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HEALTH EFFECTS REVIEW

Polybrominated Diphenyl Ethers (PBDEs)

Introduction

Polybrominated diphenyl ethers (PBDEs) are persistent and bioaccumulative compounds used as fire retardants in polyurethane foams, plastics, and textiles. Common products that contain PBDEs include furniture cushions, carpet backings, electrical insulation, and computer and television casings [1]. These chemicals are structurally similar to polychlorinated biphenyls (PCBs), which are known neurotoxicants. The three most widely used PBDEs are penta-BDE, octa-BDE, and deca-BDE [2]. The fully brominated deca-BDE, once believed to be environmentally stabile and biologically inert, is now shown to be bioavailable and toxic [3]. Deca-BDE is also converted to lower weight congeners both in the sunlight and when metabolized in animals.

PBDEs are not chemically bound to foam or plastic and can be released into the environment. Studies of body burdens of PBDEs in blood, tissue, and breast milk suggest that concentrations in North America are 10-100 times higher than in Europe and that concentrations have been increasing over time [4]. These levels are of concern for neurodevelopmental effects in the fetus and early childhood. This report summarizes studies of PBDE human exposure levels and their potential health effects.

Routes of Exposure

PBDEs were first discovered in the environment in 1979 and their presence today is felt around the world [5]. Worldwide production of PBDEs has more than tripled over the last two decades to 67,000 tons per year [6]. Estimates of market demand for all PBDEs suggest that 58% of world demand occurs in Asia, 26% in the Americas, and 14% in Europe [7]. Because of the widespread use of PBDEs as flame-retardants, they have been found throughout the environment, in animals and humans.

Routes of human exposure and the relative importance of different exposure routes remains unknown. Much like PCBs, PBDEs are bioaccumulative in fat and have been found in animal species throughout Canada and the United States, including harbor seals [8], fish [6, 9], herring gull eggs [10], and beluga whales [11]. PBDEs enter the food chain through fatty foods like fish. While very little data is available on exposure to PBDEs from food consumption, the contribution is believed to be in the range of 0.2-0.7 micrograms per day [12]. Occupational exposure of PBDEs has been identified for employees of computer recycling facilities. PBDEs from computers, foams, carpets, and electrical appliances also appear to contaminate indoor human environments, making inhalation of contaminated dust and dermal absorption of chemicals possible exposure routes as well [7].

Human Exposure Levels

PBDE has been detected in people all over the world to varying degrees. An extensive meta-analysis of concentrations showed that North Americans had the highest levels, followed by Europeans and then Asians [13]. Over the past thirty years, human levels of PBDEs have increased by a factor of almost 100. Concentrations of PBDEs in U.S. samples are approximately 20 times higher than European samples.

Pioneering work examining PBDE levels in humans was conducted in Sweden. A trend study examining PBDE levels in Swedish human milk from 1972 to 1997 showed that total PBDE levels increased from 0.07 to 4.02 ng/g over the 25 years [14].

A recent Swedish milk study of 39 Swedish women found a median PBDE value in breast milk of 3.4 ng/g [12]. The maximum value of 28.2 ng/g was an outlier, with the second highest value being only 9.4 ng/g. Both smoking and body mass index (BMI) were found to be significantly associated with PBDEs in breast milk. The study also considered age, computer usage frequency, place of residence, birth weight of the child, consumption of fish, and consumption of alcohol, but no significant relationships were found.

A study of Swedish mother/son pairs revealed extremely high levels of PBDEs in 10 of the 220 subjects sampled, with one subject exceeding 1000 ng/g [15]. Occupational exposure was not a factor in any of the extreme cases. In two instances, both mother and son had extremely high levels, suggesting a source of PBDEs may be in the home environment.

In the U.S., Mazdai et al. conducted a study of 12 mother/infant pairs [16]. Maternal and cord blood were analyzed for PBDEs, and results showed a high correlation between fetal and mother blood samples. Concentrations in the maternal blood samples ranged from 15 to 580 ng/g, and concentrations in the fetal blood samples ranged from 14 to 460 ng/g. These concentrations were 20 to 106 times higher than concentrations reported in Swedish studies.

A study by Schecter et al. looked at PBDE levels in breast milk and found similar concentration ranges [4]. The concentrations ranged from 6.2 to 419 ng/g. This study further supported evidence suggesting levels in the U.S. were up to 100 times higher than in Europe.

Studies by Ryan et al. [17] show that levels of PBDEs in breast milk samples from Canada were much higher than Sweden, but still lower than the U.S. However, where data has shown that levels in Sweden have started to decline, levels in North America are still increasing over time. In fact, levels of PBDEs in North America are estimated to be doubling every two to five years [18].

Toxicity

Based on animal data and similarities with PCBs, thyroid hormone disruption and neurobehavioral effects are two

important health concerns of PBDEs. Structurally similar to thyroxine, a thyroid hormone, PBDEs compete with thyroxine for binding to plasma transport proteins, one of the vehicles that the hormones use in their transport around the body. The ability of PBDEs to bind to the transport proteins may facilitate exposure to the fetal brain.

Studies in mice and rats showed decreased thyroxine levels in both the fetuses and the dams as a result of PBDE exposure [2]. This may have serious effects on proper brain development during the early postnatal period. The mother is the major source of thyroid hormone during critical periods of brain development in the fetus.

Studies were conducted that investigated effects of PBDE congeners on the neurobehavior of mice [19, 20]. Results showed that PBDE congeners administered to mice caused permanent aberrations in spontaneous behavior and reduced learning and memory functions in adult mice. The neurobehavioral effects were dose-dependent and worsened with age. Because there are many critical windows for neurodevelopment, further studies are needed that dose throughout the various stages of nervous system development and that accurately assess the health risk of low doses of PBDEs.

Policy

The lowest dose that resulted in adverse health effects in animal studies is orders of magnitude greater than exposure estimates and body burdens facing humans [2]. With that being said, there is still very little known about PBDEs compared to other chemicals like PCBs. More studies are needed on non-dietary sources and toxicological endpoints. Despite the lack of current toxicological information and human epidemiological studies, the European Union felt that the potential future health risks were serious enough to take precautionary action. Concern stemmed from the fact that PBDEs are persistent, bioaccumulative, structurally similar to PCBs and thyroid hormones, and are found at high levels in the environment and in people. In July 2003, the European Commission announced that the sale and use of penta-BDE and octa-BDE would be banned starting in 2006 [5], thus setting an example for the world to follow.

In Canada, where PBDEs are not manufactured but are imported, emphasis has been placed on risk management measures [1]. Based on screening assessments, Environment Canada and Health Canada have concluded that PBDEs posed a risk to some environmental organisms, but that human exposure levels were low. By taking action to reduce PBDE levels in the environment, the increasing trend in human PBDE levels will likely be slowed.

In the U.S., policy is being implemented at a state level. In August 2003, California passed a law banning penta- and octa-BDE starting in 2008. The state of Maine passed a bill to ban penta- and octa-BDE by January 2006 and deca-BDE by January 2008, becoming the first state to ban deca-BDE assuming safer alternatives exist.

The assumption of safer alternatives is critical to the success of these bans. Great Lakes Chemical, a major producer of brominated chemicals, is voluntarily phasing out the production of penta- and octa-BDE by the end of 2004 [21]. This voluntary recall was made possible in part due to the development of an alternative product, Firemaster® 550. EPA recently completed a preliminary assessment of Firemaster® 550 and concluded that this alternative chemical is not persistent, bioaccumulative or toxic to aquatic organisms [22].

Great Lakes Chemical is the only manufacturer of penta-BDE in the U.S., and EPA is working with the remaining suppliers of octa-BDE to nationally phase out octa-BDE as well. However, there are no plans to phase out deca-BDE, which accounts for the largest percentage of PBDEs produced. While manufactures argue deca-BDE does not bioaccumulate and is not toxic, recent scientific evidence contradicts these claims [3]. Not only does deca-BDE break down in sunlight to lower weight congeners [21], it has been shown to result in behavioral disturbances after being fully absorbed by mice [2]. An interesting study would be to compare PBDE congener levels in California and Maine after the bans are imposed to determine the effect of deca-BDE.

Conclusion

PBDE levels in humans throughout the world have been increasing for several decades. Despite the lack of toxicological and epidemiological data, steps have been taken to monitor levels and ban production in Europe. Similar monitoring programs for PBDEs in breast milk should be implemented in North America as well. PBDEs, like PCBs, are believed to affect the neurodevelopment of fetuses. Further research is needed to assess the toxicological endpoints associated with PBDEs and determine the routes of exposure.

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