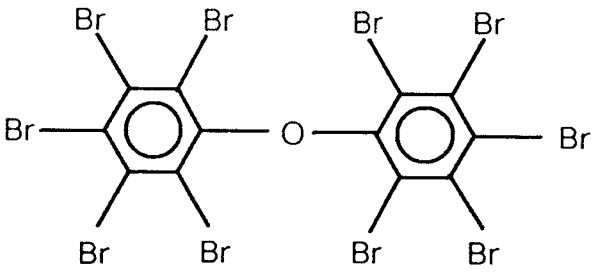
# DECABROMODIPHENYL OXIDE

## 1. Chemical and Physical Data

### 1.1 Synonyms

*Chem. Abstr. Services Reg. No.*: 1163-19-5
*Chem. Abstr. Name*: Benzene, 1,1'-oxybis[2,3,4,5,6-pentabromo]-
*IUPAC Systematic Name*: Bis(pentabromophenyl) ether
*Synonyms*: DBDPO; decabrom; decabromobiphenyl ether; decabromobiphenyl oxide; decabromodiphenyl ether; decabromophenyl ether; ether, bis(pentabromophenyl); pentabromophenyl ether

### 1.2 Structural and molecular formulae and molecular weight




$C_{12}Br_{10}O$ Mol. wt: 959.17

### 1.3 Chemical and physical properties of the pure substance

(*a*) *Description*: White powder (AmeriBrom, 1987; White Chemical Co., 1988)

(*b*) *Boiling-point*: Decomposes at 425°C (US Environmental Protection Agency, 1988)

(*c*) *Melting-point*: 290-305°C (AmeriBrom, 1987; White Chemical Co., 1988)

(*d*) *Spectroscopy data*: Infrared (prism [635D]; prism-FT [1057D]) and ultraviolet spectral data have been reported (Pouchert, 1981, 1985; National Toxicology Program, 1986).

(*e*) *Solubility*: Very slightly soluble (20-30 ppb [μg/l]) in water at 25°C; slightly soluble in acetone, benzene, dichloromethane, *ortho*-xylene, methanol, methyl ethyl ke-

tone, pentane and toluene (AmeriBrom, 1987; US Environmental Protection Agency, 1988; White Chemical Co., 1988; AmeriBrom, undated)

(*f*) *Volatility*: Vapour pressure, < 1 mm Hg at 250 °C (Tabor & Bergman, 1975; AmeriBrom, 1987)

(*g*) *Stability*: Stable under normal temperatures and pressures (White Chemical Co., 1988); thermal or light-catalysed decomposition of decabromodiphenyl oxide may release carbon monoxide, carbonyl bromide and hydrogen bromide, lower congeners of brominated diphenyl oxide (1-8 bromines/molecule) and polybrominated dibenzodioxins and dibenzofurans (1-6 bromines/molecule) (AmeriBrom, 1987; Watanabe & Tatsukawa, 1987; Aldrich Chemical Co., 1988; US Environmental Protection Agency, 1988; White Chemical Co., 1988)

(*h*) *Specific gravity*: 3.0 at 20 °C (AmeriBrom, 1987)

(*i*) *Octanol/water partition coefficient (P)*: log P, 5.24 (US Environmental Protection Agency, 1988)

### 1.4 Technical products and impurities

*Trade names*: AFR 1021; Berkflam B 10E; BR 55N; Bromkal 81; Bromkal 82-ODE; Bromkal 83-10DE; Caliban F/R-P 39P; Caliban F/R-P 44; DE 83; DE 83R; DP 10F; EB 10FP; EBR 700; Flame Cut BR 100; FR 300; FR 300BA; FR P-39; FR 1210; FRP 53; FR-PE; FR-PE(H); Planelon DB 100; Saytex 102; Saytex 102E; Tardex 100

Decabromodiphenyl oxide is available at purities usually greater than 97% (Tabor & Bergman, 1975; AmeriBrom, 1987; Aldrich Chemical Co., 1988; Ethyl Corp., 1988a,b; White Chemical Co., 1988). The commercial products typically contain a minimum of 81-83% bromine (83% theoretical; Tabor & Bergman, 1975; AmeriBrom, undated). Differences in manufacturing processes may affect the nature and amounts of impurities in the product (Larsen, 1980). Isomers of nonabromodiphenyl oxide and octabromodiphenyl oxide have been reported as impurities in decabromodiphenyl oxide (Timmons & Brown, 1988). Up to four impurities were reported in four lots of a commercial decabromodiphenyl oxide with purities ranging from 94% to 99%. Two isomers of nonabromodiphenyl oxide were identified as the major impurities (National Toxicology Program, 1986). A technical product of 88.1% purity containing 11% nonabromodiphenyl oxide, 0.5% octabromodiphenyl oxide and 0.1% hexabromobenzene is available (Klusmeier *et al.*, 1988). A company in Japan has produced decabromodiphenyl oxide with about 3% nonabromodiphenyl oxide as an impurity (Watanabe & Tatsukawa, 1987).

## 2. Production, Use, Occurrence and Analysis

### 2.1 Production and use

(*a*) *Production*

Decabromodiphenyl oxide is produced by bromination of diphenyl oxide in the presence of a Friedel-Crafts catalyst (Larsen, 1980). Decabromodiphenyl oxide has been re-

ported to be manufactured in a batch process involving closed vessels during the reaction and drying cycle (US Environmental Protection Agency, 1988).

Commercial production of decabromodiphenyl oxide in the USA began in 1976. The production quantity ranks second among brominated flame retardants, after tetrabromobisphenol A; the combined world-wide capacity is approximately 18 000 tonnes. Five manufacturers have been reported in Japan (Anon., 1984), three in the USA (US Environmental Protection Agency, 1988), two in Belgium and one each in Israel, Switzerland and the UK.

(*b*) *Use*

Decabromodiphenyl oxide is an unreactive, additive flame retardant widely used for its thermal stability and its low cost in thermoplastic resins, thermoset resins, textiles and adhesives. The major applications are in high-impact polystyrene, glass-reinforced thermoplastic polyester moulding resins, low-density polyethylene extrusion coatings, polypropylene (homo- and copolymers), acrylonitrile-butadiene-styrene rubber, nylon and polyvinyl chloride (Tabor & Bergman, 1975; AmeriBrom, undated).

Approximately 12 thousand tonnes of decabromodiphenyl oxide are used annually world-wide, about two-thirds in high-impact polystyrene applications such as television and radio cabinets. Textile applications, such as in polyester fibres and in coatings for automobile fabrics, tarpaulins and tents, account for a further 900 tonnes.

A mixture of decabromodiphenyl oxide and antimony trioxide has been used to treat nylon and polyester/cotton fabrics destined for industrial safety apparel and tents (Mischutin, 1977; LeBlanc, 1979). Decabromodiphenyl oxide is also used in insulation for wire and electrical cable (Ethyl Corp., 1988b).

(*c*) *Regulatory status and guidelines*

The recommended occupational exposure limit for decabromodiphenyl oxide in the USA in 1980 was 5 mg/m$^3$ as an 8-h time-weighted average. The maximum allowable concentration in workplace air in the USSR in 1987 was 3 mg/m$^3$ (Cook, 1987).

## 2.2 Occurrence

(*a*) *Natural occurrence*

Decabromodiphenyl oxide is not known to occur as a natural product.

(*b*) *Occupational exposure*

Analysis of wipe samples collected during an industrial hygiene survey in 1977 and 1978 in a decabromodiphenyl oxide manufacturing plant in Sayreville, NJ, USA, indicated that workers in the reactor area were exposed to 3.6 mg/cm$^2$ and those in the distillation area to 5.9 mg/m$^2$. Analysis of personal samples collected on workers in the mill area indicated that the airborne levels of decabromodiphenyl oxide were 0.08-0.21 mg/m$^3$ as an 8-h time-weighted average. Following a spill in the mill area, personal airborne levels were 1.3-1.9 mg/m$^3$ (Bialik, 1982).

(c) *Other*

Decabromodiphenyl oxide was found at 33-375 μg/kg (dry-weight basis) in river sediment from the Neya River and Second Neya River in Osaka, Japan. Marine sediment from Osaka Bay, however, did not contain detectable levels. Decabromodiphenyl oxide was identified at 20 μg/kg (dry-weight basis) in one of three estuary sediment samples from Osaka but was not detected in samples from Tokyo, Matsuyama or Hiroshima (Watanabe *et al.*, 1986, 1987a,b).

Decabromodiphenyl oxide was reported to occur at 1.4 μg/kg (wet-weight basis) in one of three mussels collected from Osaka Bay. It was not found in mullet, goby, sea bass or horse mackerel from this area or in mussel, mullet, goby, sardine, mackerel or hairtail from other locations. It was not found in human adipose tissue obtained from a hospital in Osaka (Watanabe *et al.*, 1987a,b).

Zweidinger *et al.* (1978) reported levels of none detected to 1 g/kg decabromodiphenyl oxide in sediment samples near a flame-retardant manufacturing facility in the USA. Decabromodiphenyl oxide was also found (limit of detection, 10 μg/kg) in sediment from the vicinity of bromine facilities in El Dorado and Magnolia, AR, and in sludge samples from a discharge-treatment zone of a polybrominated biphenyl facility in Bayonne, NJ (DeCarlo, 1979).

Decabromodiphenyl oxide was detected at concentrations up to 5 μg/kg in samples of human hair obtained from barber shops in the same towns in Arkansas. The author estimated that at least 5% of these populations had detectable levels of decabromodiphenyl oxide in their hair (DeCarlo, 1979).

### 2.3 Analysis

Selected methods for the analysis of decabromodiphenyl oxide are presented in Table 1.

## 3. Biological Data Relevant to the Evaluation of Carcinogenic Risk to Humans

### 3.1 Carcinogenicity studies in animals

*Oral administration*

*Mouse*: Groups of 50 male and 50 female B6C3F$_1$ mice, nine weeks of age, were fed 0, 2.5 or 5.0% decabromodiphenyl oxide (purity, 94-97%; two lots) in the diet for 103 weeks, and all survivors were killed at 112-113 weeks of age. Body weights and survival of treated animals were comparable to those of controls. The combined incidence of hepatocellular adenomas and carcinomas in males was 8/50 in controls, 22/50 in low-dose and 18/50 in high-dose animals (trend not significant). The combined incidence of thyroid gland follicular-cell adenomas and carcinomas was: 0/50 in control males, 4/50 in low-dose males and 3/50 in high-dose males, and 1/50 in control females, 3/50 in low-dose females and 3/50 in high-dose females (neither trend significant; National Toxicology Program, 1986).

**Table 1. Methods for the analysis of decabromodiphenyl oxide**

| Sample matrix | Sample preparation | Assay procedure[a] | Limit of detection | Reference |
|---|---|---|---|---|
| Air | Collect on glass fibre filter; extract with acetone | GC/MS<br>GC/ECD<br>TLC/SD | 20-100 ng/m$^3$[b]<br>Not given<br>Not given | Zweidinger *et al.* (1979) |
| | Collect on glass fibre filter/ Florisil solvent system; desorb with hexane | GC/ECD | 0.3 μg/sample | Eller (1985) |
| Sewage | Extract with chloroform, evaporate; dissolve residue in ethanol | GC/MS | 0.06 mg/dm$^3$ | Kaart & Kokk (1987) |
| Sediment | Extract with diethyl ether; decant; extract with acetone and toluene | GC/MS and TLC/SD | 100 μg/kg | Zweidinger *et al.* (1978) |
| | Extract with acetone; chromatograph on Florisil | NAA and GC/ECD | < 5 μg/kg | Watanabe *et al.* (1986, 1987a,b) |
| Human adipose tissue | Grind with excess anhydrous sodium sulfate; extract with hexane; chromatograph on Florisil | NAA and GC/ECD | < 100 μg/kg | Watanabe *et al.* (1987a) |
| Marine organisms | Homogenize; extract with acetone/hexane mixture; chromatograph on Florisil | GC/ECD and GC/MS | < 0.5 μg/kg | Watanabe *et al.* (1987b) |
| Fish | Grind tissue; extract; clean on Florisil column | GC/ECD | 10 μg/kg | Miller & Puma (1979) |
| Feed | Extract with tetrahydrofuran; filter; elute isocratically with methanol or water/acetonitrile | HPLC | Not given | National Toxicology Program (1986) |

[a]Abbreviations: GC/MS, gas chromatography/mass spectrometry; GC/ECD, gas chromatography/electron capture detection; TLC/SD, thin-layer chromatography/spectrophotometric determination; NAA, neutron activation analysis; HPLC, high-performance liquid chromatography

[b]Variable, due to daily fluctuations

*Rat*: Groups of 25 male and 25 female Sprague-Dawley rats, six to seven weeks of age, were fed 0, 0.01, 0.1 or 1.0 mg/kg bw per day decabromodiphenyl oxide (purity, 77.4%; nonabromodiphenyl oxide, 21.8%; octabromodiphenyl oxide, 0.8%) in the diet for 100-105 weeks. Ingestion of decabromodiphenyl oxide did not influence survival rates, and mean body weights of treated groups were similar to those of controls. No discernible toxicological effect was produced by decabromodiphenyl oxide, and no significant difference in the number of rats developing tumours (total number of tumours or specific type of tumour) was observed between treated and control groups when evaluated by Fisher's exact probability test (Kociba *et al.*, 1975). [The Working Group noted the very low dose levels used.]

Groups of 50 male and 50 female Fischer 344/N rats, seven to eight weeks of age, were fed 0, 2.5 or 5.0% decabromodiphenyl oxide (purity, 94-97%; two lots) in the diet for 103 weeks, and all survivors were killed at 111-112 weeks of age. Body weights of treated rats were not significantly different from those of controls, but after week 75 survival in the treated groups was lower than that in controls. Significant increases in the incidences of neoplastic nodules of the liver [adenomas (Maronpot *et al.*, 1986)] were seen in animals of each sex: they occurred in 1/50 control males and 7/50 at the low dose and 15/49 at the high dose ($p < 0.001$, incidental tumour test for trend); and in females they occurred in 1/50 controls, 3/49 at the low dose and 9/50 at the high dose ($p = 0.002$, incidental tumour test for trend). No difference in the incidence of hepatocellular carcinomas was seen among the groups. The incidence of acinar-cell adenomas of the pancreas was significantly increased in males: control, 0/49; low-dose, 0/50; high-dose, 4/49 ($p = 0.017$, incidental tumour test for trend). A high incidence of mononuclear-cell leukaemia was observed in treated and control rats of each sex: in males, the overall rates were 30/50 controls and 33/50 low-dose and 35/50 high-dose animals; the adjusted rates were 67.9%, 81.9% and 82.8%, respectively ($p = 0.028$, life-table test for trend; National Toxicology Program, 1986).

## 3.2 Other relevant data

(*a*) *Experimental systems*

(i) *Absorption, distribution, excretion and metabolism*

$^{14}$C-Labelled decabromodiphenyl oxide suspended in corn oil was given by gavage to male and female Sprague-Dawley rats at a dose of 1 mg/kg bw. Less than 1% of the label appeared in the urine over 16 days; over 99% of the label appeared in the faeces within two days. Trace amounts of label were detected in the adrenal gland and spleen after sacrifice on day 16 (Norris *et al.*, 1975). When concentrations of 0.025-5% $^{14}$C-labelled decabromodiphenyl oxide were given in the diet to male Fischer 344 rats, more than 99% of the radioactivity was recovered in the faeces and gut contents after 72 h. The liver contained approximately 0.5% of the consumed dose 24 h after feeding; for the high dose, this level declined to 0.016% after 72 h. Labelled material extracted from the liver was found to be mainly unchanged decabromodiphenyl oxide. Trace amounts of label were found in the kidney, spleen, lung, brain, muscle, fat and skin. By 72 h after an intravenous dose of $^{14}$C-labelled decabromodiphenyl oxide, the faeces and gut contents contained 74% of the dose, suggesting significant biliary excretion. Of the extracted faecal label, 63% was metabolites of decabromodiphenyl oxide and 37% was the parent compound. Labelled materials were found in the muscle, skin, liver, fat, kidney and lungs (El Dareer *et al.*, 1987).

Bromine concentrations were determined periodically in selected tissues in two-year dietary studies in male and female Sprague-Dawley rats. A slight increase in the bromine content of liver was observed with 1% in the diet, and increases were seen in adipose tissue with concentrations of 0.1 and 1% (Kociba *et al.*, 1975).

(ii) *Toxic effects*

Decabromodiphenyl oxide has low acute toxicity (Norris *et al.*, 1973; Kociba *et al.*, 1975; Norris *et al.*, 1975).

Daily administration of 0.1 mmol (96 mg)/kg bw decabromodiphenyl oxide in corn oil by gavage to male Sprague-Dawley rats for 14 days resulted in significantly increased liver weights but did not significantly induce NADPH cytochrome c reductase or cytochrome P450 activities (Carlson, 1980).

In 30-day studies in which male Sprague-Dawley rats were maintained on diets containing 0.01-1% decabromodiphenyl oxide (approximately 8-800 mg/kg bw per day), microscopic examination of the livers was reported to have revealed centrilobular cytoplasmic enlargement and vacuolization (with the 1% dose). Other treatment-related lesions were reported to include hyaline degenerative cytoplasmic changes in the kidney (with the 1% dose) and thyroid hyperplasia (with the 0.1 and 1% doses; Norris *et al.*, 1975).

Fourteen-day studies in which concentrations of up to 10% decabromodiphenyl oxide were administered in the diet to Fischer 344 rats and B6C3F$_1$ mice resulted in no death, no compound-related clinical sign and no gross pathological effect. Similarly, no toxic effect was seen in 90-day studies in which doses of up to 5% were administered in the diet. Liver weights were not recorded in these studies, but, in supplementary studies, liver weights of Fischer 344 rats were increased by up to about 40% following consumption of diets containing 0, 2.5 or 5% decabromodiphenyl oxide for 11 days (National Toxicology Program, 1986).

Non-neoplastic changes observed in two-year studies in which dietary concentrations of 2.5 and 5.0% decabromodiphenyl oxide were fed to Fischer 344 rats and B6C3F$_1$ mice (see also section 3.1) included slightly increased incidences of thrombosis, degeneration of the liver, fibrosis of the spleen and acanthosis of the forestomach in male rats. In mice, an increased incidence of centrilobular hypertrophy of the liver and a slight increase in the frequency of granulomas of the liver were observed in treated males. Follicular-cell hyperplasia of the thyroid gland was more prevalent in male and female mice treated with either dose. The incidence of gastric ulcers was increased in female mice fed the high dose (National Toxicology Program, 1986).

(iii) *Effects on reproduction and prenatal toxicity*

Sprague-Dawley rats were administered 10-1000 mg/kg bw decabromodiphenyl oxide per day by gavage on gestation days 6-15. Some embryotoxicity but no malformation was reported (Norris *et al.*, 1973, 1975). [The Working Group noted that details were not given.]

(iv) *Genetic and related effects* (see Appendix 1)

Decabromodiphenyl oxide was not mutagenic to several strains of *Salmonella typhimurium*, in the presence or absence of an exogenous metabolic system from Aroclor 1254-induced rat liver and Syrian hamster liver. It was not mutagenic at the TK locus in L5178Y mouse lymphoma cells and did not cause sister chromatid exchange or chromosomal aberrations in the Chinese hamster CHO cell line (National Toxicology Program, 1986).

No increase in the incidence of chromosomal aberrations was reported in parental or neonate bone-marrow cells of rats given 3-100 mg/kg bw decabromodiphenyl oxide per day in the diet for 90 days (Norris *et al.*, 1975). [The Working Group noted that details were not reported.]

(*b*) *Humans*

No data were available to the Working Group.

### 3.3 Case reports and epidemiological studies of carcinogenicity to humans

No data were available to the Working Group.

## 4. Summary of Data Reported and Evaluation

### 4.1 Exposure data

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.

### 4.2 Experimental carcinogenicity data

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.

### 4.3 Human carcinogenicity data

No data were available to the Working Group.

### 4.4 Other relevant data

In single studies, decabromodiphenyl oxide did not induce sister chromatid exchange or chromosomal aberrations in Chinese hamster cells in culture or mutations in mouse cells in culture. In one study, decabromodiphenyl oxide was not mutagenic to bacteria in the presence or absence of an exogenous metabolic system.

## Summary table of genetic and related effects of decabromodiphenyl oxide

| **Nonmammalian systems** | | | | | | | | | | | | | **Mammalian systems** | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Proka-ryotes | | Lower eukaryotes | | | | Plants | | | Insects | | | | *In vitro* | | | | | | | | | | | | | | *In vivo* | | | | | | | | | | | | | |
| | | | | | | | | | | | | | Animal cells | | | | | | | | Human cells | | | | | | | | Animals | | | | | | | Humans | | | | |
| D | G | D | R | G | A | D | G | C | R | G | C | A | D | G | S | M | C | A | T | I | D | G | S | M | C | A | T | I | D | G | S | M | C | DL | A | D | S | M | C | A |
| | –[1] | | | | | | | | | | | | | –[1] | –[1] | | –[1] | | | | | | | | | | | | | | | | | | | | | | | | |

A, aneuploidy; C, chromosomal aberrations; D, DNA damage; DL, dominant lethal mutation; G, gene mutation; I, inhibition of intercellular communication; M, micronuclei; R, mitotic recombination and gene conversion; S, sister chromatid exchange; T, cell transformation

*In completing the table, the following symbol indicates the consensus of the Working Group with regard to the results for each endpoint:*

–[1] considered to be negative, but only one valid study was available to the Working Group

### 4.5 Evaluation[1]

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

No data were available from studies in humans on the carcinogenicity of decabromodiphenyl oxide.

**Overall evaluation**

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

## 5. References


Aldrich Chemical Co. (1988) *Material Safety Data Sheet: Pentabromophenyl Ether*, Milwaukee, WI

AmeriBrom (1987) *Material Safety Data Sheet: FR-1210*, New York, NY

AmeriBrom (undated) *Technical Product Data Sheet: Decabromodiphenyl Oxide*, New York, NY

Anon. (1984) *Specialty Chemicals Handbook*, Tokyo, The Chemical Daily Co., pp. 39-40

Bialik, O. (1982) *Endocrine Function of Workers Exposed to PBB and PBBO (Terminal Progress Report)*, Cincinnati, OH, National Institute for Occupational Safety and Health

Carlson, G.P. (1980) Induction of xenobiotic metabolism in rats by short-term administration of brominated diphenyl ethers. *Toxicol. Lett., 5*, 19-25

Cook, W.A. (1987) *Occupational Exposure Limits—Worldwide*, Washington DC, American Industrial Hygiene Association

DeCarlo, V.J. (1979) Studies on brominated chemicals in the environment. *Ann. N.Y. Acad. Sci., 320*, 678-681

El Dareer, S.M., Kalin, J.R., Tillery, K.F. & Hill, D.L. (1987) Disposition of decabromodiphenyl ether in rats dosed intravenously or by feeding. *J. Toxicol. environ. Health, 22*, 405-415

Eller, P.M. (1985) *NIOSH Manual of Analytical Methods*, 3rd ed. (*DHHS (NIOSH) Publ. No. 84-1000*), Washington DC, US Government Printing Office, pp. 5013-1-5013-5

Ethyl Corp. (1988a) *Product Information: Saytex 102*, Baton Rouge, LA, Bromine Chemicals Division

Ethyl Corp. (1988b) *Product Information: Saytex 102E*, Baton Rouge, LA, Bromine Chemicals Division

Kaart, K.S. & Kokk, K.Y. (1987) Spectrophotometric determination of decabromodiphenyl oxide in industrial sewage. *Ind. Labor, 53*, 289-290

Klusmeier, W., Vögler, P, Ohrbach, K.-H., Weber, H. & Kettrup, A. (1988) Thermal decomposition of decabromodiphenyl ether. *J. anal. appl. Pyrol., 13*, 277-285

Kociba, R.J., Frauson, L.O., Humiston, C.G., Norris, J.M., Wade, C.E., Lisowe, R.W., Quast, J.F., Jersey, G.C. & Jewett, G.L. (1975) Results of a two-year dietary feeding study with decabromodiphenyl oxide (DBDPO) in rats. *J. Combust. Toxicol., 2*, 267-285

Larsen, E.R. (1980) Flame retardants (halogenated). In: Mark, H.F., Othmer, D.F., Overberger, C.G., Seaborg, G.T. & Grayson, M., eds, *Kirk-Othmer Encyclopedia of Chemical Technology*, 3rd ed., Vol. 10, New York, John Wiley & Sons, pp. 385-386


[1]For description of the italicized terms and criteria for making the evaluation, see Preamble, pp. 25-29.

LeBlanc, R.B. (1979) What's available for FR textiles. *Text. Ind., 143*, 78-83

Maronpot, R.R., Montgomery, C.A., Jr, Boorman, G.A. & McConnell, E.E. (1986) National Toxicology Program nomenclature for hepatoproliferative lesions of rats. *Toxicol. Pathol., 14*, 263-273

Miller, L.J. & Puma, B.J. (1979) Analytical characteristics of late-eluting halogenated flame retardants. *J. Assoc. off. anal. Chem., 62*, 1319-1326

Mischutin, V. (1977) Safe flame retardant. *Am. Dyest. Rep., 66*, 51-56

National Toxicology Program (1986) *Toxicology 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

Norris, J.M., Ehrmantraut, J.W., Gibbons, C.L., Kociba, R.J., Schwetz, B.A., Rose, J.Q., Humiston, C.G., Jewett, G.L., Crummett, W.B., Gehring, P.J., Tirsell, J.B. & Brosier, J.S. (1973) Toxicological and environmental factors involved in the selection of decabromodiphenyl oxide as a fire retardant chemical. *Appl. Polym. Symp., 22*, 195-219

Norris, J.M., Kociba, R.J., Schwetz, B.A., Rose, J.Q., Humiston, C.G., Jewett, G.L., Gehring, P.J. & Mailhes, J.B. (1975) Toxicology of octabromobiphenyl and decabromodiphenyl oxide. *Environ. Health Perspect., 11*, 152-161

Pouchert, C.J., ed. (1981) *The Aldrich Library of Infrared Spectra*, 3rd ed., Milwaukee, WI, Aldrich Chemical Co., p. 635

Pouchert, C.J., ed. (1985) *The Aldrich Library of FT-IR Spectra*, Milwaukee, WI, Aldrich Chemical Co., p. 1057

Tabor, T.E. & Bergman, S. (1975) Decabromodiphenyl oxide—a new fire retardant additive for plastics. In: Bhatnagan, V.M., ed., *Fire Retardants: Proceedings of 1974 International Symposium on Flammability and Fire Retardants, May 1-2, 1974, Cornwall, Ontario, Canada*, Westport, CT, Technomic Publishing, pp. 162-179

Timmons, L.L. & Brown, R.D. (1988) Analysis of the brominated fire retardant decabromodiphenyl oxide for low and trace levels impurities. *Chemosphere, 17*, 217-233

US Environmental Protection Agency (1988) *Information Review: Brominated Diphenyl Ethers (Report No. IR-516)*, Washington DC, TSCA Interagency Testing Committee

Watanabe, I. & Tatsukawa, R. (1987) Formation of brominated dibenzofurans from the photolysis of flame retardant decabromobiphenyl ether in hexane solution by UV and sun light. *Bull. environ. Contam. Toxicol., 39*, 953-959

Watanabe, I., Kashimoto, T. & Tatsukawa, R. (1986) Confirmation of the presence of the flame retardant decabromobiphenyl ether in river sediment from Osaka, Japan. *Bull. environ. Contam. Toxicol., 36*, 839-842

Watanabe, I., Kashimoto, T., Kawano, M. & Tatsukawa, R. (1987a) A study of organic bound halogens in human adipose, marine organisms and sediment by neutron activation and gas chromatographic analysis. *Chemosphere, 16*, 849-857

Watanabe, I., Kashimoto, T. & Tatsukawa, R. (1987b) Polybrominated biphenyl ethers in marine fish, shellfish and river and marine sediments in Japan. *Chemosphere, 16*, 2389-2396

White Chemical Co. (1988) *Material Safety Data Sheet: Decabromodiphenyl Oxide*, Newark, NJ

Zweidinger, R.A., Cooper, S.D. & Pellizzari, E.D. (1978) Identification and quantitation of brominated fire retardants. In: Van Hall, C.E., ed., *Measurement of Organic Pollutants in Water and Wastewater, Symposium of the American Society for Testing and Materials, 19-20 June 1978, Denver, CO (ASTM Technical Publication No. 686)*, Philadelphia, PA, American Society for Testing and Materials, pp. 234-250

Zweidinger, R.A., Cooper, S.D., Erickson, M.D., Michael, L.C. & Pellizzari, E.D. (1979) Sampling and analysis for semivolatile brominated organics in ambient air. In: Schuetzle, D., ed., *Monitoring Toxic Substances* (*ACS Symposium Series 94*), Washington DC, American Chemical Society, pp. 217-231