional, exposure to these compounds is expected to be relatively constant if they originate from consumer products present in the home, and our exposure estimates are likely representative of longer term exposure.

The present study is the first to explore human exposure and associated effects of some of these compounds. It is also the first study to relate dust concentrations of decaBDEs and other alternate BFRs to hormone levels. These findings provide further evidence of altered hormone levels in relation to BFR exposure, and show that house dust may be an important source of exposure to both PBDEs and alternate flame retardants. Further research is needed to confirm these findings and to determine specific sources of BFR exposure. More reliable biomarkers are needed for some BFRs. Research is also needed to determine the public health implications of alterations in hormone levels by environmental exposures.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Brominated flame retardants (BFRs) including PBDEs and alternates are measured
- Exposure to BFRs is characterized from concentrations in participant vacuum bag dust
- Exposure to PBDEs and alternate FRs was associated with alterations in hormone levels

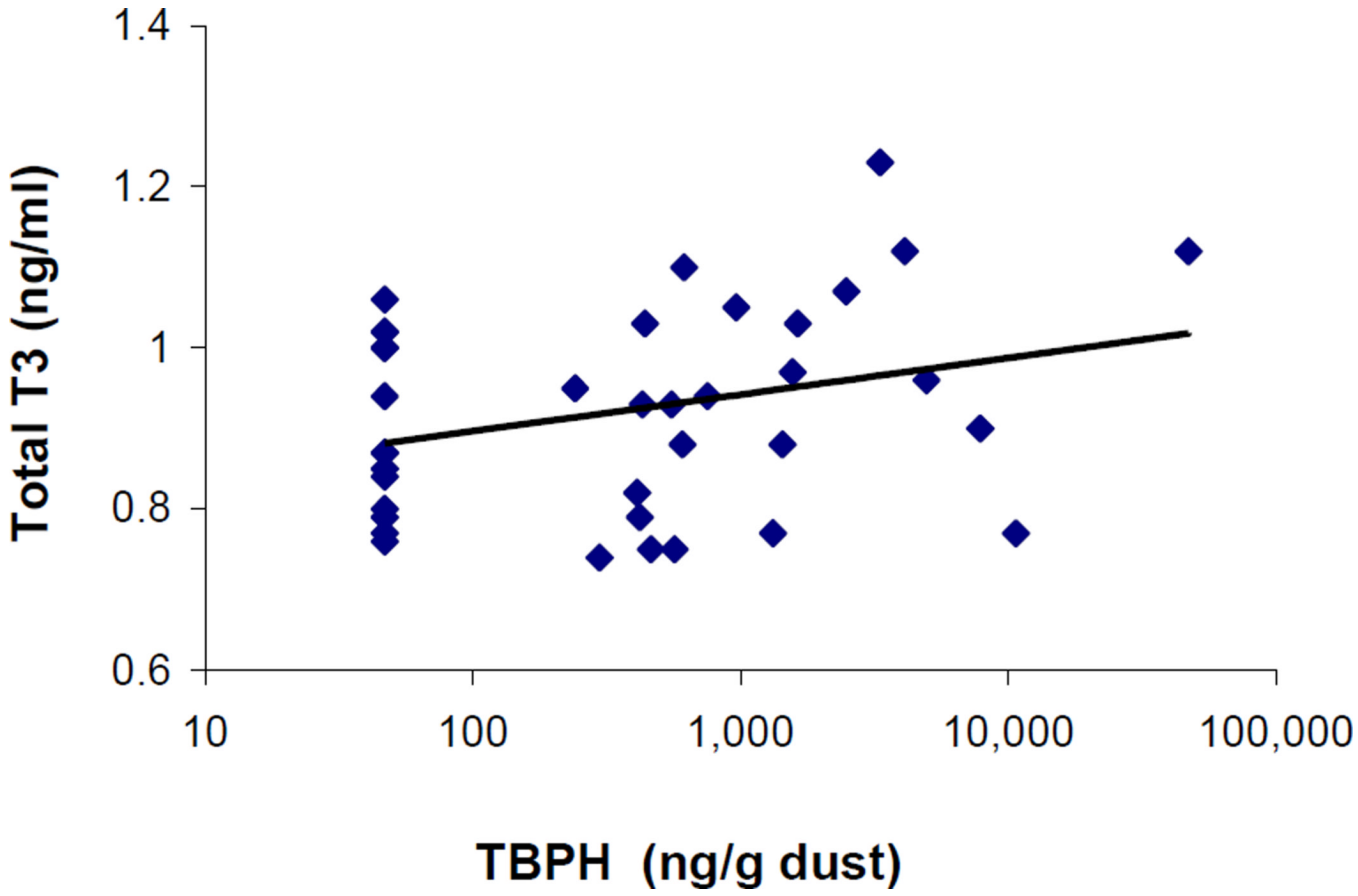

Figure 1.
Scatterplot of HBCD in house dust and ln-transformed free androgen index (FAI) (n = 38, Spearman's r = 0.46, p = 0.004). One outlier with a concentration of HBCD less than the detection limit was removed and did not affect the positive association.

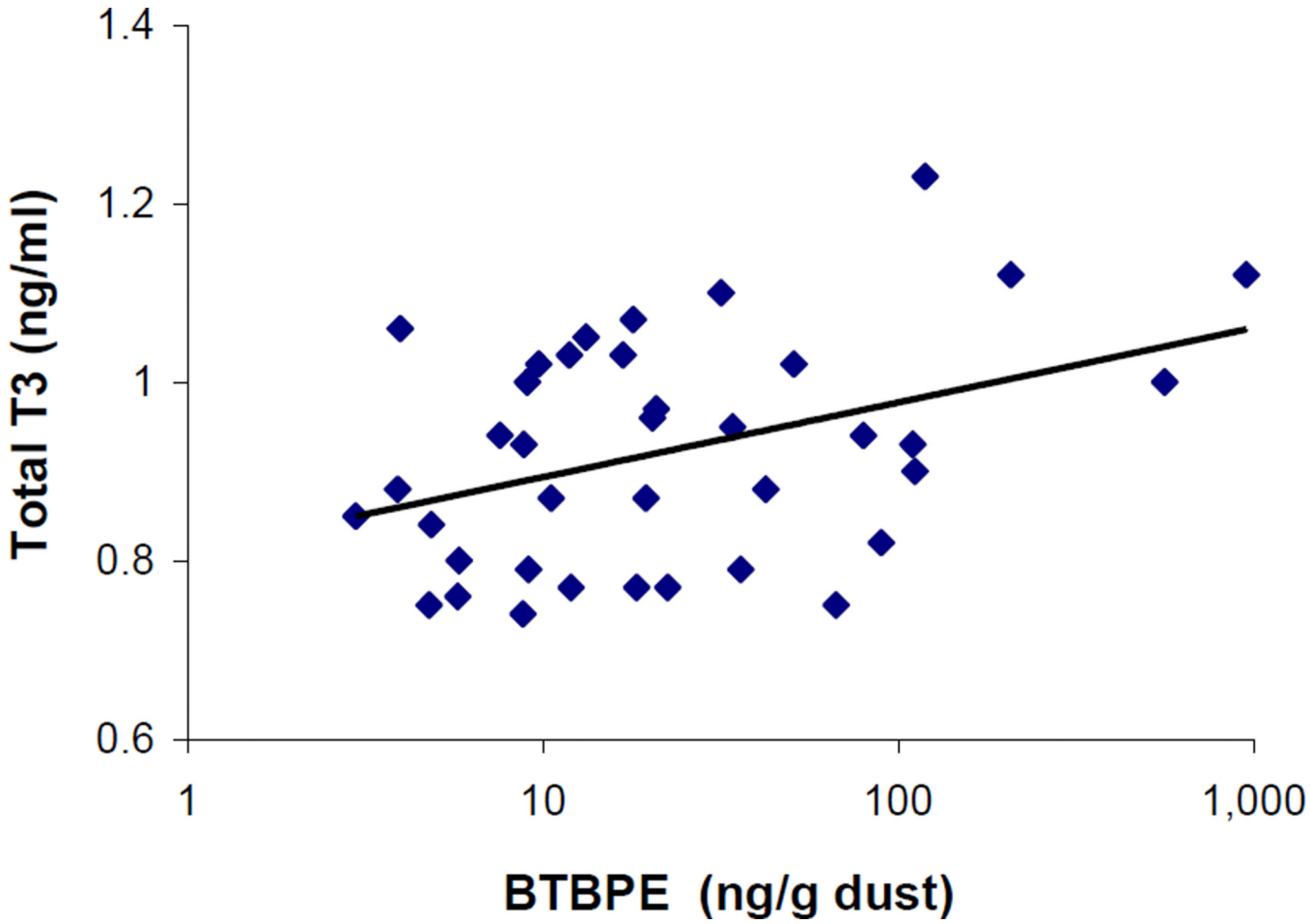

Figure 2.
Scatterplot of BTBPE in house dust and serum total T3 (n = 38, Spearman's r = 0.33, p = 0.04).

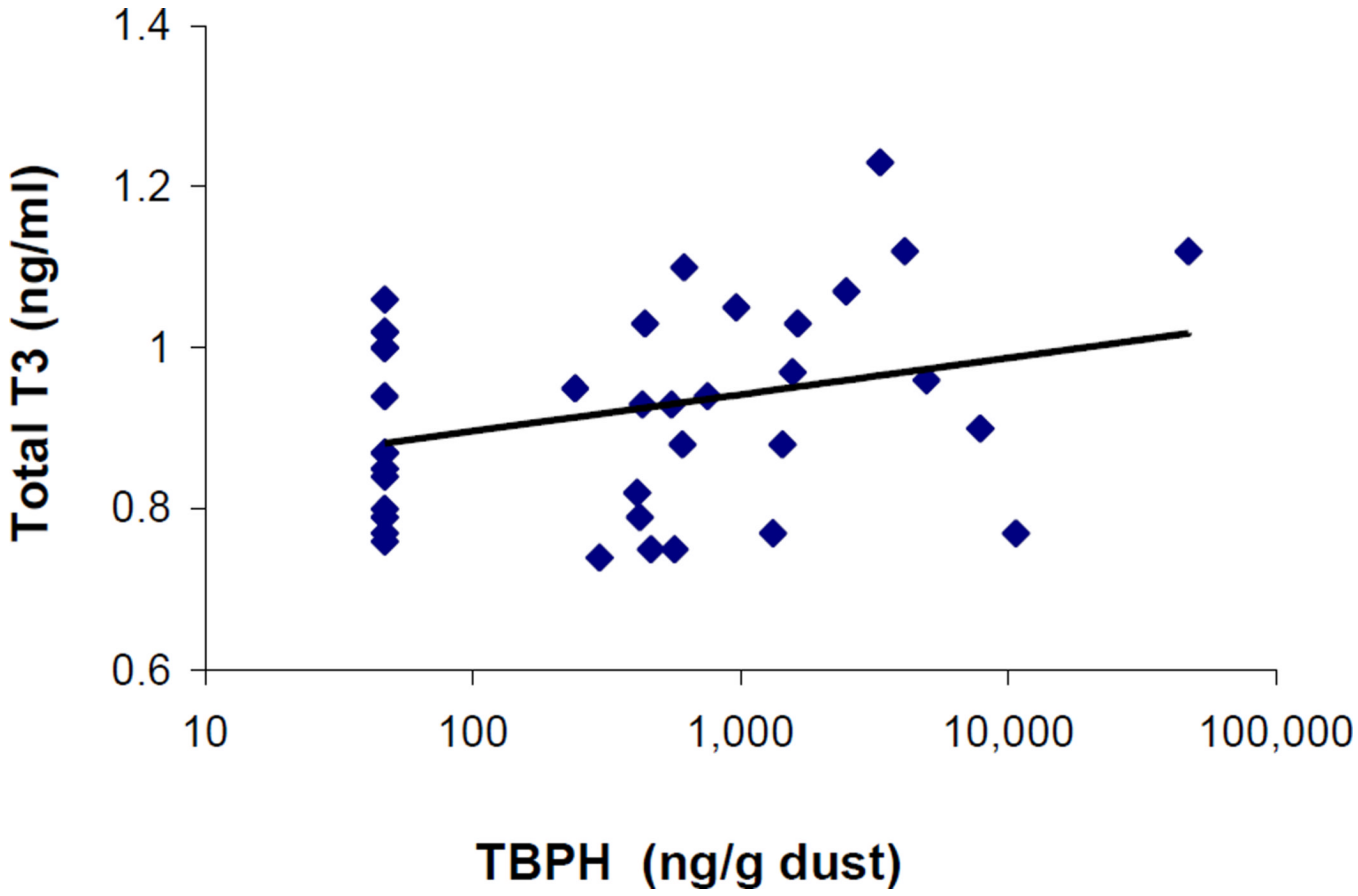

Figure 3.
Scatterplot of TBPH in house dust and serum total T3 (n = 38, Spearman's r = 0.30, p = 0.07).

Table 1

Distribution of flame retardants in house dust, ng/g (n = 38)

		Percentiles					
	Mean[d]	**25th**	**50th**	**75th**	**90th**	**Maximum**	**% detected [e]**
PentaBDEs[a]	1391	667	1049	2,936	5,707	22,300	100
OctaBDEs[b]	37.6	21.9	30.5	66.9	116	1,181	100
DecaBDEs[c]	2,287	1,387	1,800	3,228	6,609	38,483	100
TBB	409	68.4	68.4	1,494	13,221	72,460	47
HBCD	197	107	246	391	1,103	1,999	97
BTBPE	22	8.90	18.2	48.7	113	953	100
TBPH	377	47	435	1,408	4,363	47,110	63
sDP	3.16	2.47	4.26	7.38	14.6	43.1	89
aDP	9.60	6.00	8.85	14.7	29.8	68.4	100

[a] PentaBDE is sum of BDE 47, 99 and 100

[b] OctaBDE is sum of BDE 183 and 201

[c] DecaBDE is sum of BDE 206, 207, 208 and 209

[d] Geometric mean

[e] Percent of samples with detectable concentration of analyte

Table 2

Spearman correlation coefficients among select flame retardants in house dust (n=38).

	BDE	47	99	100	183	201	206	207	208	209	TBB	TBPH	BTBPE	HBCD	aDP	sDP
BDE 47	r	1.00	0.93	0.94	0.52	0.40	0.17	0.22	0.14	0.13	0.31	0.38	0.37	0.27	0.11	0.18
	p-value		<0.0001	<0.0001	0.0009	0.01	0.30	0.19	0.40	0.44	0.06	0.02	0.02	0.11	0.5	0.28
BDE 99	r		1.00	0.97	0.49	0.36	0.19	0.26	0.21	0.17	0.26	0.31	0.35	0.19	0.10	0.12
	p-value			<0.0001	0.002	0.03	0.27	0.11	0.21	0.29	0.12	0.06	0.03	0.26	0.57	0.47
BDE 100	r			1.00	0.54	0.41	0.22	0.29	0.23	0.22	0.31	0.37	0.39	0.17	0.13	0.15
	p-value				0.0004	0.01	0.19	0.08	0.16	0.19	0.05	0.02	0.01	0.3	0.45	0.36
BDE 183	r				1.00	0.87	0.21	0.36	0.18	0.16	0.50	0.44	0.47	0.05	0.29	0.34
	p-value					<0.0001	0.20	0.02	0.28	0.33	0.002	0.006	0.003	0.75	0.08	0.03
BDE 201	r					1.00	0.39	0.53	0.37	0.30	0.60	0.55	0.43	0.12	0.40	0.52
	p-value						0.02	0.0005	0.020	0.07	<0.0001	0.0003	0.007	0.49	0.01	0.0008
BDE 206	r						1.00	0.93	0.94	0.84	0.28	0.28	0.28	−0.24	0.22	0.37
	p-value							<0.0001	<0.0001	<0.0001	0.09	0.09	0.09	0.14	0.19	0.02
BDE 207	r							1.00	0.96	0.83	0.40	0.35	0.32	−0.16	0.24	0.43
	p-value								<0.0001	<0.0001	0.01	0.03	0.05	0.33	0.15	0.008
BDE 208	r								1.00	0.85	0.31	0.30	0.28	−0.20	0.23	0.36
	p-value									<0.0001	0.05	0.07	0.09	0.23	0.16	0.02
BDE 209	r									1.00	0.29	0.19	0.27	−0.16	0.22	0.36
	p-value										0.08	0.26	0.11	0.33	0.19	0.03
TBB	r										1.00	0.79	0.31	0.04	0.30	0.39
	p-value											<0.0001	0.06	0.80	0.07	0.01
TBPH	r											1.00	0.43	0.16	0.32	0.46
	p-value												0.01	0.35	0.05	0.004
BTBPE	r												1.00	−0.09	0.45	0.47

	BDE	47	99	100	183	201	206	207	208	209	TBB	TBPH	BTBPE	HBCD	aDP	sDP
	p-value													0.59	0.004	0.003
HBCD	r													1.00	0.20	0.15
	p-value														0.23	0.36
aDP	r														1.00	0.83
	p-value															<0.0001
sDP	r															1.00
	p-value															

Shading indicates significant correlation (p</= 0.05). Dark shading indicates very strong correlation (r>0.80).

Table 3

Percent difference[a] in hormone level (95% confidence intervals,) relative to population median, associated with an interquartile range (IQR) increase in house dust BFR concentration

	PentaBDE[c] (n=62)[f]			OctaBDE[d] (n=38)			DecaBDE[e] (n=38)		
			p-value			p-value			p-value
FSH	−20.2	(−34.7, −2.5)	0.03	4.4	(−13.2, 25.6)	0.65	1.8	(−15.5, 22.7)	0.85
LH	−9.4	(−24.1, 8.2)	0.28	15.5	(0.6, 32.6)	0.05	−6.6	(−19.2, 8.1)	0.37
Inhibin B	18.2	(−4.8, 41.3)	0.13	−5.8	(−29.4, 17.7)	0.63	−6.2	(−29.9, 17.5)	0.61
Testosterone[b]	3.6	(−7.4, 14.7)	0.52	9.0	(0.8, 17.2)	0.03	−9.4	(−17.6, −1.2)	0.02
SHBG	16.8	(0.7, 35.4)	0.05	9.2	(−6.1, 27.0)	0.26	−10.2	(−22.8, 4.4)	0.17
FAI	−10.6	(−22.5, 3.2)	0.13	−0.7	(−13.9, 14.5)	0.92	0.4	(−13.0, 16.0)	0.95
Estradiol	17.1	(0.0, 34.2)	0.05	14.4	(−2.8, 31.7)	0.11	−8.5	(−26.3, 9.3)	0.36
Prolactin	10.8	(−6.0, 30.7)	0.23	13.8	(−2.9, 33.4)	0.12	4.5	(−11.4, 23.3)	0.60
Free T4	3.6	(0.6, 6.5)	0.02	3.3	(1.0, 5.6)	0.01	−1.7	(−4.2, 0.9)	0.20
Total T3	5.4	(0.0, 10.7)	0.05	4.3	(−0.2, 8.8)	0.07	1.7	(−3.1, 6.4)	0.50
TSH	14.1	(−4.7, 36.7)	0.16	21.2	(0.8, 45.8)	0.05	11.1	(−8.5, 34.9)	0.29

[a] Adjusted for age and BMI.

[b] Free testosterone estimated using equation by Vermeulen et al. 1999.

[c] PentaBDE is sum of BDE 47, 99 and 100. IQR = 2268 ng/g for n=38 and 2985 ng/g for n=60.

[d] OctaBDE is sum of BDE 183 and 201. IQR = 39 ng/g.

[e] DecaBDE is sum of BDE 206, 207, 208 and 209. IQR = 1876 ng/g.

[f] Includes additional 24 samples from prior preliminary analysis, and models are also adjusted for difference in dust analytical method.

Table 4

Factor pattern for 8 independent variables, representing weightings of each congener, for all PBDE congeners detected in house dust.

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Factor 6	Factor 7	Factor 8
BDE 17	0.61921	−0.27052	0.19168	0.17357	−0.41984	−0.04351	0.33098	−0.07932
BDE 25	0.19201	−0.36883	−0.12747	0.39082	−0.30498	−0.46457	−0.01396	0.49010
BDE 28/33	**0.81550**	−0.38798	0.06208	0.07015	−0.03569	0.07351	−0.06067	−0.07033
BDE 30	−0.13225	−0.02905	0.20550	−0.53665	0.12428	−0.28102	0.68385	0.01557
BDE 47	**0.95770**	−0.21810	0.02513	0.01519	−0.00523	0.04297	−0.00737	−0.08296
BDE 49	0.42602	−0.00713	0.15241	−0.25128	−0.50311	0.52132	0.07574	0.15202
BDE 66	0.73432	−0.32019	0.08162	0.18837	−0.29711	0.08202	0.20863	−0.16743
BDE 75	0.23279	−0.28282	0.03633	0.49519	0.36456	0.55011	0.15913	−0.17713
BDE 85/155	**0.96164**	−0.17075	0.02102	−0.06266	0.03537	−0.10549	−0.07274	−0.01817
BDE 99	**0.96213**	−0.21373	0.04277	−0.03854	−0.00481	−0.08907	−0.02396	−0.03014
BDE 100	**0.97057**	−0.17617	0.00468	−0.06570	0.04365	−0.07927	−0.07759	−0.02223
BDE 138	0.38529	−0.17239	0.54641	−0.17250	0.36090	0.23085	−0.26340	0.15432
BDE 153	**0.95584**	−0.14743	−0.06018	−0.08995	0.03895	−0.11845	−0.05213	0.02262
BDE 154	**0.84431**	−0.01778	−0.07175	−0.22286	0.24656	−0.19706	−0.22573	0.13240
BDE 183	0.53488	0.19800	−0.77153	−0.11729	0.11291	0.00159	−0.03000	−0.03953
BDE 201	0.43644	0.42526	−0.73047	−0.00085	0.09663	−0.00121	−0.00375	−0.10207
BDE 202	0.40385	0.63789	−0.02366	−0.06827	0.06280	−0.17517	0.17920	−0.29929
BDE 203/200	0.06711	0.61525	−0.46298	0.28807	−0.22872	0.26425	0.08367	0.23107
BDE 205	0.13333	−0.01979	0.18782	0.66479	0.46476	−0.23387	0.27388	0.09357
BDE 206	0.30397	**0.85720**	0.31233	0.08345	−0.05574	−0.05673	−0.03512	−0.00946
BDE 207	0.40369	**0.87831**	0.12270	0.10306	−0.05670	−0.06045	0.02305	−0.06370
BDE 208	0.35133	**0.84867**	0.31784	0.10512	−0.07158	−0.04356	0.02223	−0.04902
BDE 209	0.31736	**0.76528**	0.42895	0.02329	−0.00433	0.02991	−0.17121	0.20390
Variability [a]	0.3631	0.5547	0.6483	0.7123	0.7695	0.8234	0.8691	0.9025

[a]Cumulative proportions of the variability explained by each factor, as determined by eigenvalues of the correlation matrix from the factor analysis. Eight factors account for 90 percent of the variability of all congeners.

Bolding indicates which congeners are weighted more heavily. Factor 1 is weighted most by PentaBDE congeners and Factor 2 is weighted most by DecaBDE congeners.