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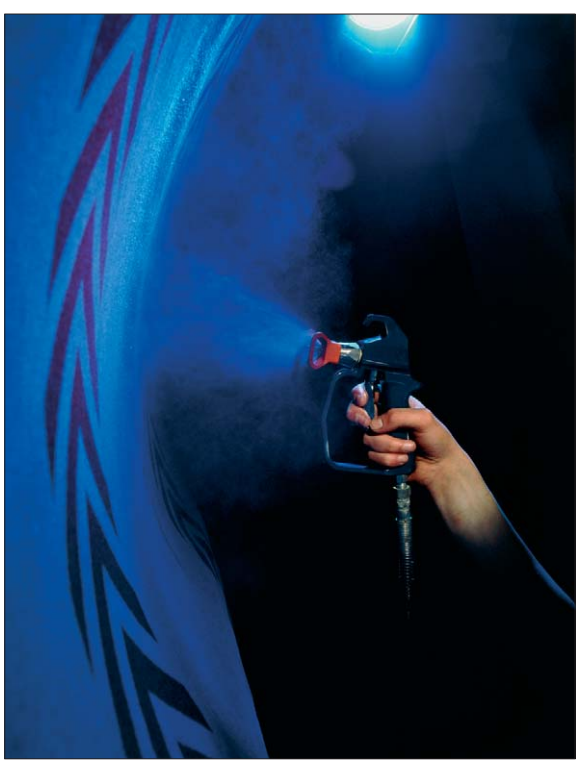
Brominated flame retardants—safety at what cost?

Polybrominated diphenyl ethers (PBDEs) are a group of brominated compounds (209 congeners) that are typically used as flame retardants in the manufacture of furniture, textiles, and electronic equipment. PBDEs have been used since the 1970s. However, because of increasing evidence of potential health risks associated with exposure to PBDEs, European and US regulatory authorities have taken steps to reduce exposure in human beings by the exclusion of penta-brominated and octa-brominated flame retardants from these markets. Additionally, the deca-brominated mixture has been targeted for phasing out from 2008. Although restricted use and replacement of PBDEs will minimise their release into the environment, a substantial problem still remains because of the large amount of existing consumer products in homes and workplaces to which people might be exposed.

Human exposure to PBDEs is reflected by the large increase in the concentration of such chemicals in breast milk. PBDEs in breast milk of North American women increased from less than 1 μg/L to 200 μg/L over a 25-year period,[1] and the concentration of PBDEs in the breast milk of Swedish women increased by 60 times between 1972 and 1997.[2] The bioaccumulative nature of PBDEs and the increasing concentrations of these compounds in human samples mirror previous reports for polychlorinated biphenyls (PCBs) and DDT before their bans. However, by contrast with exposure to PCBs and DDT, which show higher tissue concentrations with increasing age, PBDE concentrations in infants are greater than in adults.[3,4]

Wu and co-workers[5] investigated the body burdens of first-time mothers to determine key routes of exposure to PBDEs. Breast milk and household dust were analysed for PBDE congeners and information about dietary habits was collected by questionnaire. Although statistically significant associations were seen between PBDEs in breast milk and consumption of dairy fat and meat, the inaccuracy of dietary information, because of questionnaire constraints, resulted in a large portion of the current model remaining unexplained. A more intricate model is needed to explain the relation between dietary exposure and PBDEs in breast milk. Another limitation was the absence of data on the concentration



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of PBDEs in cohort diets, which makes it impossible to estimate the contribution of this exposure pathway to body burdens of PBDE. Market-basket surveys[6,7] have highlighted the variability in PBDE concentration in food products, although adult dietary intake of PBDEs could range from 0·9 to 1·5 ng/kg a day, which represents less than 20% of the total PBDE daily exposure.[8]

By contrast with adults, nursing infants might receive up to 300 ng/kg of PBDEs a day via breast milk,[6] which highlights the importance of this pathway of exposure for children aged 1 year or younger. Unlike for dietary exposure, Wu and colleagues recorded a strong positive correlation between the concentration of PBDEs in breast milk and that of household dust. Other studies have suggested that incidental ingestion of household dust is a major contributor to exposure to PBDEs in young children and adults,[9,10] although little information is available about the contribution of dust inhalation to exposure. Exposure to PBDEs via household dust is again more important for younger age groups than for adults: toddlers' exposure to PBDEs from household dust could be 100 times greater than that for adults[9] because of the increased frequency of hand-to-mouth contact.

Irrespective of the exposure pathway, the increased exposure of infants and young children to PBDEs is of great concern because of the risk of adverse effects during early stages of development.[10] Although no large-scale epidemiological studies have looked at the effect of PBDEs in human beings, especially children,[4] animal bioassays suggest that the most sensitive endpoint for PBDE toxicity is thyroid function (ie, induction of thyroid hyperplasia and changes in production of thyroid hormones).[11] Exposure to penta-bromodiphenyl ether alters the expression of proteins in the striatum and hippocampus that have roles in neurotransmitter functioning and other aspects of cognition.[12] As a result, exposure of fetuses in utero or infants via breast milk and dust to PBDEs might lead to neurological deficits, which have been observed after exposure to the structurally similar PCBs. The challenge for environmental health professionals is to enhance the understanding of factors that affect the fate, transport, and bioavailability of PBDEs in indoor environments, to develop biomarkers for the assessment of exposure to PBDEs, and to elucidate the effect of such exposure in susceptible populations.

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