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Viewpoint

Are PBDEs an environmental equity concern? Exposure disparities by socioeconomic status

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Author's Viewpoint

RHONDA SAUNDERS

There has been a surge of new research highlighting the potential health consequences of polybrominated diphenyl ethers (PBDEs), brominated flame retardants which have been added to consumer products since the 1970s to reduce flammability of electronics and furniture. Due to their persistent nature and tendency to bioaccumulate, these compounds are ubiquitous and have been detected in human tissues, marine mammals, house dust, and virtually any biological or environmental media taken from anywhere on the planet.

Exposure assessment studies have documented body burden levels in various populations and investigated contributors of human exposure. These studies suggest that PBDE exposures are not homogeneous across diverse groups. For example, recent studies have shown higher exposures among young children compared to adults (*1*). This finding is consistent with exposure profiles of other environmental contaminants, such as lead, where dust is an important exposure media; indeed, children spend more time close to the ground and engage in hand-to-mouth behavior which may increase their dust intake. There are also significant geographic differences in PBDE exposures with much higher serum, breast milk, and house dust levels reported in the U.S. compared to Europe. Within the U.S., PBDE congeners characteristic of penta-BDE (e.g., BDE-47, -99, and -100) occur at higher concentrations in house dust and blood samples collected from Californians compared to other U.S. residents. This difference in exposure levels within the U.S. is likely due to California's unique furniture flammability standard (TB117), which appears to be associated with body burdens that are twice as high as the rest of the U.S. and 10-fold higher than levels in Europe (*2*).

A third group that may face disproportionate exposures to PBDEs is people of lower socioeconomic status (SES). Several studies have independently found that non-White racial and ethnic groups, as well as populations with lower household income, and lower educational attainment, have significantly higher PBDE body burdens.

The first study to show a difference in PBDE exposures by SES was published by Zota et al. in 2008 using nationally representative data from 2040 participants in NHANES (*2*). After controlling for geographic residence, race/ethnicity, age, sex, and country of origin, the authors found that individuals residing in lower income households (<$20,000/year) had significantly higher PBDE exposures compared to those from higher income households (≥$20,000/year) ($\Sigma PBDE_6$ = 50.4 vs 37.7 ng/g lipid; $p < 0.01$). Two recent studies provide further evidence that PBDE body burdens may differ by SES. In a study of 100 children aged 2−5 years old from California, Rose et al. found higher body burden levels of nearly all measured congeners (including BDE-47, -153, and -209) in children born to mothers with lower educational attainment (less than college degree versus college degree or above) (*1*). Similarly, a study among an ethnically diverse population of 6−8 year old girls in California and Ohio (N = 600) that measured congeners characteristic of penta-BDE found that the PBDE sum ($\Sigma PBDE_6$) was lower in girls with higher educated care-givers (*3*). This association persisted after adjustment for age, BMI, race/ethnicity, geographic site, and maternal age.

Racial and ethnic differences in PBDE exposure are more complex and appear to vary by demographic composition of the study population and the covariates included in statistical models. The first study to examine differences in PBDE exposure by race/ethnicity was Sjodin et al.'s study of PBDE exposures in NHANES (*4*), which did not account for geographic residence. After adjusting for age, they found that levels of BDE-47 and BDE-99 (but not BDE-100 and BDE-153) were significantly lower in Whites compared to Mexican Americans and Blacks. They also examined univariate associations between BDE congeners and country of origin and found that BDE-153, but not BDE-47, was significantly

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lower among foreign-born residents compared to their native-born counterparts.

When Zota et al. (*2*) analyzed the same data set and accounted for geographic residence (California versus the rest of the U.S.) and several other covariates (see list above), they did not find significant differences in PBDE exposure by race/ethnicity, but did find significantly lower exposures in $\Sigma PBDE_6$ and BDE-47 among foreign-born residents compared to their native-born counterparts, which is consistent with the comparatively high usage of these chemicals in the U.S. Sjodin's finding (*4*) of higher PBDE levels in Mexican Americans may result from the large proportion of Mexican Americans from California surveyed in NHANES. Similarly, his lack of association between country of origin and BDE-47 may be due to the high proportion of foreign-born participants from California in NHANES.

The two new reports of PBDE levels in Californian children also found differences in PBDE body burden by race/ethnicity. Windham et al.'s study (*3*) of preadolescent girls found that Black girls had significantly higher levels than White girls ($PBDE_6 = 95.5$ vs 73.4 ng/g lipid) with Hispanics having intermediate values and Asians having the lowest values. These differences persisted after adjusting for geographic site, BMI, age, primary caregiver's education, and maternal age. Unfortunately, this study did not account for caregiver's country of origin. In contrast, Rose et al. (*1*) found that among their largely Hispanic and White study population, Hispanic children had lower levels of higher brominated congeners (ΣBDE- 197−209) but no significant differences in race/ethnicity were seen among the lower brominated congeners (ΣBDE-28−153). Children of foreign born mothers, 52% of whom were Hispanic, had significantly lower levels of higher brominated congeners.

The root causes of these disproportionate exposures have not been well researched. One potential pathway linking higher PBDE body burden to lower SES populations may involve differences in housing stock and furniture quality. For example, the physical weathering and crumbling of PBDE-treated foam in older furniture, more often found in lower income homes, may release greater amounts of penta-BDE compounds into indoor environments (*5*). In addition, cheaper furniture may be manufactured in ways that either use more of these chemicals or allow the release of these chemicals in greater amounts during use. There may also be SES differences in housing factors, such as air exchange rates, which can modify exposure. Previous studies have shown that indoor air pollutants may be elevated in lower SES homes due to poor ventilation and small apartment size. Consistent with this idea, Rose et al. (*1*) found that children living in larger homes had significantly lower levels of BDE-209 and higher maternal education was correlated with larger homes. Dust concentrations within these larger homes might be lower since sources strengths (e.g., the total quantity of PBDEs across all pieces of furniture) might not scale linearly as square footage increases.

Another potential pathway that could contribute to PBDE exposure disparities is dietary differences. Differences in diet could lead directly to increases in PBDE exposure through intake of contaminated foods, such as animal fat. Alternatively, differences in dietary composition could modify absorption or metabolism of PBDE exposures from the indoor environment. Similarly, iron or calcium deficiency can increase children's absorption of lead from contaminated environments.

These observed differences in PBDE body burden by SES are intriguing particularly as these patterns differ from those observed for PCBs, another group of persistent, organohalogen pollutants that tend to be higher among higher SES groups suggesting that simplified characterization of dietary patterns do not account entirely for these trends (*3*). These studies also point to the complex interactions among race, class, and geographic residence (both former and present) and the need for focused studies which thoughtfully model and interpret socioeconomic influences of environmental exposures.

Given that PBDEs exposures may be higher among lower income populations and have the ability to disrupt the thyroid system, future studies should evaluate the impact of these exposures on thyroid-mediated health end points, such as preterm birth and hypertension, where there is a persistence of SES health disparities. Additionally, animal studies should examine potential interactions between PBDEs and other stressors which may be elevated in vulnerable populations to avoid underestimating potential health risks and to better account for background susceptibility due to environmental and nonenvironmental stressors.

Finally, community-based participatory research (CBPR) strategies can be used to examine exposure levels in low-income communities of color to assess potential disparate burdens of PBDE exposures in these populations. The disparate impact of lead exposure on low-income communities of color is associated with housing quality, age of housing stock, and other structural factors that have placed these communities at higher risk of adverse health effects from this hazard. We can learn valuable lessons from the history of lead to inform how we examine potential SES disparities in PBDE exposures which appear to be linked to poorer housing quality and the wearing of consumer products such as furniture foam. It is also important to recognize that the potential persistence of PBDEs and other semivolatile organic compounds (SVOCs) in the indoor environment adds complexity to the problem of addressing disparities in exposure and their associated health effects because disparities may be long-lived in communities and resistant to short-term changes in the causal determinants. In addition to enhancing our understanding of exposure patterns in diverse communities, CBPR can help scientists elucidate economically and culturally appropriate intervention strategies to reduce PBDE exposures.

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