2,2',4,4'-Tetrabromodiphenyl ether (BDE-47); CASRN 5436-43-1

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR 2,2',4,4'-TETRABROMODIPHENYL ETHER (BDE-47)

File First On-Line 06/30/2008

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	yes	06/30/2008
Inhalation RfC (I.B.)	qualitative discussion	06/30/2008
Carcinogenicity Assessment (II.)	yes	06/30/2008

I. HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

The congener 2,2',4,4'-tetrabromodiphenyl ether (BDE-47) has not been previously assessed in IRIS. A health assessment of the tetrabromodiphenyl ether homolog group (CASRN 40088-47-9) was previously entered on IRIS on 08/01/1990. Information was not available to derive an RfD or RfC or to assess the carcinogenic potential of the tetrabromodiphenyl ether homolog group.

I.A. REFERENCE DOSE (RfD) FOR ORAL EXPOSURE

Substance Name — 2,2',4,4'-Tetrabromodiphenyl ether (BDE-47)
CASRN — 5436-43-1
Section I.A. Last Revised — 06/30/2008

The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD is intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action. It is expressed in units of mg/kg-day. Please refer to the guidance documents at http://www.epa.gov/iris/backgrd.html for an elaboration of these concepts. Because RfDs can be derived for the noncarcinogenic health effects of substances that are also carcinogens, it is essential to refer to other sources of information concerning the carcinogenicity of this chemical substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Point of Departure*	UF	RfD
Neurobehavioral effects	$BMDL_{1SD}$: 0.35 mg/kg	3000	0.0001 mg/kg-day
Single dose gavage study in mice	BMD_{1SD}: 0.47 mg/kg		
Eriksson et al., 2001			

* Conversion Factors and Assumptions - $BMDL_{1SD}$ = 95% lower confidence limit on the maximum likelihood estimate of the dose corresponding to a change in the mean equal to one SD of the control mean. BMD_{1SD} = maximum likelihood estimate of the dose corresponding to a change in the mean equal to one SD of the control mean.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES

The only study suitable for dose-response assessment is the neurobehavioral study of Eriksson et al. (2001). In this study, male NMRI mice were administered on postnatal day (PND) 10 single oral doses of 0, 0.7, or 10.5 mg/kg of BDE-47 (>98% purity) in a fat emulsion. Spontaneous motor behavior was tested at ages 2 and 4 months in groups of eight male mice randomly selected from three to four different litters, and the mice were tested once only. Spontaneous motor behavior was tested for a 60-minute period divided into three 20-minute periods. Locomotion (horizontal movement), rearing (vertical movement), and total activity (all types of vibration within the test cage [i.e., those caused by mouse movement, shaking/tremors, and grooming]) were measured. In order to study time-dependent changes in

habituation (2 month-old vs. 4-month-old mice), data from the spontaneous motor behavior tests were used. An habituation ratio was calculated between the performance periods 40-60 minutes and 0-20 minutes for each of the three different variables: locomotion, rearing, and total activity. The habituation ratio was used to analyze alteration in habituation of 2-month-old and 4-month-old treated mice, within each treatment group, in comparison with their respective controls. Swim-maze performance, a measure of learning and memory ability, was tested in groups of 16-18 mice randomly selected from three to four different litters at age 5 months, given the high dose of BDE-47 (10.5 mg/kg). There were no clinical signs of dysfunction in the treated mice throughout the experimental period nor were there any significant deviations in body-weight gain in the BDE-47 treated mice compared with the vehicle-treated mice.

Control mice showed habituation (i.e., a decrease in locomotion, rearing, and total activity) in response to the diminishing novelty of the test chamber over the three 20-minute test periods. Data for the three spontaneous behavior variables (horizontal movement, vertical movement, and total activity) are only available in graphic form and could not be used for quantitative assessment (attempts to obtain numerical values and other information on the data from the authors were not successful). Numerical values, suitable for dose-response assessment, are only available for the habituation ratio. For all three spontaneous motor behavior variables (locomotion, rearing, and total activity), 2-month-old mice receiving 10.5 mg/kg BDE-47 displayed significantly less activity than controls during the first 20-minute period (hypoactivity) but were significantly more active than controls during the third 20-minute period (hyperactivity). The aberrations in spontaneous motor behavior were more pronounced in 4-month-old mice than in 2-month-old mice, indicating worsening with increasing age. In mice given 10.5 mg/kg BDE-47, the habituation capability was significantly reduced in 4-month-old mice compared with 2-month-old mice for all three variables (locomotion, rearing, and total activity). Performance of 5-month-old mice in the swim-maze learning/memory test, presented in graphic form only, was not affected at any dose. The no-observed-adverse-effect level (NOAEL) in this study was 0.7 mg/kg and the lowest-observed-adverse-effect level (LOAEL) was 10.5 mg/kg for changes in spontaneous motor behavior and decreased habituation capability in adult male mice, worsening with increasing age.

The RfD for BDE-47 was derived by applying the benchmark dose (BMD) approach to the data on habituation response to BDE-47 exposure collected by Eriksson et al. (2001). In the case of motor activity, there is no specific change that is generally regarded as indicative of an adverse response. In the absence of some idea of the level of response to consider adverse, the benchmark response (BMR) selected was a change in the mean equal to one SD from the control mean. Habituation ratios (based on the spontaneous behaviors of locomotion, rearing, and total activity) were modeled as continuous variables by fitting the linear, polynomial, and

power models. Habituation ratios for total activity in 2- and 4-month-old male mice were the most suitable endpoints for developing a point of departure (POD).

Of the BMDs and the BMD lower confidence limits (BMDLs) estimated from the continuous models that provided an adequate fit, the lowest BMD and BMDL were obtained by fitting a linear model to habituation ratios based on decreased total activity habituation in 4-month-old mice. The estimated BMD_{1SD} is 0.47 mg/kg, and the $BMDL_{1SD}$ is 0.35 mg/kg.

I.A.3. UNCERTAINTY FACTORS

UF = 3,000

A total uncertainty factor (UF) of 3,000 was applied: 10 for extrapolating animal data to humans (UF_A interspecies variability), 10 for susceptible human subpopulation (UF_H interhuman variability), 3 for extrapolating from a single-dose duration to a chronic exposure duration (UF_S), and 10 to account for a deficient database (UF_D).

A default interspecies UF_A of 10 was applied because the data were insufficient to characterize toxicokinetic and toxicodynamic differences between rodents and humans.

A default intraspecies UF_H of 10 was applied to account for variations in susceptibility within the human population (or interhuman variability). This factor accounts for humans who may be more sensitive than the general population to exposure to BDE-47.

A UF_S of 3 was used for extrapolating effects seen in a single-exposure neurodevelopmental study to a lifetime exposure. Exposure on PND 10 occurred during a period of rapid brain development in mice. Brain development does not continue at an equivalent rate across the lifespan and is more quiescent during adult life stages. There are a wide variety of brain structures that have very limited critical windows during development, particularly in early life. These critical windows translate to susceptible periods of exposure that are very short in duration. Toxicokinetic data suggest that a mouse urinary protein becomes functional at some time between PNDs 28 and 40, leading to a dramatic increase in BDE-47 urinary excretion, especially in males. This reduces the total body burden, including the levels of radiolabel reaching the brain 24 hours after dosing in older mice compared to young mice. These data suggest that neurodevelopmental risk in neonatal mice may be greater than at later ages because of the postnatal brain-growth spurt and coincident increased retention of BDE-47 and/or its metabolites. Thus, it is not necessary to make a 10-fold adjustment for exposure duration.

A UF_L for LOAEL-to-NOAEL extrapolation was not used because the Agency's current approach is to address this factor as one of the considerations in selecting a benchmark response for benchmark dose modeling. In this case, a change in the mean equal to 1 SD of the control mean was assumed to represent a minimal biologically significant change.

A UF_D of 10 was used to account for database uncertainty. The available oral database for BDE-47 lacks prenatal developmental neurotoxicity studies, reproductive toxicity studies, and standard chronic or subchronic studies of systemic toxicity.

Application of a total UF of 3,000 to the BMDL1SD of 0.35 mg/kg results in a reference dose for BDE-47 of 0.0001 mg/kg-day.

I.A.4. ADDITIONAL STUDIES/COMMENTS

Richardson et al. (2008) administered a single oral gavage (in corn oil) dose of 3, 10, or 100 mg/kg-day BDE-47 (purity >98%) to female C57BL/6 mice for 4 days. Serum T_4 was decreased (approx. 43%) in the 100 mg/kg-day dose group compared with the controls. There were no effects on liver weight and serum T_4 at the two lower doses of BDE-47. In examining UGT induction, the authors showed increases in UGT1A1, UGT2B5, and UGT1A7 expression at 100 mg/kg-day. UGT1A7 expression was increased at 10 mg/kg-day. The changes in UGT isoform expression correlated with the observed T_4 decreases. However, in contrast to the observed changes in UGT mRNA expression, BDE-47 treatment did not change hepatic T_4-UGT enzyme activity. The authors suggested that the T_4-UGT enzyme assay was not adequately sensitive to measure changes in activity of individual UGT isoforms.

Significant correlations were observed between decreases in T_4 and increased PROD activity ($R^2 = 0.57$, $p < 0.0001$) and T_4 and increased CYP-2B10 expression ($R^2 = 0.44$, $p < 0.005$). A significant increase (47%) in the expression of a major glucuronide transporter, hepatic MRP3 mRNA at 100 mg/kg-day BDE-47 was significantly correlated with T_4 decreases ($R^2 = 0.46$, $p < 0.001$). Significant, dose-dependent decreases in hepatic MDR 1A (but not MDR 1B), a transporter for glucuronides and thyroid hormones, were observed at all doses of BDE-47; however, these decreases did not correlate with the decreases in T_4 ($R^2 = 0.17$, $p = 0.08$). A thyroid hormone uptake transporter, MCT8, was significantly decreased (0.8-fold) at 100 mg/kg-day. The authors suggested that MCT8 may play a role in thyroid hormone changes, although the decrease in MCT8 mRNA expression did not correlate with T_4 decreases ($R^2 = 0.02$, $p = 0.56$). A major rodent transport protein in the serum, transthyretin, was significantly decreased at 100 mg/kg-day and correlated with the decrease in T_4 ($R^2 = 0.61$, $p < 0.0001$). Richardson et al. (2008) noted that the parallel decreases in transthyretin and T_4 support the hypothesis that BDE-47 interferes with transthyretin transport of T_4.

Despite the possibility of thyroid hormone involvement in the neurodevelopmental impact of BDE-47 on the habituation response in male mice exposed to a single dose on PND 10, there are no mode-of-action data that link thyroid hormones to the observations of Eriksson et al. (2001). Thyroid hormone levels and behavioral activity were not comeasured in the study in mice of Eriksson et al. (2001).

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Low
Data Base -- Low
RfD -- Low

Confidence in the principal study is low because the study was conducted in male mice only, the protocol was unique and did not conform to health effects test guidelines for neurotoxicity, the dosing regimen did not include gestation and lactation exposure, several pups per litter were used for the behavioral testing, and only single doses were given. Confidence in the database is low because it lacks prenatal developmental neurotoxicity studies, reproductive toxicity studies, and standard chronic or subchronic studies of systemic toxicity. As a result, the overall confidence in the RfD assessment is low.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document — U.S. EPA (2008).

This document has been reviewed by EPA scientists, interagency reviewers from other federal agencies, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the T*oxicological Review of 2,2',4,4'-Tetrabromodiphenyl Ether (BDE-47)* (U.S. EPA, 2008). ***To review this appendix, exit to the toxicological review, Appendix A, Summary Of External Peer Review And Public Comments And Disposition (PDF)***.

Agency Completion Date -- 06/30/2008

I.A.7. EPA CONTACTS

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

I.B. REFERENCE CONCENTRATION (RfC) FOR INHALATION EXPOSURE

Substance Name — 2,2',4,4'-Tetrabromodiphenyl ether (BDE-47)
CASRN — 5436-43-1
Section I.B. Last Revised — 06/30/2008

The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC considers toxic effects for both the respiratory system (portal of entry) and for effects peripheral to the respiratory system (extrarespiratory effects). The inhalation RfC (generally expressed in units of mg/m^3) is analogous to the oral RfD and is similarly intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action.

Inhalation RfCs are derived according to *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994). Because RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens, it is essential to refer to other sources of information concerning the carcinogenicity of this chemical substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.B.1. INHALATION RfC SUMMARY

No data are available for deriving a reference concentration for BDE-47.

I.B.2. PRINCIPAL AND SUPPORTING STUDIES

Not applicable.

I.B.3. Uncertainty Factors

Not applicable.

I.B.4. Additional Studies/Comments

Not applicable.

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

I.B.5. CONFIDENCE IN THE INHALATION RfC

Not applicable.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- U.S. EPA (2008)

This document has been reviewed by EPA scientists, interagency reviewers from other federal agencies, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of 2,2',4,4'-Tetrabromodiphenyl Ether (BDE-47)* (U.S. EPA, 2008). ***To review this appendix, exit to the toxicological review, Appendix A, Summary Of External Peer Review And Public Comments And Disposition (PDF).***

Agency Completion Date -- 06/30/2008

I.B.7. EPA CONTACTS

PPlease contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name — 2,2',4,4'-Tetrabromodiphenyl ether (BDE-47)
CASRN — 5436-43-1
Section II. Last Revised — 06/30/2008

This section provides information on three aspects of the carcinogenic assessment for the substance in question: the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral and inhalation exposure. Users are referred to Section I of this file for information on long-term toxic effects other than carcinogenicity.

The rationale and methods used to develop the carcinogenicity information in IRIS are described in the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a) and the *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (U.S. EPA, 2005b). The quantitative risk estimates are derived from the application of a low-dose extrapolation procedure, and are presented in two ways to better facilitate their use. First, route-specific risk values are presented. The "oral slope factor" is a plausible upper bound on the estimate of risk per mg/kg-day of oral exposure. Similarly, a "unit risk" is a plausible upper bound on the estimate of risk per unit of concentration, either per µg/L drinking water (see Section II.B.1.) or per µg/m^3 air breathed (see Section II.C.1.). Second, the estimated concentration of the chemical substance in drinking water or air when associated with cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000 is also provided.

II.A. EVIDENCE FOR HUMAN CARCINOGENICITY

Epidemiologic studies of exposure to BDE-47 and cancer occurrence in humans are not available. Animal chronic toxicity/carcinogenicity studies have not been conducted with BDE 47.

II.A.1. WEIGHT-OF-EVIDENCE CHARACTERIZATION

Under the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a), there is "inadequate information to assess the carcinogenic potential" of 2,2',4,4'-tetrabromodiphenyl ether (BDE-47).

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

II.A.2. HUMAN CARCINOGENICITY DATA

Not applicable.

II.A.3. ANIMAL CARCINOGENICITY DATA

Not applicable.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

BDE-47 demonstrated low or no recombinogenic potential in two in vitro Chinese hamster cell assays. Helleday et al. (1999) examined the effects of BDE-47 at concentrations of 0-40 µg/mL in two in vitro V79 Chinese hamster cell-line assays, Sp5 and SDP8, for intragenic recombination at an endogenous locus in mammalian cells. Results from this study indicate that BDE-47 is weakly recombinogenic in the SPD8 cell line assay with up to a 1.8-fold increase at 40 µg/mL but not recombinogenic in the Sp5 cell line. This difference in assay results may be due to different levels of sensitivity and mechanisms among the Sp5 and SPD8 cell lines. Based on these results, BDE-47 appears to be weakly mutagenic at best in mammalian cells.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

II.B.1. SUMMARY OF RISK ESTIMATES

Not applicable.

II.B.2. DOSE-RESPONSE DATA

Not applicable.

II.B.3. ADDITIONAL COMMENTS

Not applicable.

II.B.4. DISCUSSION OF CONFIDENCE

Not applicable.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

II.C.1. SUMMARY OF RISK ESTIMATES

Not applicable.

II.C.2. DOSE-RESPONSE DATA

Not applicable.

II.C.3. ADDITIONAL COMMENTS

Not applicable.

II.C.4. DISCUSSION OF CONFIDENCE

Not applicable.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA (2008)

This document has been reviewed by EPA scientists, interagency reviewers from other federal agencies, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of 2,2',4,4'-Tetrabromodiphenyl Ether (BDE-47)* (U.S. EPA, 2008). ***To review this appendix, exit to the toxicological review, Appendix A, Summary Of External Peer Review And Public Comments And Disposition (PDF)***.

II.D.2. EPA REVIEW

Agency Completion Date -- 06/30/2008

II.D.3. EPA CONTACTS

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — 2,2',4,4'-Tetrabromodiphenyl ether (BDE-47)
CASRN — 5436-43-1

VI.A. ORAL RfD REFERENCES

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Hallgren, S; Sinjari, T; Hakansson, H; et al. (2001) Effects of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) on thyroid hormone and vitamin A levels in rats and mice. Arch Toxicol 75(4):200-208.

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VI.B. INHALATION RfC REFERENCES

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VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Helleday, T; Tuominen, KL; Bergman, A; et al. (1999) Brominated flame retardants induce intragenic recombination in mammalian cells. Mutat Res 439:137-147.

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VII. REVISION HISTORY

Substance Name — 2,2',4,4'-Tetrabromodiphenyl ether (BDE-47)
CASRN — 5436-43-1
File First On-Line - 06/30/2008

Date	Section	Description
06/30/2008	I., II.	RfD, RfC, and cancer assessment first on line

VIII. SYNONYMS

Substance Name — 2,2',4,4'-Tetrabromodiphenyl ether (BDE-47)
CASRN — 5436-43-1
Section VIII. Last Revised — 06/30/2008

- Benzene, 1,1'-oxybis(2,4-dibromo)-
- 2,2',4,4'-Tetrabromodiphenyl ether
- PBDE 47
- BDE-47