



Australian Government

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**Department of Health and Ageing**  
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The *Industrial Chemicals (Notification and Assessment) Act 1989* (the Act) commenced on 17 July 1990. As required by Section 5 of the Act, a Chemical Gazette is published on the first Tuesday in any month or on any days prescribed by the regulations.

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## 1 PUBLIC RELEASE OF DRAFT PRIORITY EXISTING CHEMICAL REPORT FOR HEXABROMOCYCLODODECANE (HBCD)

In accordance with section 60E(1) of the *Industrial Chemicals (Notification and Assessment) Act 1989* (the Act), as amended, notice is hereby given by the Director that the draft Priority Existing Chemical (PEC) assessment report on Hexabromocyclododecane (HBCD) is now available for public comment.

Under Section 60D of the Act, the draft PEC report was given to applicants for 28 days to enable corrections of any errors. The draft report has been corrected and is now available for public comment. The report focuses on assessment of risks to workers handling HBCD or its products, to the general public coming in contact with articles containing HBCD and to the environment.

Recommendations are made to reduce the risks identified for workers and for the environment.

The draft report (hard or read-only electronic copy) can be obtained by contacting Ms Julie Brown via phone **(02) 8577 8870** or fax **(02) 8577 8888** or e-mail to [julie.brown@nicnas.gov.au](mailto:julie.brown@nicnas.gov.au).

The draft report is also available on the NICNAS website at <http://www.nicnas.gov.au/consultations.asp>.

Under Section 60E(2) of the Act, any requests to vary the draft report should be received in writing to NICNAS by close of business (5:30 pm) on **22 December 2011**. This is a statutory deadline, which cannot be extended.

### Submission format for variation requests

Any requests to vary the draft report must be made with respect to the draft report and be accompanied by a completed application form (NICNAS Form 4a) which is available on the NICNAS website at [http://www.nicnas.gov.au/Forms/Existing\\_Chemicals/Form4a\\_PDF.pdf](http://www.nicnas.gov.au/Forms/Existing_Chemicals/Form4a_PDF.pdf).

Applications should clearly outline any amendment or change(s) requested. All applications for variation must identify the exact words, sentence or paragraph in the report to be varied and then state replacement words, sentences or paragraphs. The rationale behind any request for variation must be clearly explained, with references where relevant.

Requests for variation should be sent to: NICNAS, GPO Box 58, Sydney NSW 2001.

### Briefings

Briefings on the findings and recommendations of the report will be held in Sydney and Melbourne, on 6, 7 or 9 December, depending on the level of interest. Please indicate your interest in attending the briefing and preference of city on the attached sheet by **29 November 2011** via e-mail or fax to NICNAS.



## 2 OVERVIEW FOR DRAFT HBCD REPORT

### Overview

#### Background

Hexabromocyclododecane (HBCD) CAS No. 25637-99-4 was declared a Priority Existing Chemical (PEC) for a full risk assessment under the *Industrial Chemicals (Notification and Assessment) Act 1989* (the Act) by notice in the Commonwealth *Chemical Gazette* of 7 June 2005.

HBCD is one of a number of polybrominated flame retardants (PBFs). In 2001, a preliminary PEC assessment of PBFs as a group was conducted by NICNAS, as there was concern over the widespread use of flame retardants in household and industrial situations, and a report focusing on occupational, public health and environmental exposure to PBFs was published. The report recommended that a full risk assessment be conducted once testing of these chemicals was completed internationally.

A follow-up survey conducted by NICNAS in October 2004 indicated a significant increase in the use of HBCD in Australia compared to the period 1998–99. It was determined that a full risk assessment of HBCD as a PEC would allow assessment of its potential occupational, public health and environmental risks and formulation of appropriate recommendations for the safe use of the chemical.

#### Manufacture and importation

HBCD is not manufactured in Australia. It is imported into Australia as raw or technical grade powder or granules, in expandable polystyrene (EPS) resin, as liquid dispersions and as a component of the plastic in finished articles. Finished articles containing HBCD include extruded polystyrene (XPS) boards, office equipment such as inkjet printers, projectors, scanners and ventilation units for offices. The assessment showed that there was a decrease in the import of HBCD over the years, with 91 tonnes imported in 2005–06 and approximately 51.5 tonnes in 2009–10. It was reported in 2010 that the powder form of HBCD is no longer being imported into Australia.

#### Uses

HBCD is an additive flame retardant, meaning that it is incorporated into the polymer matrix but does not chemically bind to it. HBCD is used in the EPS resin form in domestic and industrial building insulation, packaging of industrial products and beanbag fills. HBCD is also used in XPS boards in domestic and industrial insulation and in automotive. Other uses are as a polypropylene resin in housing for domestic electrical appliances and as a textile coating additive in blinds, public seating and garments. A small amount of raw HBCD is used in the manufacture of flame retarded polystyrene masterbatch, which is used in an injection moulding process in the manufacture of ceiling fan covers.

## Health effects

Limited data are available on the toxicokinetics and metabolism of HBCD. Studies in rats indicate that HBCD is rapidly absorbed from the gastrointestinal tract with a half-life of approximately 2 h. HBCD is widely distributed in the body, with the highest concentrations found in adipose tissue and muscles, followed by the liver, lung, kidney, blood and brain. In animal experiments, excretion was also rapid, with majority of the radiolabelled HBCD excreted in the faeces as metabolites or non-extractable radioactivity within 72 h. Only a small percentage was detected in the urine. Three metabolites were extracted from the faeces of rats following administration of radiolabelled HBCD; however, the identity of the metabolites was not known.

HBCD has low acute oral, dermal and inhalation toxicity. It is not an eye or skin irritant in animals. It is not a skin sensitiser in animals or humans. There are no data available on the potential for respiratory sensitisation.

Repeat dose toxicity studies consistently revealed dose-dependent increases in liver weights in 28 d and 90 d studies in both sexes; in a 90 d study, up to a 48% increase in liver weight in female rats was reported at the highest dose. The liver weight changes were reported to be reversible except at the highest dose tested. Histopathological changes were minimal and only reported in the 90 d studies. The increase in liver weight was likely to be due to enzyme induction and an adaptation response; however, the magnitude of increase and its persistence after recovery period indicate that the increase in liver weight could be considered an adverse effect.

Thyroid effects were also noted in female rats. Relative thyroid weights were increased by 33% in mid- and high-dose females. There was minimal thyroid follicular cell hypertrophy. Serum concentrations of thyroid hormone (T4) were dose-dependently decreased at all doses in males and females. Increased pituitary weights were also noted in female rats. These changes in liver, thyroid hormone and pituitary could possibly be explained by enzyme induction in the liver, leading to increased excretion of thyroid hormone and stimulation, by feedback mechanism, of pituitary and thyroid. T4-uridine-diphosphate glucuronyl transferase in the liver has been shown to be induced in rats (both sexes) treated with HBCD.

Based on increase in liver weights, a no observable adverse effect level (NOAEL) of 10 mg/kg bw/d was established for repeated dose toxicity.

HBCD was not genotoxic in any of the animal tests and did not show carcinogenic properties in a chronic study in mice.

In reproductive toxicity studies in rats, HBCD did not show any marked adverse effects on fertility parameters. In a 2-generation reproductive study, no significant changes were noted in copulation index, gestation index or the number of implantations. Reduced primordial follicles were noted in mid- and high-dose F1 females; however, they were within the range of historical control values from 4 earlier studies performed in the same laboratory that performed the current study. Moreover, the number of primordial follicles were the same at the mid and high doses, with no dose response relationship. There were also large variations within the groups. No significant difference was seen between control and HBCD-treated groups in the incidence of clinical signs of toxicity in either male or female F0 and F1 rats during the test period.

The 28 d repeat-dose study reported inhibited oogenesis and reduced number of follicles, with sparse ripening follicles in the ovaries at the highest dose of 4700 mg/kg bw/d. No effects were noted in male rats exposed to the same high dose, except for small inner sexual organs. Prostate weights were increased in a 90 d oral study in rats. The effects seen in male and female rats are considered to be mediated through the endocrine system.

A clear NOAEL for effects on fertility could not be established in any of the reproductive studies. However, based on the reduced fertility index (not statistically significant) and reduced primordial follicles in the F1 generation rats in the 2-generation reproductive study, a NOAEL of 10.2 mg/kg bw/d was considered.

Developmental effects in rats were seen in 2 reproductive toxicity studies. In a 1-generation study, bodyweights of F1 pups were decreased in a dose-dependent manner. The time to vaginal opening was delayed in the female pups at the top dose. Testes weights were decreased at low doses, with a significant dose-response relationship. Systemic effects at high doses of HBCD were decreased kidney and thymus weights in both sexes and decreased adrenals, heart, brain and prostate weights in F1 males. A decrease in bone mineral density in females and decreased total mineral content, total area and cortical thickness in males in the femur and tibial bones were also noted. Both male and female pups showed marked dose-dependent decreases in liver apolar retinoid levels.

In a 2-generation reproductive toxicity study, a dose-dependent increase in pup mortality during lactation was observed in the F2 generation (35% at the high dose and ~15% at the mid dose) in the absence of clear clinical signs of toxicity in the dams. Decrease in bodyweights of male F1 and F2 pups and female F2 pups were also noted at the high dose. In the F2 generation, the incidence of pups showing complete eye opening was lowered compared to controls. Delayed development in F2 pups was indicated by the reduced incidence of male and female pups showing eye opening on postnatal day (PND) 14. 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 PND 5 at the higher dose level. Other effects noted in F2 weanlings were decreased weights of kidney, brain, spleen, adrenal, epididymis, ventral prostate and ovary at mid and high doses.

There were also indications of developmental neurotoxicity in a study in adult mice that were exposed to HBCD as pups. However, this study was not performed according to OECD guidelines.

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 developmental toxin. A NOAEL of 10.2 mg/kg bw/d was established for developmental toxicity of HBCD. HBCD also has the potential to cause harm when transferred through lactation.

Selecting a NOAEL for risk characterisation from developmental studies would estimate risk only for a small section of the population (females of child-bearing age) and thus a NOAEL based on adverse effects observed in both male and female animals should also be used for estimating 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 exposure and health risk**

HBCD is used in consumer articles as an additive flame retardant. Consumers using the treated products may be exposed to HBCD that diffuses out of the articles. Exposure to HBCD from treated articles is expected to be mostly through the dermal route. Exposure through inhalation or ingestion of treated products is considered negligible.

A potential source of dermal exposure to HBCD 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 by HBCD and by inhalation of indoor and outdoor air. Exposure from inhalation was estimated to be very low, resulting in low risk of adverse effects by this route. On the basis of limited available information, exposure to HBCD from dietary sources appears to be very low.

Exposure to HBCD could also occur through ingestion or inhalation of dust/soil, especially in children. 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. Australian data on HBCD concentrations in breast milk are not available. 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 exposure and 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.

A NOAEL for exposure via the inhalation route was not available to determine the inhalation risk to workers. However, since HBCD administered orally was determined to be completely absorbed (100% absorption), the NOAEL from an oral study was used to estimate risk from inhalation of HBCD during occupational handling. For determining risk from dermal exposure, modelled dermal exposure data were converted to internal dose using the dermal absorption values for powder, granules and liquid formulations. Total exposure was determined as the sum of the internal dose estimated from dermal exposure values and dermal absorption rate and inhalation exposure assuming 100% absorption from respiratory tract. In the occupational exposure assessment the median and the 90th percentile values are used to represent the typical and reasonable worst-case estimates, respectively.

The risk of harmful effects by inhalation to workers handling the powder or granular formulations during importation and transport is likely to be negligible, except in the event of an accident or spill. Workers in the polymer industry may be exposed during weighing HBCD, compounding, conversion or moulding activities. Both the powder and the granular forms are used in compounding in Australia. The risk to workers of acute adverse health effects such as inhalation toxicity, skin, eye and respiratory irritation and skin sensitisation is low. However, the risk of chronic harmful effects from exposure during weighing of HBCD powder is high. Risk to workers is low during weighing of the granular form and compounding in the typical scenarios. However, in the realistic worst-case scenarios for all of these processes, margins of exposures were considerably lower than 100, indicating high risk to workers using powdered as well as granular HBCD.

Risk to workers handling semi-finished and end use 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. Workers handling polystyrene foam products containing HBCD during activities such as cutting, sawing and machining to manufacture shaped products can also be exposed to HBCD. However, the concentration of the chemicals in these products is very low, and the risk of adverse health effects from inhalation or dermal exposure is not expected to be of concern.

### **Environmental effects**

For many of the aquatic toxicity tests conducted, the EC<sub>50</sub>/LC<sub>50</sub> values could not be identified. Several tests measured endpoints in terms of total HBCD and, based on solubility, it appears that, for all trophic levels, the no observed effect concentration (NOEC) for HBCD in acute aquatic toxicity tests is at or around the measured total HBCD solubility of 3.4 µg/L.

The lowest EC<sub>50</sub> was for the marine diatom *S. costatum*, with a geometric mean EC<sub>50</sub> of 10.5 µg/L. 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. Based on these data, HBCD is very toxic to aquatic organisms.

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<sub>50</sub> 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<sub>10</sub> of 21.6 mg/kg was calculated for this study.

### **Environmental exposure**

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.

There is considerable uncertainty regarding the fate of HBCD in the environment. While laboratory data indicate that HBCD degrades faster under anaerobic conditions than aerobic conditions, the mechanisms for this are unclear. Monitoring data from sediments in the environment (where conditions are most likely to be anaerobic) show a wide range of HBCD levels and do not indicate anaerobic degradation of HBCD, suggesting persistence in the environment is much longer than that shown in laboratory studies. Further, the detections in biota and abiotic samples in remote regions provide additional evidence of the persistent nature of HBCD in the environment.

There is also uncertainty about the isomer composition of HBCD found in the environment. While the  $\gamma$  isomer is dominant in commercial formulations, the  $\alpha$  isomer is dominant in higher trophic level animals. This can be explained by bioisomerisation (formation of the  $\alpha$ -HBCD from  $\gamma$ -HBCD in certain animals) or there may be a preference for uptake of the  $\alpha$  isomer. Further, the  $\alpha$  isomer is approximately 20 times more soluble than the  $\gamma$  isomer, so, where HBCD is present in the water column,  $\alpha$ -HBCD would be expected to dominate.

HBCD is very bioaccumulative, with bioconcentration factors (total HBCD in whole fish of between 8800 and 13 000). In addition, HBCD levels in biota support a conclusion that the substance bioaccumulates and biomagnifies through the food chain.

Overall, the available evidence indicates that HBCD is persistent and very bioaccumulative in the environment.

### **Environmental risks**

Calculating 'safe' concentrations for compounds such as HBCD that are persistent in the environment, are bioaccumulative and also biomagnify in the food chain is difficult because potential adverse effects may not become evident for very long periods of time – much longer than can be captured by standard toxicity testing.

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  $>1$  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. Similarly, for the terrestrial compartment, this occurred 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 Australian sediments have the potential to cause adverse effects. A potential local risk is also determined for terrestrial organisms from levels in soils amended with biosolids. However, this risk subsided to acceptable level for soils irrigated with effluent for up to 10 years.

Calculation of a risk quotient in air is not possible; however, it must be considered that the presence of HBCD in the atmosphere warrants concern in light of strong evidence that the substance is persistent and has the potential to travel long distances.

Overall, as a result of certain exposure scenarios, there is sufficient evidence that HBCD is persistent and bioaccumulative in the environment. In addition, it is very toxic to aquatic organisms. HBCD is considered to meet the Persistent Organic Pollutants (POPs) criteria for persistence, bioaccumulation and toxicity listed under the Stockholm Convention on Persistent Organic Pollutants (the Stockholm Convention). Because of these characteristics, the risks posed by this chemical cannot be fully quantified using standard methodology, and a cautious risk management approach may be necessary.

## Recommendations

This section provides recommendations arising from the assessment of HBCD. Recommendations are directed principally at regulatory bodies and importers and formulators of HBCD and HBCD products. Implicit in these recommendations is that best practice is implemented to minimise occupational and public exposure and environmental impact.

HBCD is persistent, bioaccumulative and toxic, especially to aquatic organisms. Furthermore, it is not only considered to meet the POPs criteria as prescribed under the Stockholm Convention but also monitoring data for persistence, and laboratory and field data for bioaccumulation and bio-magnification, indicate that the chemical substantially exceeds the criteria. In addition, while the POPs criteria do not prescribe numerical values for toxicity, there are ecotoxicity data indicating the potential for damage to the environment. The predicted half-life in air is marginally greater than 2 days, and monitoring data show evidence of long-range atmospheric transport of HBCD.

In animal studies, HBCD showed adverse effects in repeat-dose and developmental toxicity studies, although effects in repeat dose and reproductive toxicity studies were not significant enough to warrant classification based on these effects. There may be health concerns based on the effects on liver and thyroid and indications of developmental and transient neurobehavioral effects. Exposure assessment indicates that there is potential for workers handling HBCD to be exposed to levels that will pose a risk of adverse health effects. Public exposure to HBCD in the environment resulting from release of HBCD into household dust is not of concern due to the estimated low-level exposure.

The assessment indicates that the greatest risk is to the environment and workers handling HBCD, and this needs to be managed.

## Occupational health and safety

### Recommendation 1 (to Safe Work Australia)

#### Classification

Based on the assessment of the available hazard data and in accordance with the *Approved criteria for classifying hazardous substances* (NOHSC, 2004), HBCD is determined to be hazardous and is classified as:

- R63 Possible risk of harm to the unborn child (Toxic to reproduction, Category 3).
- R64 May cause harm to breastfed babies.

It is recommended that this classification for HBCD be included in the Hazardous Substances Information System (HSIS) as soon as possible.

The appropriate risk phrases for mixtures containing HBCD are as follows:

<u>Risk Phrase</u>	<u>Concentration Cut-off</u>
R63	≥5%
R64	≥1%

The following safety phrases are also recommended for HBCD:

- S22 Do not breathe dust.
- S60 This material and its container must be disposed of as hazardous waste.

- S61 Avoid release to the environment. Refer to special instructions/Material Safety Data Sheets.

The classification of HBCD under the Globally Harmonised System (GHS) of Classification and Labelling is provided in Appendix 1 along with the signal words and hazard statements and is as follows:

#### Health Hazards

- Toxic to reproduction Category 2

#### Environmental Hazards

- Acute toxicity Category 1
- Chronic toxicity Category 1

### **Recommendation 2 (to industry): Hazard communication**

#### **MSDS and label amendments**

It is recommended that importers and employers take note of the hazard classification, and amend Material Safety Data Sheets (MSDS), labels and training material, paying particular attention to the following points:

- inclusion of the health hazards, risk and safety phrases as contained in Recommendation 1
- correct information on the concentration cut-offs for mixtures containing HBCD as provided in Recommendation 1.

### **Recommendation 3 (to industry)**

Based on the assessment findings, powdered or granulated HBCD should be handled under local exhaust ventilation. Workers should wear face masks, gloves and overalls to reduce exposure to HBCD.

### **Recommendation 4 (to industry)**

Manufacturers and importers of flame retardant articles should voluntarily phase out the import and use of HBCD chemical, and articles containing the chemical, as an interim measure to support the objectives of the Action Plan in Recommendation 6.

### **Recommendation 5 (to government): Compliance with State and Territory legislation**

It is recommended that the State and Territory occupational health and safety authorities review uptake of the new information in the MSDS and labels, and the safety measures recommended in this assessment.

## **Environmental safety**

### **Recommendation 6 (to the (to the Standing Council for Environment and Water (SCEW)**

It is recommended that the SCEW develop an Action Plan to reduce and eventually eliminate HBCD levels in the Australian environment, giving consideration to the fact that HBCD is currently under discussion for listing in the Stockholm Convention. The Action Plan should constitute a national approach involving federal, State and Territory agencies and should address the introduction of HBCD into Australia as a chemical entity in products and in articles. The Action Plan should include, but may not be limited to:

- a. measures to discontinue introduction of HBCD into Australia for further processing over a 5-year period
- b. measures to discontinue introduction of HBCD into Australia as part of finished articles
- c. monitoring import and release of HBCD over a 5-year period
- d. developing guidelines, in partnership with industry, for managing or disposing of existing stocks and articles containing HBCD
- e. coordinating with National Waste Policy to ensure that the plan addresses the issues posed by HBCD and disposal of articles containing HBCD
- f. evaluation of the effectiveness of the reduction program by analysing sediments for HBCD levels at regular intervals.