RISK ASSESSMENT OF HEXABROMOCYCLODODECANE (HBCD)

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Introduction

Brominated flame retardants (BFRs) represent a major industry, involving high-production chemicals with a wide variety of uses, mainly because of their low cost and high performance efficiency. Hexabromocyclododecane (HBCD; CAS No. 25637-99-4) is one of a number of polybrominated flame retardants (PBFRs) used mostly in the expandable and extruded polystyrenes (EPS and XPS) in domestic and industrial building insulation, packaging of industrial products and beanbag fills. It is also used as a textile coating additive in blinds, public seating and garments. HBCD is an additive flame retardant, meaning that it is incorporated into the polymer matrix but does not chemically bind to it

In the last 10-15 years HBCD has attracted attention as a chemical of concern at a regional and global level by national authorities and international bodies such as the European Union (EU) and the United Nations (UN). Several environmental monitoring programs conducted primarily in Europe and North America revealed that increasing levels of HBCD were detected in air, sewage sludge, sediment, fish, aquatic birds, marine mammals and other wildlife. HBCD was also found to be highly toxic to aquatic organisms.

Currently there are no restrictions on the manufacture, import and use of HBCD in Australia. There are no environmental restrictions on the use of HBCD in any State or Territory in Australia. It is not listed in Safe Work Australia's Hazardous Substances Information System (HSIS), the Standard for Uniform Scheduling of Medicines and Poisons (Australian Government, 2010) or on the Australian National Pollutant Inventory.

In 2001 Australia conducted a preliminary assessment of HBCD along with other PBFRs (NICNAS, 2001). The assessment focused on use patterns and potential exposure to PBFRs in Australia. Recommendations included a full assessment of HBCD, focusing on risk to workers handling HBCD, the general public and the environment, and for industry to carefully consider the selection of PBFR compounds for use and ensure that those known to be hazardous are avoided (NICNAS, 2001). A survey conducted by NICNAS in October 2004 on the extent of use of brominated flame retardants indicated a significant increase in the use of HBCD in Australia compared to the period 1998–99. A comprehensive assessment of risks posed by HBCD to human health and the environment was conducted.

The objectives of this assessment were to:

- identify the extent of use of HBCD in Australia
- assess the health and environmental hazards associated with HBCD
- estimate the potential public, occupational and environmental exposure to HBCD in Australia
- evaluate the potential risk of adverse effects to the general public, workers and the environment
- make recommendations to minimise public and occupational health risks and environmental risks.

Human and environmental health effects and exposure to HBCD were assessed to determine risk posed by the import/manufacture, use, storage, handling and disposal of HBCD.

Materials and Methods

Information for the assessment was obtained from manufacturers and importers (introducers) of HBCD and HBCD-products, from published literature sources, the EU Risk Assessment Report on HBCD (EU RAR, 2008), and the International Uniform Chemical Information Database (IUCLID) for HBCD. Various international

databases were accessed to obtain toxicological and exposure information on HBCD and a comprehensive search of internet sources was undertaken. This paper discusses the characterisation of risk to human health and the environment from use of HBCD and HBCD products in Australia.

Results and Discussion

Manufacture and importation

HBCD is not manufactured in Australia. It is imported into Australia as liquid dispersions, in EPS and XPS resins and as a component of plastic in finished articles, such as office equipment, projectors, scanners and ventilation units for offices. Assessment of import data indicated that there was a decrease in the import of HBCD over the years; with 91 tonnes imported in 2005–06 to approximately 60.5 tonnes in 2009–10. In addition, technical grade HBCD (powder or granules) has not been imported into Australia since 2010.

Human health effects of HBCD

Limited data are available on the toxicokinetics and metabolism of HBCD. Animal studies indicated that HBCD is rapidly absorbed from the gastrointestinal tract and widely distributed in body organs and tissues. Excretion in animals was also rapid, with majority of HBCD being excreted in the faeces as metabolites within 72 h.

Acute exposure to HBCD did not show any effects, however, repeated exposure revealed effects on the liver, such as dose-dependent increases in liver weights with a no observed adverse effect level (NOAEL) of 10 mg/kg bw. Absence of histopathological changes in the liver indicated liver enzyme induction as a cause for increase in liver weight. This was considered an adverse effect as it was significantly higher in treated animals when compared to controls.

Effects on pituitary and thyroid hormone system were also noted which provided further evidence for enzyme induction in the liver, leading to increased excretion of thyroid hormone and stimulation, by feedback mechanism, of pituitary and thyroid. HBCD was not genotoxic in any of the animal tests and did not show carcinogenic properties in a chronic study in mice.

HBCD did not show any marked adverse effects on fertility parameters in laboratory animals, however, developmental effects in rats were seen in two studies. In a one-generation study, bodyweights of F1 pups were decreased in a dose-dependent manner. The time to vaginal opening was delayed in female pups at the top dose. Testes weights were decreased at low doses, with a significant dose-response relationship. At high doses of HBCD decreased organ weights were noted in F1 animals. All F1 pups also showed marked dose-dependent decreases in liver apolar retinoid levels.

In a 2-generation reproductive toxicity study, a dose-dependent decrease in body and organ weights and increase in pup mortality during lactation was observed in F2 generation rats in the absence of clear clinical signs of toxicity in the dams. The development of basic reflexes was also affected at the highest dose, leading to shorter time response in the surface righting reflex in F1 male pups on PND5.

Based on low bodyweights of pups and high mortality during lactation at the mid and high dose in the 2-generation reproductive study, HBCD was considered to be a developmentally toxic chemical. A No Observed Adverse Effect Level (NOAEL) of 10.2 mg/kg bw/d was established for developmental toxicity of HBCD. HBCD was considered to have the potential to cause harm when transferred through lactation.

Environmental effect

HBCD is very toxic to aquatic organisms with an EC_{50} of10.5 μg/L in the marine diatom *S. costatum*, The lowest chronic toxicity indices were for *Daphnia magna*, with a NOEC of 3.1 μg/L and a maximum acceptable toxic concentration (MATC) of 4.2 μg/L.

For terrestrial ecotoxicity, test results for plants (seedling emergence study only) showed no effects due to HBCD exposure up to a measured soil level of 6200 mg/kg. The earthworm reproduction study produced an EC_{50} of 771 mg/kg soil. A NOEC of 128 mg/kg was also established in this study, even though a 15% inhibition

of reproduction (compared with the controls) was still observed at the lowest tested mean measured concentration of 51.5 mg/kg. An extrapolated EC_{10} of 21.6 mg/kg was calculated for this study.

Risk Characterisation

Determining the risk posed by HBCD based on developmental studies would result in a risk estimate relevant for a small section of the population (females of child-bearing age). Thus an adverse health effects observed in both male and female animals will give a better estimate of risk to the general population. The other pronounced effect of HBCD in animal studies was increase in liver weight in both male and female animals. The NOAEL for this effect in a reliable and well-conducted 28 d oral study was 10 mg/kg bw/d, which is very similar to the NOAEL from the reproductive study. Therefore the risk calculated using this NOAEL would also cover risk from repeated exposure in the occupational use situation as well as to the general population, especially since HBCD is known to be persistent.

Public health risk

The main source of exposure to HBCD by the general public is from contact with consumer articles treated with HBCD. The HBCD may leach from the article resulting in exposure via the dermal route. .A potential source of dermal exposure is automotive upholstery treated with HBCD. Estimates of dermal exposure from this source, however, indicated very low exposure and therefore low risk to adults as well as children.

Indirect exposure to HBCD through the environment may occur by consumption of food and drinking water contaminated with HBCD and by inhalation of indoor and outdoor dust. Indoor dust may contain HBCD released from HBCD-containing articles in the house. International data of concentrations of HBCD in household dust showed great variability. Estimation of exposure via this route showed that toddlers have the highest exposure; however, the risk of developing adverse health effects was quite low.

On the basis of available information, exposure to HBCD from dietary sources also appeared to be very low. Oral exposure of infants to HBCD was estimated using HBCD levels in human breast milk reported in a UK study. The risk to infants was estimated to be low through exposure to HBCD in breast milk.

Occupational health risk

The extent of occupational exposure to HBCD depends on the form of HBCD used – powder or granular forms or aqueous solution – the nature of the work and the different use patterns. Exposure to workers handling HBCD was estimated based on exposures during importation and packaging, in the polymer industry, in the textile industry and while handling HBCD-containing products/articles.

Exposure to HBCD from both dermal and inhalation exposure routes was determined. The calculation of the internal dose assumed dermal absorption values of 2% for granules and 4% for powder and liquid formulations and 100% absorption from respiratory tract.

Workers in the polymer industry may be exposed to HBCD during weighing, compounding, conversion or moulding activities. The risk of acute adverse health effects such as inhalation toxicity, skin, eye and respiratory irritation and skin sensitisation was low. However, the risk of harmful effects from long term exposure to HBCD powder and granules during these processing was estimated to be high. Risk to workers in the textile industry was unacceptable when treating textiles with HBCD products.

Risk to workers handling semi-finished or end-use products, such as cutting, sawing and machining polystyrene foam products is low, as these products contain HBCD at very low concentrations and the HBCD is either incorporated into a plastic matrix or fixed onto fibres.

Environmental risk

The majority of HBCD (>95%) is used to produce flame retardant EPS resins and the environment is unlikely to be directly exposed, except during disposal of the resins. As a result of product manufacturing and textile

treatment, the amount of HBCD released to air, water and solid waste is estimated to be 242, 609 and 44 kg, respectively, per annum.

HBCD is very bioaccumulative, with a bioconcentration factor of 13 000 in fish. Its levels in biota indicate that the substance bioaccumulates and biomagnifies through the food chain. Monitoring data from sediments in the environment show a wide range of HBCD levels and do not indicate degradation of HBCD, suggesting persistence in the environment. Detection of HBCD in biota and abiotic samples in remote regions provides additional evidence that HBCD is persistent in the environment.

A risk quotient method that compares toxicity to environmental exposure was used to estimate risk. Using derived predicted 'no effect concentrations (PNECs)' for water, sediment and soil, and comparing these to exposure estimates for different exposure scenarios, predicted environmental concentration (PEC)/PNEC ratios of >1 were identified for a number of scenarios. For surface water and sediment, this occurred when there were local releases from industries manufacturing end-use products with resins containing HBCD, or where HBCD is used in manufacturing end-use textiles. For the terrestrial compartment, the PEC/PNEC ratios were >1 when there were local releases (both processing and end-use product manufacturing operations) from agricultural soils treated with biosolids, or from soils that are irrigated using effluent from treatment plants. The risk to aquatic species arising from use of HBCD in plastic or textile industry is low, as indicated by risk quotients (RQ) of <1. However, the sediment RQ for most use scenarios of HBCD indicated that HBCD concentrations in the A potential local risk was also determined for terrestrial organisms from levels in soils amended with biosolids.

Conclusions and recommendations

The assessment indicated significant health risk to workers handling HBCD. It is also very toxic to aquatic organisms. There is also sufficient evidence that HBCD is persistent and bioaccumulative in the environment.

There are currently no measures in place in Australia to protect the environment or workers. To mitigate these risks, NICNAS has recommended that:

- importers of the HBCD chemical and manufacturers and importers of HBCD containing products and articles, move to alternatives in applications where safer options and technologies are commercially available;
- the Standing Council on Environment and Water develop an Action Plan to address the currently unacceptable risk of HBCD levels in the Australian environment, taking into account the NICNAS risk assessment, the assessment conducted by an international multilateral body and practical issues for industry; and
- SafeWork Australia include the appropriate health hazard classification in its Hazardous Substances Information System. The classification provides information on the hazards of HBCD to workers.

References

Australian Government (2010) Standard for the uniform scheduling of medicines and poisons, No.1,1 September 2010. In: Poisons Standard 2010, published by the Australian Government under the *Therapeutic Goods Act 1989*. At http://www.comlaw.gov.au/Details/F2010L02386

NICNAS (2001) Polybrominated flame retardants (PBFRs): Priority Existing Chemical Assessment Report No. 20. National Industrial Chemicals Notification and Assessment Scheme, Sydney.