Institute of Occupational Medicine
consulting

Review of the potential human exposure to decaBDE and the associated risks to health

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Introduction

Decabromodiphenyl ether (decaBDE) is the fully brominated end member of a class of molecules in which two aromatic rings are linked by a single oxygen atom. It is a synthetic compound that is not known to occur naturally. Other types of brominated diphenyl ethers have been found in wildlife in remote areas of the world and this has given rise to concern about the persistence of this class of compounds within the environment. This report describes current levels of human exposure to decaBDE, summarises what is known about its toxicity and assesses the potential risks to human health associated with current levels of exposure of the general population and those who come into contact with it at work.

The information presented in this report is based on that published by other reviewers, a reappraisal of the studies that were previously reviewed and fresh information from more recently published investigations. A fully referenced technical report is available.

Description

DecaBDE is a solid of low volatility that is virtually insoluble in water and aqueous media and has a limited solubility in organic solvents. It is used as a fire retardant in polymers used in electrical equipment and in upholstery textiles including the rubber backing of some carpets.

The molecular structure of decaBDE is distinct from that of some other halogenated aromatic hydrocarbons, most notably that of dioxin. In dioxins, the two aromatic rings are in the same plane (coplanar) whereas those of decaBDE are at right angles. This means that the interactions of decaBDE with molecular receptors in the body will be very different from those of dioxins and coplanar PCBs or PBBs, and that it is highly unlikely that decaBDE will have similar toxicological properties these compounds.

Human exposure

DecaBDE is a widely used fire retardant that is likely to be present in small quantities throughout the urban environment, but has a low potential for long-range transport. It has been found in tiny quantities in air (about 0.00001 ugm^{-3}, compared with typical concentrations of airborne particles[1] of 15-30 ugm^{-3} in urban areas). DecaBDE is also present in variable quantities in sediments with the highest concentrations arising downstream of wastewater treatment works and plants where decaBDE is produced or used. In the built environment, decaBDE has been reported in dust in normal houses and offices. A small proportion of the decaBDE found in indoor environments may have been emitted from electrical equipment, but the major source is likely to be dust generated from decaBDE-treated textiles and carpet backing. DecaBDE does not accumulate in wildlife or food and unlike other persistent organic chemicals has not been reported to occur in wildlife or sediments from remote areas of the world.

[1] Measured as PM_{10} – the fraction of airborne particulate that penetrates to the lungs

The main sources of exposure of the general population to decaBDE are likely to be inhaled air and through contact with dust at home or in the office. The efficiency of absorption of decaBDE following inhalation has not been investigated but a large proportion of inhaled particles containing decaBDE are likely to be ultimately swallowed and only a small proportion of the decaBDE in these particles is likely to be absorbed. The absorption of decaBDE from dust in contact with the skin is likely to be negligible. A small proportion of very small children may be exposed to decaBDE in dust as a result of inadvertent ingestion of dirt while putting other non-food items in their mouths or even purposefully eating dirt. Such behaviour is likely to be temporary and would not give rise to a substantial lifetime intake. Some adults may also inadvertently ingest decaBDE in dust through hand-mouth contact and/or eating, smoking or drinking with dusty hands. The absorption of decaBDE from ingested dust is likely to be relatively small (about 1% of the ingested dose). Food and water are not major sources of exposure to decaBDE. In contrast, food is likely to be the main source of exposure to other BDEs for the general population.

Retention within the body

The results of animal experiments suggest that most ingested decaBDE is rapidly eliminated in the faeces. Both animal and human data suggest that decaBDE is largely removed within a few days of exposure and that there is little long-term retention of decaBDE in tissue.

Trace quantities of decaBDE have been found in samples of human blood, breast milk and adipose tissue. The highest concentrations of decaBDE in blood (<0.3-9.9 ng per gram of lipid in serum or less than 0.000001% of the fat content of serum) have been reported in workers recycling electrical goods in Sweden. In comparison, the serum of hospital cleaners contained between <0.3 and 3.9 ng/ gram lipid. DecaBDE accounted for about 20% of the measured BDEs in recycling workers but less than 10% of the BDEs in the hospital workers. The recycling workers were believed to have been exposed to decaBDE in air as a result of emissions arising from the recycling of electrical equipment containing these compounds. The samples from the hospital cleaners may be more representative of general population and show that people are exposed to small amounts of decaBDE, even in the absence of a specific source of exposure at work. The quantities of decaBDE found in human samples are very small compared with the quantities found of other persistent pollutants such as polychlorinated biphenyls (PCBs).

Toxicity in animal experiments

Most of the published investigations of the toxicology of decaBDE in animals were undertaken in the 1970s and 1980s and the data reported does not include blood or tissue concentrations of decaBDE that would allow ready comparison with the limited biological data available for humans.

Animal feeding experiments undertaken in the 1970s with a commercial mixture of BDEs that contained about 77% decaBDE found a no effects level of 8 mg/kg body weight/day (equivalent to a daily dose of 560 mg for a 70 kg human) and a lowest observed effects level of 80 mg/kg body weight/day for adverse effects on the thyroid and liver. It is unclear whether these effects on the liver and thyroid were caused by decaBDE and other experimental evidence suggests that they are likely to have arisen largely or wholly as a result of exposure to more toxic lower brominated BDEs in the received dose. Lower brominated BDEs have greater structural similarities to thyroid hormones than decaBDE and are therefore more likely to interfere with thyroid function. Although it is possible that some metabolites of

DecaBDE may also resemble thyroid hormones, its potential to disrupt thyroid function or induce liver enzymes is much less that that of less halogenated aromatic compounds.

In later experiments undertaken during the 1980s with a much purer formulation of decaBDE, rats exposed to extremely high doses for two years (>1000 mg/kg/day, equivalent to more than 70 g/day for a 70 kg human) in their diet developed neoplastic nodules of the liver, but there was no significant difference in cancer numbers between the treated and untreated groups. There was a high incidence of leukaemia in both treated and untreated animals.

Investigations of the effects of decaBDE (77% pure) on reproduction and the unborn foetus found no adverse effects associated with maternal exposures to less than 100 mg/kg/day in feeding experiments, equivalent to a human dose of 7 g/day. At even more extreme levels of maternal exposure (1000 mg/kg/day), the formation of the skull was delayed in the young who also developed fluid retention under the skin. Given the low purity of the preparation used in these experiments, it is likely that the observed effects were largely due to exposure to lower brominated BDEs in the administered dose. A more recent experiment with 97% pure formulation of decaBDE found no adverse effects on the development of rats associated with maternal exposures to 1000 mg/kg/day.

In a separate recent feeding experiment in newly born mice, adverse effects on the developing brain have been reported at lower levels of exposure than associated with other toxic endpoints. The investigation, however, used an experimental design and species that are not typically used to evaluate neurotoxic risk to humans, so the significance of the findings is uncertain. The level of exposure associated with adverse effects was several orders of magnitude larger than those relevant to human babies, being equivalent to that associated with the consumption of well over a kilogram of house dust. Mice exposed to 2.2 mg/kg body weight on their 3rd day of life developed behavioural defects that were reported to worsen with age. The adverse effects of decaBDE on the developing brain appeared to be confined to a short period within the development of the mouse brain when the brain was undergoing a post-natal spurt in growth. Exposure during the later stages of brain development did not have the same adverse effects. The timing of the brain growth spurt in humans is somewhat different from that in mice and the relevant period of exposure could occur during the final months of pregnancy and throughout the first two years of life. The transfer of decaBDE from mother to unborn child is expected to be extremely small. Even, if the transfer of decaBDE from mother to child was 100% efficient, however, it has been calculated that a mother would have to receive an exposure of between 50 and 1000 mg/ day for the child to receive a level of exposure similar to that associated with the reported effects in mice. This is more than 100000 times the level of exposure to decaBDE for a typical adult. There are no other reports of neurotoxicity associated with exposure to decaBDE and further investigations to confirm the findings of this study in other species could be valuable.

Effects in humans

There is virtually no information about the effects of decaBDE in humans. Prior to the late 1990s decaBDE was regarded as an inert, nontoxic dust and workplace exposure to decaBDE was controlled to the same levels as for other inert, nontoxic particulate materials. It is probable that historically some workers involved in the production of decaBDE were routinely exposed to mean concentrations in air of the order of 1-5 mgm^{-3} over a typical 8 hour shift. There is only one report of possible adverse effects that may be attributable to exposure to decaBDE. Hypothyroidism was found in some of a small number of workers at a single plant. These workers had been exposed to both decaBDE and polybrominated biphenyls (PBBs). No detailed exposure information is available and the report describing the study provides no information about the workplace in which exposure occurred. The limited information available from animal experiments suggests that PBBs are likely to have a greater potential to disrupt thyroid function than decaBDE. It is probable, therefore, that the observed effects were due to exposure to PBBs rather than decaBDE or possibly both types of flame retardant contributed to the observed effects. It is also possible that the apparent excess of hypothyroidism had arisen by chance. Given that decaBDE was produced or used at a large number of other sites, the absence of other reports of ill health, strongly suggests an absence of effects. It is possible, but unlikely, that exposure to decaBDE could give rise to some metabolites that are sufficiently similar to a key thyroid hormone to cause receptors in the body to over-regulate the concentration of the true hormone. Any such effects would, however, be much more likely to occur with less extensively brominated BDEs or PBBs than with decaBDE.

Assessment of potential risk to human health

The highest potential levels of exposure of the general population to decaBDE are tiny in comparison to the oral reference dose calculated by the US Environmental Protection Agency (EPA). The EPA is normally highly protective in its approach to chemical regulation. In the case of decaBDE, the RfD cited by the EPA is likely to be unreasonably low as it is based on the findings of experiments in which the administered dose was only 77% pure. The observed effects in these experiments are likely to have been largely due to exposure to lower brominated BDEs. Levels of exposure to decaBDE are even smaller in comparison to the no effects level observed in animals. There is no evidence that decaBDE accumulates to any degree within the human body and no reason to expect that current levels of exposure of the general population to decaBDE are likely to be hazardous to health.

The members of the general population who are likely to have the greatest exposures to decaBDE, both in terms of their intake relative to body mass and also because of potential exposure from mouth contact with non-food items are young children between 6 months and 2 years in age. Even allowing for extensive mouth contact with non-food items, however, mean exposures of this age group are highly unlikely to exceed 0.1% of the lowest observed effects level in animals. The transfer of decaBDE from mothers to unborn or breastfed babies is likely to be extremely small. The levels of exposure of the unborn child and newly born child (less than 6 months old) of mothers without specific occupational exposure to decaBDE, are unlikely to exceed 0.001% of the lowest observed effects level. For mothers with relatively high levels of workplace exposure to decaBDE, published estimates of the levels of exposure of a breastfed child to decaBDE are less than 1% of the dose reported to give rise to possible neurotoxic effects in mice. This level of exposure is in turn much less than 1% of the better established lowest effects levels reported for other health endpoints in experiments that have used relatively high purity preparations of decaBDE.

The levels of exposure to decaBDE of workers involved in recycling electrical equipment are tiny in comparison to the no effects level observed in animals. Levels of exposure are also likely to be very much smaller than those to which workers involved in the production of decaBDE were exposed to in the past, without apparently developing adverse health effects. Even if the effects reported from a single study of workers exposed to both decaBDE and PBBs are partly or wholly attributable to decaBDE, levels of exposure are likely to have been many times higher than for present day recycling workers. There is no evidence that decaBDE accumulates to any degree within the human body and no reason to expect that current levels of exposure of recycling workers to decaBDE are likely to be hazardous to health.

Conclusions

Current levels of environmental or workplace exposure to decaBDE, including exposure in food, are extremely small. The risk of adverse health effects arising from these exposures is negligible and can be further reduced in the workplace by the use of standard occupational hygiene measures. The presence of relatively elevated concentrations of decaBDE in the blood of workers involved in recycling electronics does not imply an associated risk to health.