Decabromodiphenyl Oxide

Document History
Published: 1996
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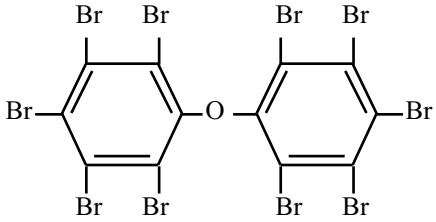
Rebranded: 2025

This OEL was originally established in 1996 and updated in 2001. A literature search to identify new toxicity information for Decabromodiphenyl oxide was performed in August 2008. No new studies or data relevant to the OEL were identified.

I. IDENTIFICATION[1,2]

Chemical Name: Decabromodiphenyl oxide (DBDPO)
Synonyms: Decabromodiphenyl ether (DBDPE); ether, bis(pentabromophenyl); benzene. 1,1 oxy-bis(2,3,4,5,6- Pentabromo-)
CAS Number: 1163-19-5
Molecular Formula: $C_{12}Br_{10}O$
Structural Formula:



II. CHEMICAL AND PHYSICAL PROPERTIES[1–5]

Physical State and Appearance: White to off-white powder
Odor Description and Threshold: No data available.
Molecular Weight: 960
Specific Gravity: 3.0
Conversion Factors: 1 mg/m^3 = 0.025 ppm;
1 ppm = 39.26 mg/m^3
Boiling Point: Decomposes at 425°C (797°F)
Melting Point: 290–306°C (554–583°F)
Vapor Pressure: < 10^{-6} mmHg at 20°C (68°F)
<1 mmHg at 250°C (482°F)
2 mmHg at 278°C (532°F)
5 mmHg at 306°C (583°F)
Saturated Vapor Concentration: < 1316 ppm at 250°C (482°F)
Flash Point: No data available
Flammability Limits: No data available
Autoignition Temperature: No data available
Relative Density of Saturated Air: No data available
Solubility in Water: 20–30 ppb at 20°C (68°F)
Stability: Stable
Reactivity and Incompatibilities: None known

III. USES AND VOLUME

A typical composition of the technical product in the 1970s was 77.4% DBDPO, 21% non-Abromodiphenyl oxide, and 0.8% octabromodiphenyl oxide.[3,4]

The purity of the product has improved and typical commercial product today is greater than or equal to 97% pure DBDPO. DBDPO is used as a flame-retardant agent for high-impact polystyrene, acrylonitrile-butadiene styrene (ABS) polymers, nitrile resins, adhesives, polyethylene, polyester, polystyrene, polyvinyl acetate, polyvinyl chloride, thermoset applications of epoxy resins and unsaturated polymers and has found acceptance as a fire retardant in synthetic fibers. Some concern has been expressed, however, over combustion of flame-retardant agents such as DBDPO and the potential formation of polybrominated dibenzodioxin (PBDD) or poly-brominated dibenzofuran (PBDF).[3–6]

IV. ANIMAL TOXICITY DATA

A. Acute Toxicity

1. *Oral Toxicity*

Rats: LD_{50} > 5000 mg/kg[1–3,5]

2. *Eye Irritation*

Rabbits: Transient, mild irritation.[1–3,5–6]

3. *Skin Absorption*

Rabbits: LD_{50} = > 2000 mg/kg[1]

4. *Skin irritation*

Rabbits: Practically nonirritating to slight irritation to intact skin.[1,2] Slight erythema and edema to intact skin.[3–5]

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5. *Inhalation Toxicity*

   Rats: LC 5 = >48.2 mg/L

6. *Acnegenic Activity*

   Rabbits: Negative; DBDPO applied as a 10% chloroform solution caused only a erythematous response and a slight exfoliation during the month-long study.[3,5]

B. Subacute Toxicity

Oral administration of 96 mg/kg of body weight of DBDPO suspended in corn oil fed by gavage to rats for 14 days had no significant effect on liver enzymes but did increase liver weights.[7]

In another 14-day study, groups of 5 males and 5 females of both rats and mice were fed diets containing 0, 5000, 10,000, 20,000, 50,000, and 100,000 ppm DBDPO daily. These dietary concentrations would result in dosages of approximately 0, 500, 1000, 2000, 5000, and 10,000 mg/kg/day. There were no treatment-related effects with regard to clinical signs, body weights, survival, gross pathology, or histopathology).[6]

C. Subchronic Toxicity

Feeding studies on both male and female rats produced no toxic symptoms when 8 mg/kg or 80 mg/kg of body weight per day was administered in the diet for 30 days. At a dietary level of 800 mg/kg of body weight per day for 30 days, the following changes were observed: centrilobular cytoplasmic enlargement and vacuolation in the liver and hyaline degenerative cytoplasmic changes in the kidney. Thyroid hyperplasia was also noted in rats given 80 or 800 mg/kg of body weight per day for 30 days. A no observable effect level (NOEL) was determined to be 8 mg/kg, with marginal effect noted at 80 mg/kg. The technical grade of DBDPO in this study was 77.4% pure.[5,6]

In a 13-week study, groups of 10 males and 10 females of both rats and mice were fed diets containing 0, 3, 100, 200, 12,500, 25,000, and 50,000 ppm DBDPO daily. These dietary concentrations would result in dosages of approximately 0.2, 7, 413, 833, 1667, and 3333 mg/kg/day. The DBDPO used in this study was 97%–99% pure. There were no treatment related effects with regard to clinical signs, consumption, body weights, survival, gross pathology or histopathology.[6]

D. Chronic Toxicity/Carcinogenicity[6]

In a 2-year feeding study, groups of 25 male and 25 female rats were fed 0, 0.01, 0.1, or 1.0 mg/kg of body weight of DBDPO per day. Increased bromine content in adipose tissue was noted in the two highest dose feeding groups; however, no discernible toxicological effects were revealed in the study. No dose-related deaths were observed, and body weight and food consumption of treated rats were not significantly different from the control rats. The same study did not show a significant increase in tumors over control rats.

In another study, groups of 50 F344 rats and B6C3F1 mice of each sex were fed 0, 25,000, or 50,000 ppm DBDPO for two years. The average dosages for treated rats were approximately 1160 mg/kg /day and 2395 mg/kg/day, and for mice they were approximately 3480 mg/kg/day and 7215 mg/kg/day. No compound-related effects on clinical signs, body weight, feed consumption, or survival were reported for wither species. Some treatment-related histopathological changes were reported for both rats and mice.

Rats

Treatment-related effects included an increase in neoplastic nodules in the liver for males (1/50, 7/50, and 15/49 for the control, low-, and high-dose groups, respectively) and females (1/50, 3/49, and 9/50 for the control, low-, and high-dose groups, respectively). The incidence was statistically significant for the low-dose males and the high dose of both sexes. There was no treatment-related increase in hepatocellular carcinomas.

The National Toxicology Program (NTP) concluded that the increase in the hepatic neoplastic nodules provided “some evidence of carcinogenicity” in male and female rats.

Other possible treatment-related nonneoplastic histopathologic changes included an increase in hematopoiesis in the spleens of treated females, and acanthosis of the forestomach in treated males.

Mice

Treatment-related effects included an increase in the combined incidence of hepatocellular

adenomas and carcinomas in males (8/50, 22/50,and 18/50, for the control, low-, and high-dose groups, respectively). The increase at the low dose was statistically significant. There was also an increase in the combined incidence of thyroid follicular cell adenomas and carcinomas in males (0/50, 4/50, and 3/50 for the control, low-, and high-dose groups, respectively), but the increase was not statistically significant; however, there was a concomitant increase in follicular cell hyperplasia (2/50, 10/50, and 19/50 for the control, low-, and high-dose groups. respectively) in male mice. The NTP concluded that the increase

in hepatocellular neoplasms in low-dose males and the slight increase in thyroid tumors provided "equivocal evidence of carcinogenicity" in male mice.

The NTP concluded that the increase in hepatocellular neoplasms in low-dose males and the slight increase in thyroid tumors provided "equivocal evidence of carcinogenicity" in male mice. The NTP concluded that there was no evidence of carcinogenicity in female mice.

Other possible treatment-related histopathologic changes included an increased in centrilobular hypertrophy in the livers of male mice and an increase in occurrence of hepatic granulomas in the low-dose males. Also, an increase in ulcers of the stomach was noted in the high-dose females.

E. Reproductive/Developmental Toxicity

Oral administration of 10, 100, or 1000 mg/kg of body weight of DBDPO in corn oil suspension to pregnant rats on Days 6–15 of gestation produced no teratogenic response; however, some subcutaneous edema and delayed ossification of bones were noted in fetuses from the highest dose level group.[3–5]

In another study, rats were fed 3, 30, or 100 mg/kg per body weight of DBDPO per day for 90 days before mating. Rats were fed during mating, gestation, and lactation. The reproductive capacity of rats was not affected by the DBDPO included in the diets.[3]

F. Genotoxicity

One short-term study revealed no increase in cytogenic aberrations compared with controls in the bone marrow cells from rats given 3, 30, or 100 mg/kg/day of DBDPO in the diet for 90 days prior to mating, during mating, and during gestation and lactation. Bone marrow cells from neonates of these rats showed no increase in cytogenetic aberrations when compared with controls.[3,5,8]

DBDPO was not mutagenic when tested in 5 strains of Salmonella typhimurium bacteria (TA1535. TA1537, TA1538, TA98, and TA100) — the Ames Assay — with or without metabolic activation.[6,9,10]

In the mouse Lymphoma L5I78YITK*/, assay, DBDPO was found to be not mutagenic with or without metabolic activation.[6,11]

DBDPO did not cause chromosomal aberrations or sister-chromatid exchanges (SCEs) in Chinese hamster ovary cells either with or without metabolic activations.[6]

G. Metabolism/Pharmacokinetics

1. Oral Toxicity

Rats: Material balance studies in rats revealed that more than 99% of an oral dose was eliminated in the feces of treated rats within 2 days. Small amounts of the radio- labeled chemical were detected only in the adrenals (0.01 of the dose/g tissue).[3,5]

Tissue distribution studies in rats that received 0.1 mg/kg/day in the diet for 180 days. Showed no accumulation of bromine in any tissue. When the rats were exposed to a dietary level of 1.0 mg/kg/day, a dose-related bromine concentration increase was observed in adipose tissue at, and subsequent to, 3 and 6 months, respectively.

A metabolism study in rats revealed that 99% of the radioactivity, from an oral dose of '4C-labeled DBDPO, was recovered in the feces after 72 hr. Labeled material extracted from the liver was identified mainly as unchanged DBDPO. The kidney. spleen. lung, brain, muscle, fat, and skin contained trace amounts of DBDPO)[6,12]

2. Intravenous Toxicity

Rats

An intravenous dose of i4C labeled DBDPO resulted in 74% of the dose identified in the gut and feces within 74 hr. Of the material extracted from the feces, 63% were metabolites of DBDPO and 37% was unchanged DBDPO. Muscle, skin, liver, lungs. kidney. and adipose tissue contained labeled DBDPO.[12]

V. HUMAN USE AND EXPERIENCE

Industrial hygiene surveys determined employee 8-hr time-weighted average (TWA) exposures of 1–4 mg/rn^3 with excursions up to 42 mg/rn^3 during short-term tasks. Airborne concentrations up to 400 mg/m^3 were measured during a dumping operation, which was carried out by employees wearing respirators. In this study, more than 90% of the particulate collected was smaller than 10 microns in diameter.[6]

Personal sampling of workers in a mill area of a DBDPO manufacturing plant increased 8-hr TWAs of 0.08–0.21 mg/m^3.[13]

In a Repeated Insult Human Patch Test, application of a homogenous 5% suspension of DBDPO in petrolatum 3 times/week for 3 weeks to the skin of 50 subjects did' not induce sensitization during the induction period or the subsequent challenge period two weeks later.[5]

VI. RATIONALE

DBDPO, based on acute toxicity testing, would be considered to be practically nontoxic via ingestion, inhalation, or dermal absorption. DBDPO is not an eye or skin irritant and is not considered to be a skin sensitizer. Genotoxicity testing using several different assays indicates DBDPO to be non-genotoxic. Based on reproductive and developmental assays, DBDPO is not considered to be a reproductive hazard. Absorption and metabolism studies indicated DBDPO is poorly absorbed from the gastrointestinal tract.

A subchronic rat feeding study did reveal lesions in the liver, kidney, and thyroid gland. Doses of 8, 80, and 800 mg/kg of body weight for 30 days were used in this study and a NOEL of 8 mg/kg was established with a marginal lowest observed effect level (LOEL) of 80 mg/kg identified. The technical grade of DBDPO was found to be 77.4% pure.

Subsequent subchronic rat feeding studies for 13 weeks using pure grades of DBDPO revealed no significant compound-related gross or microscopic pathologic effects. Earlier conducted studies identifying lesions in the liver, kidney, and thyroid may be associated with the presence of the lesser brominated diphenyl oxides present in 77.4% pure technical grade DBDPO.

Chronic toxicity and carcinogenic testing using 2-year feeding studies on mice and rats with 94%–99% pure DBDPO led the NIP to conclude that "there was some evidence of carcinogenicity for male and female F34.4/N rats as shown by increased instances of neoplastic nodules of the liver in low dose (25,000 ppm) males and high dose (50,000 ppm) groups of each sex. There was equivocal evidence of carcinogenicity for male B6C3F1 mice as shown by increased incidences of hepatocellular adenomas or carcinomas (combined) in the low-dose group and high-dose groups, and of thyroid gland follicular cell adenomas or carcinomas (combined) in both dose groups. There was no evidence of carcinogenicity for female B6C3F1 mice receiving 25,000 ppm or 50,000 ppm in the diet. Several non-neoplastic lesions were observed at increased incidences, the most notable being thyroid gland follicular cell hyperplasia in male mice.[6]

The International Agency for Research Cancer (IARC) evaluation of DBDPO was "limited evidence for the carcinogenicity of decabromodiphenyl oxide" in animals. IARC's overall evaluation of DBDPO was "not classifiable as to its carcinogenicity to humans (Group 3).[14]

Additional toxicity testing has been conducted since AIHA recommended an OEL guide of 5 mg/rn^3 as an 8-hr TWA in 1980; however, the lack of general toxicity observed at all but extremely high doses, the lack of genotoxicity, and the modest increase in tumors (generally benign) at extremely high doses (1160–7215 mg/kg) in organs (i.e., liver and thyroid), Commonly affected by nongenotoxic carcinogens indicate that this compound has limited toxic potential. Based on the additional testing, and given the relatively small particulate size of DBDPO, an OEL of 5 mg/rn^3 should provide adequate worker protection.

VII. RECOMMENDED OEL GUIDE

8-hr TWA: 5 mg/m^3 (0.13 ppm)

VIII. REFERENCES

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