DECABROMODIPHENYL OXIDE

Data were last evaluated in IARC (1990).

1. Exposure Data

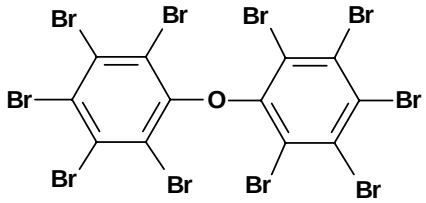
1.1 Chemical and physical data

1.1.1 *Nomenclature*

Chem. Abstr. Serv. Reg. No.: 1163-19-5

Chem. Abstr. Name: Benzene, 1,1′-oxybis[2,3,4,5,6-pentabromo]-

1.1.2 *Structural and molecular formulae and relative molecular mass*



$C_{12}OBr_{10}$ Relative molecular mass: 959.17

1.1.3 *Physical properties* (for details, see IARC, 1990)

(*a*) *Boiling point*: Decomposes at 425°C

(*b*) *Melting point*: 290–305°C

(*c*) *Conversion factor*: $mg/m^3 = 39.2 \times ppm$

1.2 Production, use and human exposure

Decabromodiphenyl oxide has been produced since the late 1970s as a flame retardant for use in plastics, especially high-impact polystyrene, and to treat textiles, such as automotive fabrics and tents. Occupational exposure to decabromodiphenyl oxide may occur during its production and use. It has also been detected in environmental samples collected near some production facilities (IARC, 1990).

2. Studies of Cancer in Humans

No data were available to the Working Group.

3. Studies of Cancer in Experimental Animals

Decabromodiphenyl oxide was tested for carcinogenicity by oral administration in one strain of mice and in two strains of rats. In one study in rats, it induced hepatocellular adenomas in animals of each sex and acinar-cell adenomas of the pancreas and mononuclear-cell leukaemia in males (IARC, 1990).

4. Other Data Relevant to an Evaluation of Carcinogenicity and its Mechanisms

4.1 Absorption, distribution, metabolism and excretion

4.1.1 *Humans*

No data were available to the Working Group.

4.1.2 *Experimental systems*

When labelled decabromodiphenyl oxide was given by gavage to rats, over 99% of the label appeared in the faeces, approximately 0.5% of mainly unchanged compound was found in the liver, less than 1% of the label was detected in the urine over 16 days and trace amounts of label were found in the kidneys, spleen, lungs, brain, muscle, fat and skin. After an intravenous dose, the faeces and gut contents contained 75% of the dose, suggesting significant biliary excretion, and of the extracted faecal label, 63% was metabolites. No evidence of induction of cytochrome c reductase or cytochrome P450 activities was seen in male rats. In a two-year dietary study in rats, the bromine content of liver and adipose tissue was slightly increased (IARC, 1990).

4.2 Toxic effects

No data were available to the Working Group.

4.3 Reproductive and developmental effects

No data were available to the Working Group.

4.4 Genetic and related effects

4.4.1 *Humans*

No data were available to the Working Group.

4.4.2 *Experimental systems* (see Table 1 for references)

In single studies, decabromodiphenyl oxide did not induce gene mutations in either bacteria or mouse lymphoma L5178Y cells and neither did it induce sister chromatid exchanges or chromosomal aberrations in Chinese hamster ovary CHO cells.

Table 1. Genetic and related effects of decabromodiphenyl oxide

Test system	Result[a]		Dose[b] (LED or HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	–	5000	US National Toxicology Program (1986)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	5000	US National Toxicology Program (1986)
SA7, *Salmonella typhimurium* TA1537, reverse mutation	–	–	5000	US National Toxicology Program (1986)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	5000	US National Toxicology Program (1986)
G5T, Gene mutation, mouse lymphoma L5178Y cells, *tk* locus *in vitro*	–	–	10	US National Toxicology Program (1986)
SIC, Sister chromatid exchange, Chinese hamster ovary CHO cells *in vitro*	–	–	500	US National Toxicology Program (1986)
CIC, Chromosomal aberrations, Chinese hamster ovary CHO cells *in vitro*	–	–	500	US National Toxicology Program (1986)

[a] –, negative
[b] LED, lowest effective dose; HID, highest ineffective dose; in-vitro tests, µg/mL

5. Evaluation

No epidemiological data relevant to the carcinogenicity of decabromodiphenyl oxide were available.

There is *limited evidence* in experimental animals for the carcinogenicity of decabromodiphenyl oxide.

Overall evaluation

Decabromodiphenyl oxide is *not classifiable as to its carcinogenicity to humans (Group 3)*.

6. References

IARC (1990) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, Vol. 48, *Some Flame Retardants and Textile Chemicals, and Exposures in the Textile Manufacturing Industry*, Lyon, pp. 73–84

United States National Toxicology Program (1986) *Toxicological and Carcinogenesis Studies of Decabromodiphenyl Oxide (CAS No. 1163-19-5) in F344/N Rats and B6C3F$_1$ Mice (Feeding Studies)* (Tech. Rep. Ser. No. 309), Research Triangle Park, NC, United States Department of Health and Human Services