SAFETY DATA SHEET

Updated on 26/09/2024

1. Material Identification

Product Name : Decabromodiphenyl oxide
Catalog Number : io-2100
CAS Number : 1163-19-5
Identified uses : Laboratory chemicals, manufacture of chemical compounds
Company : Ionz

>> R&D Use only

2. Hazards Identification

GHS Classification:

Flammable liquid (category 2)
Acute toxicity, oral (Category 3)
Acute toxicity, dermal (Category 3)
Acute toxicity, inhalation (Category 3)
Specific target organ toxicity, single exposure (Category 1)

Note

>> This chemical does not meet GHS hazard criteria for 71.3% (191 of 268) of all reports. Pictograms displayed are for 28.7% (77 of 268) of reports that indicate hazard statements.

Pictogram(s)

>> Warning

GHS Hazard Statements

>> H302 (10.8%): Harmful if swallowed [Warning Acute toxicity, oral]

>> H319 (10.1%): Causes serious eye irritation [Warning Serious eye damage/eye irritation]

>> H413 (10.1%): May cause long lasting harmful effects to aquatic life [Hazardous to the aquatic environment, long-term hazard]

Precautionary Statement Codes

>> P264, P264+P265, P270, P273, P280, P301+P317, P305+P351+P338, P330, P337+P317, and P501

Health Hazards:

>> SYMPTOMS: Symptoms of exposure to this compound may include irritation of the skin, eyes, mucous membranes and upper respiratory tract. It may also cause diarrhea, liver damage and kidney damage. Chronic exposure may cause intoxication.

>> ACUTE/CHRONIC HAZARDS: This compound is an irritant of the skin, eyes, mucous membranes and upper respiratory tract. It may be harmful by inhalation, ingestion and skin absorption. When heated to decomposition it emits toxic fumes of carbon monoxide and carbon dioxide. It may also emit fumes of hydrogen bromide. (NTP, 1992)

>> Flash point data for this chemical are not available; however, it is probably combustible. (NTP, 1992)

>> Not combustible.

3. Composition/Information On Ingredients

Chemical name : Decabromodiphenyl oxide
CAS Number : 1163-19-5
Molecular Formula : C12Br10O
Molecular Weight : 959.2000 g/mol

4. First Aid Measures

First Aid:

>> EYES: First check the victim for contact lenses and remove if present. Flush victim's eyes with water or normal saline solution for 20 to 30 minutes while simultaneously calling a hospital or poison control center. Do not put any ointments, oils, or medication in the victim's eyes without specific instructions from a physician. IMMEDIATELY transport the victim after flushing eyes to a hospital even if no symptoms (such as redness or irritation) develop.

>> SKIN: IMMEDIATELY flood affected skin with water while removing and isolating all contaminated clothing. Gently wash all affected skin areas thoroughly with soap and water. If symptoms such as redness or irritation develop, IMMEDIATELY call a physician and be prepared to transport the victim to a hospital for treatment.

>> INHALATION: IMMEDIATELY leave the contaminated area; take deep breaths of fresh air. IMMEDIATELY call a physician and be prepared to transport the victim to a hospital even if no symptoms (such as wheezing, coughing, shortness of breath, or burning in the mouth, throat, or chest) develop. Provide proper respiratory protection to rescuers entering an unknown atmosphere. Whenever possible, Self-Contained Breathing Apparatus (SCBA) should be used; if not available, use a level of protection greater than or equal to that advised under Protective Clothing.

>> INGESTION: DO NOT INDUCE VOMITING. If the victim is conscious and not convulsing, give 1 or 2 glasses of water to dilute the chemical and IMMEDIATELY call a hospital or poison control center. Be prepared to transport the victim to a hospital if advised by a physician. If the victim is convulsing or unconscious, do not give anything by mouth, ensure that the victim's airway is open and lay the victim on his/her side with the head lower than the body. DO NOT INDUCE VOMITING. IMMEDIATELY transport the victim to a hospital.

>> OTHER: Since this chemical is a known or suspected carcinogen you should contact a physician for advice regarding the possible long term health effects and potential recommendation for medical monitoring. Recommendations from the physician will depend upon the specific compound, its chemical, physical and toxicity properties, the exposure level, length of exposure, and the route of exposure. (NTP, 1992)

First Aid Measures

Inhalation First Aid

>> Fresh air, rest.

Skin First Aid

>> Rinse and then wash skin with water and soap.

Eye First Aid

>> Rinse with plenty of water (remove contact lenses if easily possible).

Ingestion First Aid

>> Rinse mouth. Give one or two glasses of water to drink.

5. Fire Fighting Measures

>> Fires involving this material can be controlled with a dry chemical, carbon dioxide or Halon extinguisher. A water spray may also be used. (NTP, 1992)

>> In case of fire in the surroundings, use appropriate extinguishing media.

6. Accidental Release Measures

Spillage Disposal:

Methods for containment and safety measures to protect workers dealing with a spillage of this chemical.

>> Personal protection: particulate filter respirator adapted to the airborne concentration of the substance. Sweep spilled substance into covered containers. If appropriate, moisten first to prevent dusting.

7. Handling And Storage

Safe Storage:

>> Separated from food and feedstuffs.

Storage Conditions:

>> Keep container tightly closed in a dry and well-ventilated place. Keep in a dry place.

8. Exposure Control/ Personal Protection

Inhalation Risk:

>> A nuisance-causing concentration of airborne particles can be reached quickly.

Effects of Long Term Exposure:

>> The substance may have effects on the thyroid.

Inhalation Prevention

>> Use ventilation.

Skin Prevention

>> Protective gloves.

Eye Prevention

>> Wear safety spectacles.

Ingestion Prevention

>> Do not eat, drink, or smoke during work.

Exposure Control and Personal Protection

Exposure Summary

>> TIH (Toxic Inhalation Hazard) - Term used to describe gases and volatile liquids that are toxic when inhaled. Some are TIH materials themselves, e.g., chlorine, and some release TIH gases when spilled in water, e.g., chlorosilanes. [ERG 2016].

9. Physical And Chemical Properties

Molecular Weight:

>> 959.2

Exact Mass:

>> 959.16804

Physical Description:

>> Decabromodiphenyl oxide is a white to off-white powder with a chemical odor. (NTP, 1992)

>> WHITE CRYSTALLINE POWDER.

Color/Form:

>> Yellow prisms from toluene

Odor:

>> Odorless

Boiling Point:

>> Decomposes at 425 °C

Melting Point:

>> 569.1 to 576.5 °F (NTP, 1992)

>> 300-310 °C

Solubility:

>> less than 1 mg/mL at 68 °F (NTP, 1992)

>> Solubility in water: none

Density:

>> 3 (NTP, 1992) - Denser than water; will sink

>> Relative density (water = 1): 3.0

Vapor Pressure:

>> less than 1 mmHg at 68 °F (NTP, 1992)

>> Vapor pressure, Pa at 21 °C: (negligible)

LogP:

>> log Kow = 9.97 /HPLC method/

>> 6.27

Stability/Shelf Life:

>> Stable under recommended storage conditions.

Decomposition:

>> When heated to decomposition it emits toxic fumes of /hydrogen bromide/.

Collision Cross Section:

Collision cross section (CCS) represents the effective area for the interaction between an individual ion and the neutral gas through which it is traveling (e.g., in ion mobility spectrometry (IMS) experiments). It quantifies the probability of a collision taking place between two or more particles.

>> 204.5 Å² [M-Br+O]-

10. Stability And Reactivity

>> Insoluble in water.

11. Toxicological Information

Toxicity Summary:

>> IDENTIFICATION AND USE: Decabromodiphenyl ether (deca-BDE) is used as a flame retardant. It is mostly used in applications in the plastics and textile industries. It is an additive flame retardant, i.e. it is physically combined with the material being treated rather than chemically combined (as in reactive flame retardants). HUMAN EXPOSURE AND TOXICITY: Deca-BDE did not produce skin sensitization in human subjects. A health assessment of workers exposed for at least 6 weeks to polybromodiphenyls and polybromodiphenyl oxides, including deca-BDE, during manufacture revealed a higher than normal prevalence of primary hypothyroidism with elevated serum concentrations of thyrotropin and low or borderline-low, serum T4 and free thyroxine indexes in 4 of the 35 occupationally exposed vs. 0 of the 89 control subjects. A significant reduction in sensory and fibula motor velocities was also observed. This primary hypothyroidism was partially reversible in 1 of the 3 workers re-evaluated one year after the initial study. The 2 other

workers reassessed still exhibited low free thyroxine indexes and high thyrotrophin values. Significant correlation was seen between length of employment and concentrations of follicle stimulating hormone in workers exposed to deca-BDE An abnormal follicle stimulating hormone value was found in only one worker. A testicular cyst was found in one exposed worker, and epididymal nodules in two others. No testicular or epididymal nodules were seen among comparisons. In other studies deca-BDE was detected in human serum, milk, and sperm. Deca-BDE induced DNA damage in human neuroblastoma cells. ANIMAL STUDIES: Deca-BDE caused no dermal response, and did not cause primary eye irritation in rabbits. Deca-BDE is poorly absorbed and does not easily penetrate the cell wall. Its acute and chronic toxicities are relatively low, with the liver and the thyroid as the primary targets, though there is some evidence of carcinogenicity. In NTP 2-year feeding studies, there was some evidence of carcinogenicity for male and female rats as shown by increased incidences 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 mice as shown by increased incidences of hepatocellular adenomas or carcinomas (combined) in the low dose group and of thyroid gland follicular cell adenomas or carcinomas (combined) in both dosed groups. There was no evidence of carcinogenicity for female mice receiving 25,000 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. Several animal studies have indicated that deca-BDE may cause developmental neurotoxicity, affecting motor and cognitive domains. Deca-BDE exposure during pregnancy and lactation impaired immune function in rats. Several in vivo and in vitro studies have also demonstrated effects of deca-BDE on thyroid hormone homeostasis. Deca-BDE was not genotoxic in Salmonella typhimurium TA98-100-1535-1537 and Escherichia coli WP2 uvr with or without activation. It was also not mutagenic in the mouse lymphoma L5178y/TK + or - assay in the presence or absence of metabolic activation. Tests for cytogenetic effects in Chinese hamster ovary cells indicated that this chemical does not cause chromosomal aberrations or sister chromatid exchanges either in the presence or absence of activation. ECOTOXICITY STUDIES: Deca-BDE was found in tissues of birds, mammals, and in aquatic species. Metabolism via debromination appears to be a major degradation route of Deca-PBE in juvenile sole in comparison to biotransformation into hydroxylated metabolites. In plants Deca-BDE exposure could cause oxidative stress and damage.

Evidence for Carcinogenicity:

Evidence that this chemical does or may cause cancer. The information here is collected from various sources by the Hazardous Substances Data Bank (HSDB).

>> Evaluation: No epidemiological data relevant to the carcinogenicity of decabromodiphenyl oxide. 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).

Carcinogen Classification:

This section provides the International Agency for Research on Cancer (IARC) Carcinogenic Classification and related monograph links. In the IARC Carcinogenic classification, chemicals are categorized into four groups: Group 1 (carcinogenic to humans), Group 2A (probably carcinogenic to humans), Group 2B (possibly carcinogenic to humans), and Group 3 (not classifiable as to its carcinogenicity to humans).

IARC Carcinogenic Agent

>> Decabromodiphenyl oxide

IARC Carcinogenic Classes

>> Group 3: Not classifiable as to its carcinogenicity to humans

IARC Monographs

>> Volume 48: (1990) Some Flame Retardants and Textile Chemicals, and Exposures in the Textile Manufacturing Industry

>> Volume 71: (1999) Re-evaluation of Some Organic Chemicals, Hydrazine and Hydrogen Peroxide (Part 1, Part 2, Part 3)

>> 3, not classifiable as to its carcinogenicity to humans. (L135)

Health Effects:

>> Polybrominated diphenyl ethers may affect the thyroid gland and liver. Animals studies have also shown that PDBEs can cause neurobehavioral alterations and affect the immune system. (L628)

Exposure Routes:

>> The substance can be absorbed into the body by inhalation.

>> Oral (L628) ; inhalation (L628) ; dermal (L628)

Target Organs:

Organs that are affected by exposure to this chemical. Information in this section reflects human data unless otherwise noted.

>> Nervous

Cancer Sites:

The site in which cancer develops due to exposure to this compound. Cancers are casually referred to based on their primary sites (e.g., skin, lung, breasts, prostate, colon and rectum).

>> Hepatic

Adverse Effects:

An adverse effect is an undesired harmful effect resulting from a medical treatment or other intervention.

>> Occupational hepatotoxin - Secondary hepatotoxins: the potential for toxic effect in the occupational setting is based on cases of poisoning by human ingestion or animal experimentation.

Minimum Risk Level:

The minimal risk level (MRL) is an estimate of the amount of a chemical a person can eat, drink, or breathe each day without a detectable risk to health

>> Intermediate Inhalation: 0.006 mg/m3 (L134) Acute Oral: 0.03 mg/kg/day (L134) Intermediate Oral: 0.007 mg/kg/day (L134)

Treatment:

Treatment when exposed to toxin

>> EYES: irrigate opened eyes for several minutes under running water. INGESTION: do not induce vomiting. Rinse mouth with water (never give anything by mouth to an unconscious person). Seek immediate medical advice. SKIN: should be treated immediately by rinsing the affected parts in cold running water for at least 15 minutes, followed by thorough washing with soap and water. If necessary, the person should shower and change contaminated clothing and shoes, and then must seek medical attention. INHALATION: supply fresh air. If required provide artificial respiration.

Interactions:

>> The aim of this study was to see how a mixture of cadmium (Cd) and decabrominated diphenyl ether (BDE209) affect thyroid function, namely thyroid-stimulating hormone (TSH), thyroxin (T4), free thyroxin (FT4), triiodothyronin (T3), and free triiodothyronin (FT3) in Wistar rats (eight per group) receiving either a single substance or their combination by gavage for 28 days. Three groups were receiving Cd alone in the doses of 2.5 mg/kg, 7.5 mg/kg, or 15 mg/kg bw a day, three groups were receiving BDE209 in the doses of 1000 mg/kg, 2000 mg/kg, or 4000 mg/kg bw a day, while nine groups were receiving different mixtures of Cd and BDE209 in these doses (3x3 design). The results have indicated that the Cd+BDE209 mixtures more potently disrupt thyroid hormone homeostasis than would be expected from these chemicals alone.

Antidote and Emergency Treatment:

>> /SRP:/ Immediate first aid: Ensure that adequate decontamination has been carried out. If patient is not breathing, start artificial respiration, preferably with a demand valve resuscitator, bag-valve-mask device, or pocket mask, as trained. Perform CPR if necessary. Immediately flush contaminated eyes with gently flowing water. Do not induce vomiting. If vomiting occurs, lean patient forward or place on the left side (head-down position, if possible) to maintain an open airway and prevent aspiration. Keep patient quiet and maintain normal body temperature. Obtain medical attention. /Poisons A and B/

Human Toxicity Excerpts:

>> /HUMAN EXPOSURE STUDIES/ In 50 human subjects, repeated application of a suspension of 5% DBDPO in petrolatum 3 times a week for 3 weeks and challenged two weeks subsequent to the last induction application did not result in skin sensitization. Skin irritation was observed in 9 out of the 50 persons. Human volunteers (80 males and 120 females) were treated with 9 induction patches of 2 batches of DBDPO (no information on purity is provided). The first sample was evaluated as received, and the second as a 2% (w/v) aqueous solution. The patches were applied once every 2 days and allowed to contact the skin for 24 hr, then the skin was graded for irritation. 15 subjects among the 200 volunteers showed some slight irritation reactions: very slight erythema - barely perceptible in 14/1,800 patches and mild - well defined erythema in 2/1,800 patches and very slight edema - barely perceptible in 1/1,800 patches. After a non-patching period of 12 days, the challenge patch was applied to detect sensitization. This study did not reveal any evidence of skin sensitization with either of the test materials in any of the subjects tested.

Non-Human Toxicity Excerpts:

>> /LABORATORY ANIMALS: Acute Exposure/ A study /was conducted/ in weanling rats to investigate the mechanism(s) by which deca-BDE interferes with thyroid hormone homeostasis. In this study, Long-Evans female rats (eight animals/dose group) were orally administered decaBDE (>98% purity) in corn oil at doses of 0, 0.3, 1, 3, 10, 30, 60, or 100 mg/kg-day for 4 consecutive days. Body weights were recorded and dosing volumes adjusted daily. Animals were sacrificed 1 day after the last dose. Serum total thyroxine (T4) and triiodothyronine (T3), serum thyroid stimulating hormone (TSH), and hepatic enzyme activities (EROD, a marker for CYP-1A1; PROD, a marker for CYP-2B1; and T4-uridine diphosphate glucuronyl transferase [T4-UDPGT]) were measured. Short-term treatment with deca-BDE did not cause any visible signs of toxicity or any effects on body-weight gain or liver-to-body-weight ratios at any dose level. Deca-

BDE (up to 100 mg/kg-day) had no effect on serum T4, T3, or TSH concentration or on hepatic UDPGT activity. Based on these observations, the highest dose of 100 mg/kg-day is identified as the no-observed-adverse-effect level (NOAEL).

National Toxicology Program Studies:

Reports from the National Toxicology Program, an interagency program supported by three government agencies (NIH, FDA, and CDC) within the Department of Health and Human Services. This program plays a critical role in generating, interpreting, and sharing toxicological information about chemicals of public health concerns.

>> NTP studies of decabromodiphenyl oxide mutagenicity indicate that it was not mutagenic in Salmonella typhimurium strains TA1535, TA1537, TA98, or TA100 in the presence or absence of Aroclor 1254 induced male Sprague Dawley rat or male Syrian hamster liver S9 when tested according to the preincubation protocol. It was also not mutagenic in the mouse lymphoma L5178y/TK + or - assay in the presence or absence of Aroclor 1254 induced male F344 liver S9. Tests for cytogenetic effects in Chinese hamster ovary cells indicated that this chemical does not cause chromosomal aberrations or sister chromatid exchanges either in the presence or absence of S9 prepared from livers of Aroclor 1254 induced male Sprague Dawley rats.

12. Ecological Information

Resident Soil (mg/kg)

>> 4.40e+02

Industrial Soil (mg/kg)

>> 3.30e+03

Tapwater (ug/L)

>> 1.10e+02

MCL (ug/L)

>> 2.00e+02

Risk-based SSL (mg/kg)

>> 6.20e+01

Oral Slope Factor (mg/kg-day)-1

>> 7.00e-04

Chronic Oral Reference Dose (mg/kg-day)

>> 7.00e-03

Volatile

>> Volatile

Mutagen

>> Mutagen

Fraction of Contaminant Absorbed in Gastrointestinal Tract

>> 1

Fraction of Contaminant Absorbed Dermally from Soil

>> 0.1

ICSC Environmental Data:

>> Environmental effects from the substance have not been investigated adequately.

Sediment/Soil Concentrations:

Concentrations of this compound in sediment/soil.

>> SEDIMENT: Decabromodiphenyl ether was found in river sediment from the Neya and Second Neya River in Osaka, Japan, 33-375 ppb (dry weight) and one of seven estuary sediments from different rivers in Japan, 20 ppb (dry weight) (1). Decabromodiphenyl ether was not detected (detection limit 5 ppb) in two samples of marine sediment from Osaka Bay, Japan(1). It was detected in soil and sediment samples near the bromine industry in Magnolia and El Dorado, AR(2). Sediment collected in 1995 and 1996 from 4 UK estuaries, selected because brominated flame retardants are

manufactured within their catchments, contained decabromodiphenyl ether at concentrations ranging from <0.6 to 3190 ug/kg dry weight (16 of 29 samples below the detection limit)(3). Surficial sediments collected at 8 different locations in the River Viskan and its tributary, River Haggan (Sweden) in 1995, both up and downstream from potential sources of brominated flame retardants, contained decabromodiphenyl ether at mean concentrations from <20 to 12,000 ng/g (as ignition loss)(4). Decabromodiphenyl ether was detected in 22 sediment samples from 17 locations throughout The Netherlands taken April, July, and September 1999 at <4-510 ug/kg dry weight(5). Decabromodiphenyl ether was detected in sediment samples in the Cinca River, Spain; upstream of Monzon it was measured at 2.1 ng/g dry weight at two sites, just down stream of Monzon it was 39.9 ng/g dry weight and another 30 m down stream was 5.7 ng/g dry weight(6). Decabromodiphenyl ether was detected in sediment of Hadley Lake, IN at 19-36 ug/kg dry weight(7). Decabromodiphenyl ether was detected at 3190 ug/kg in the sediment of the Calder River, UK, downstream of a sewage treatment plant(7). Suspended sediment samples collected from the Detroit River in 2001 and 2006 contained decabromodiphenyl ether levels ranging from 209-184,000 pg/g dry wt(8).

Fish/Seafood Concentrations:

Concentrations of this compound in fish or seafood.

>> Decabromodiphenyl ether was found in 1 of 3 mussels from Osaka Bay, Japan at 1.4 ppb, wet weight, but not found (<0.5 ppb wet weight) in 14 other samples of fish and shellfish from Osaka Bay and other locations in Japan(1). Fish and shellfish samples collected from 1994 to 1996 from 4 UK estuaries, selected because brominated flame retardants are manufactured within their catchments, did not contain measurable concentrations of decabromodiphenyl ether (<1.2 ug/kg wet weight; n=23)(2). Pike sampled at 4 locations along the River Viskan and Lake Skaresjon, Sweden, in 1995, contained decabromodiphenyl ether at non-detectable to trace concentrations in muscle lipid (detection limit=100 ng/g lipid)(3). Decabromodiphenyl ether was detected in conger eel, flounder, grey mullet, horse mackerel, red sea beam, sea bass and yellowtail at concentrations of <0.2-0.53, <0.2-3.2, <0.2-0.25, <0.2-1.4, <0.2-0.74, 0.40-0.81, and <0.2-0.60 ng/g extractable lipid, respectively(4). Decabromodiphenyl ether was detected in 35 flounder samples from 20 locations at <0.2-<6 ug/kg dry weight, 35 bream samples from 20 locations at <0.03-2.1 ug/kg dry weight, 16 marine mussel samples from 16 locations at <4-<5 ug/kg dry weight, and 16 fresh water mussel samples from 16 locations at <4-<35 ug/kg dry weight, all taken April, July, and September 1999 in the Netherlands(5). Five marine fish species, including yellowband sweetlip (Plectorhinchus diagrammus), yellow striped goatfish (Parupeneus chrysopleuron), pacific yellowtail emperor (Lethrinus atkinsoni, blue spotted grouper (Cephalopholis argus), and banded reef cod (Epinephelus fasciatus) were collected between July and August, 2012 in the vicinity of Yongxing Island, South China Sea contained decabromodiphenyl ether (plus 7 other polybrominated ether congeners) levels of 5.8-46 ng/g lw in the muscle(6).

Animal Concentrations:

Concentrations of this compound in animals.

>> Decabromodiphenyl ether was not detected in snapping turtle eggs collected June 2001 to June 2004 from 15 locations in the Great Lakes area (St Clair River, Detroit River, Wheatley Harbor, Hamilton, Bay of Quinte, Niagra, and St Lawrence, Canada, and St Lawrence in US)(1). Decabromodiphenyl ether was measured in 23 cats in 2005 to 2006 and found to be at highest concentration in pet cats eating mainly dry food followed by pet cats that ate a mix of dry and canned food and lowest in cats that eat mainly canned food(2). Decabromodiphenyl ether was detected at 17 ng/g lipid in 1 of 40 little owl (Athene noctua) eggs collected in Charleroi, Belgium 1998 and 2000(3). Decabromodiphenyl ether was found in Peregrine falcon eggs at <7-9, <20-430, and 28-190 ng/g lipid in captive, wild in southern Sweden, and northern Sweden, respectively, from eggs collected 1987-1999(4). Mean decabromodiphenyl ether concentrations of 20.35 and 40.80 ng/g were detected in juvenile and adult herring gulls (Larus argentatus) collected from the Hvaler Archipelage, Norway(5).

Average Daily Intake:

The average amount of the compound taken into the body through eating, drinking, or breathing.

>> Median daily intake of decabromodiphenyl ether to US infants (1-12 months old) ranges from 2.73-4.28 ng/day based on infant formula and cereals(1).

13. Disposal Considerations

Spillage Disposal

>> Personal protection: particulate filter respirator adapted to the airborne concentration of the substance. Sweep spilled substance into covered containers. If appropriate, moisten first to prevent dusting.

Disposal Methods

>> SRP: Recycle any unused portion of the material for its approved use or return it to the manufacturer or supplier. Ultimate disposal of the chemical must consider: the material's impact on air quality; potential migration in air, soil or water; effects on animal, aquatic and plant life; and conformance with environmental and public health regulations. If it is possible or reasonable use an alternative chemical product with less inherent propensity for occupational harm/injury/toxicity or environmental contamination.

>> Product: Offer surplus and non-recyclable solutions to a licensed disposal company. Contaminated packaging: Dispose of as unused product.

14. Transport Information

DOT

Decabromodiphenyl oxide

IATA

Decabromodiphenyl oxide

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15. Regulatory Information

Clean Water Act Requirements:

The Clean Water Act (CWA) of 1972 establishes the basic structure for regulating discharges of pollutants into the waters of the United States and regulating quality standards for surface waters. Under CWA, the U.S. Environmental Protection Agency (EPA) developed the Toxic Pollutant List (40 CFR Part 401.15) and the Priority Pollutant List (40 CFR Part 423, Appendix A). These lists are to be used by EPA and States to develop the Effluent Guidelines regulations and ensure water quality criteria and standards.

>> Toxic pollutant designated pursuant to section 307(a)(1) of the Federal Water Pollution Control Act and is subject to effluent limitations. /Haloethers/

TSCA Requirements:

This section provides information on requirements concerning this chemical under the Toxic Substances Control Act (TSCA) of 1976. TSCA provides EPA with authority to require reporting, record-keeping and testing requirements, and restrictions relating to chemical substances and/or mixtures. Certain substances are generally excluded from TSCA, including, among others, food, drugs, cosmetics and pesticides.

>> Section 8(a) of TSCA requires manufacturers of this chemical substance to report preliminary assessment information concerned with production, exposure, and use to EPA as cited in the preamble in 51 FR 41329. Effective date 1/11/90; Reporting date: 3/12/90.

Regulatory Information

The Australian Inventory of Industrial Chemicals

>> Chemical: Benzene, 1,1'-oxybis[2,3,4,5,6-pentabromo-

>> Specific Information Requirement: Obligations to provide information apply. You must tell us within 28 days if the circumstances of your importation or manufacture (introduction) are different to those in our assessment.

REACH Registered Substance

>> Status: Active Update: 13-06-2014 https://echa.europa.eu/registration-dossier/-/registered-dossier/14217

REACH Substances of Very High Concern (SVHC)

>> Substance: Bis(pentabromophenyl) ether (decabromodiphenyl ether) (DecaBDE)

>> EC: 214-604-9

>> Date of inclusion: >19-Dec-2012

>> Reason for inclusion: PBT (Article 57d); vPvB (Article 57e)

REACH List of substances subject to POPs Regulation (POPs)

>> Substance: Bis(pentabromophenyl) ether

>> EC: 214-604-9

>> Date of inclusion in the POPs Regulation: 20-Jun-2019

>> POPs Regulation Annex: Annex I, part A; Annex IV

16. Other Information

Toxic Combustion Products:

Toxic products (e.g., gases and vapors) produced from the combustion of this chemical.

>> Carbon oxides, Hydrogen bromide gas.

Other Safety Information

Chemical Assessment

>> PEC / SN / Other assessments - Decabromodiphenyl ether: Health and Environment

>> PEC / SN / Other assessments - Polybrominated flame retardants (PBFRs): Health and Environment

"The information provided is believed to be accurate but is not comprehensive and should be used as a reference. It reflects our current knowledge and is intended for safety guidance related to the product. This document does not constitute a warranty of the product's properties. Ionz is not responsible for any damages resulting from handling or contact with the product incorrectly."