Voluntary Children's Chemical Evaluation Program Pilot (VCCEPP)

Tier 1 Assessment of the Potential Health Risks to Children Associated With Exposure to the Commercial Octabromodiphenyl Ether Product

CAS No. 32536-52-0

Prepared by:

ENVIRON International Corporation

6001 Shellmound Street
Emeryville, California 94608 USA

Prepared for:

Great Lakes Chemical Corporation

One Great Lakes Boulevard
West Lafayette, Indiana 47906 USA

April 21, 2003
03-10607A

Forward

Great Lakes Chemical Corporation is pleased to present to the United States Environmental Protection Agency (USEPA) this Tier 1 assessment report. This work, which was conducted by ENVIRON International Corporation, represents our commitment to participate in the Voluntary Children's Chemical Evaluation Program Pilot (VCCEPP) as sponsor of the children's health risk assessment of the commercial octabromodiphenyl ether (octaBDE) product.

Great Lakes Chemical Corporation's participation in the VCCEPP is consistent with our commitment to fulfill our responsibilities as an industry leader and corporate citizen. Great Lakes Chemical Corporation fully endorses the principles of Responsible Care® set forth by the American Chemistry Council and has translated these codes into a course of action and a way of life. The company and its employees work proactively to develop environmentally superior products and services, improve manufacturing operations and create safe working and living conditions for our employees and the communities that surround our facilities.

April 21, 2003

Great Lakes Chemical Corporation
West Lafayette, Indiana, USA

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Acronyms and Abbreviations

µg	microgram
µmol	micromole
ABS	acrylonitrile-butadiene styrene
Agency	United States Environmental Protection Agency
AOP	Atmospheric Oxidation Program
BDE	brominated diphenyl ether
BFR	brominated flame retardant
BMD	benchmark dose
BMDL	lower confidence limit on the benchmark dose
BMDL 5	benchmark dose associated with 5% increase in response over background
BMDL10	benchmark dose associated with 10% increase in response over background
BW or bw	body weight
^{o} C	degree Celsius
CDI	chronic daily intake
cfg	see for example
cm^3 $molecule^{-1}s^{-1}$	cubic centimeters per molecule per second
CPU	central processing unit
CSF	cancer slope factor
d	day
DNA	deoxyribonucleic acid
dw	dry weight
ECB	European Chemicals Bureau
ENVIRON	ENVIRON International Corporation
EPIWIN	Estimation Program Interface for Windows
EPN	O-ethyl-O-p-nitrophenyl phenylphophonothioate
EQC	Equilibrium Concentration
EROD	ethoxyresorufin-odeethylase
EU	European Union
^{o} F	degree Fahrenheit
FPUF	flexible polyurethane foam
fw	fresh weight
g	gram
GC-MS	gas chromatography-mass spectroscopy
GLCC	Great Lakes Chemical Corporation
GLP	good laboratory practice
HI	hazard index
HIPS	high impact polystyrene
HPV	high production volume
HQ	hazard quotient
hr	hour
IRDC	International Research and Development Corporation
IRIS	Integrated Risk Information System
IUCLID	International Uniform Chemical Information Database
kg	kilogram
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter

LD_{50}	lethal dose at which 50% of animals die
lw	lipid weight
m^3	cubic meter
mg	milligram
mg/day	milligram per day
mg/kg	milligram per kilogram
mg/kg-day	milligram per kilogram per day
mL	milliliter
mmHg	millimeter of mercury
molecules/cm^3	molecules per cubic centimeter
MSDS	material safety data sheet
N/A or NA	not applicable
NCEA	National Center for Environmental Assessment
NFPA	National Fire Protection Agency
ng	nanogram
nmol	nanomole
NOAEL	no observable adverse effect level
NOEL	no observable effect level
NRC	National Research Council
OPPTS	Office of Pesticide Programs and Toxic Substances
OECD	Organization of European Cooperation and Development
Pa	Pascal
PBBE	poybromobiphenyl ether
PBBO	polybromobiphenyl oxide
PBDPE	polybromodiphenyl ether
PBDPO	polybromodiphenyl oxide
PBDE	polybrominated diphenyl ether
PBT	polybutylene terephthalate
PCB	polychlorinated biphenyl
PCV	packed cell volume
pg	picogram
pg/cm^2	picogram per square centimeter
ppb	part per billion
ppm	part per million
PROD	pentoxyresorufin-o-deethylase
Program	Voluntary Children's Chemical Evaluation Program Pilot
RfD	reference dose
RME	reasonable maximum exposure
SGOT	serum glutamic oxalacetic transaminase
SGPT	serum glutamic pyruvic transaminase
SAMS	Surface Area Modeling System
T_3	3,3',5-triiodothyronine
T_4	3,3',5,5'-tetraiodothyronine, thyroxine
TSH	thyroid stimulating hormone
TV	toxicity value
UCL_{95}	95 percent upper confidence limit on the mean
UDPGT	uridinediphosphate-glucuronosyltransferase
U. S.	United States of America
USCPSC	United States Consumer Product Safety Commission
USEPA	United States Environmental Protection Agency
VCCEPP	Voluntary Children's Chemical Evaluation Program Pilot
WEL	Worker Exposure Limit

WHO	World Health Organization
ww	wet weight

Definition of Chemical Terms

The following acronyms are used throughout this document:

Commercial octaBDE product	Refers only to the commercial octabrominated diphenyl ether product
Tetra-, penta-, hexa-, hepta-, octa-, nona-, deca- BDE	Refers only to the congener group containing either 4, 5, 6, 7, 8, 9, or 10 (respectively) bromine molecules on the diphenyl ether structure
BDE - ### deca	Refers to one of 209 specific polybrominated diphenyl ether compounds using a numbering system similar to that used for polychlorinated biphenyls

Executive Summary

Introduction

A Tier 1 assessment of the potential health risks to children and prospective parents associated with exposure to the commercial octabromodiphenyl ether (octaBDE) product (CAS No. 32536-52-0) was conducted by Great Lakes Chemical Corporation (GLCC) in accordance with United States Environmental Protection Agency (USEPA or Agency) Voluntary Children's Chemical Evaluation Program Pilot (VCCEPP; Federal Register Vol. 65, No. 248, pp. 81699-81718). The commercial octaBDE product is a brominated flame retardant produced at one chemical plant in Arkansas by GLCC and used almost exclusively by the plastic industries as an additive in the manufacture of acrylonitrile butadiene-styrene (ABS) polymers used in casings for computers and other electronic equipment.

This Tier 1 assessment is the first of three tiers in the VCCEPP and includes four screening-level evaluations – hazard, exposure, risk, and data needs. Tiers 2 and 3 of the VCCEPP more fully characterize the potential for exposure and the associated health risks posed to children. The purpose of the VCCEPP is to provide USEPA with the means to understand the potential health risks to children and prospective parents associated with exposure to chemicals. The VCCEPP Tier 1 assessment of the commercial octaBDE product addresses six basic objectives:

1. Review and summarize the available animal bioassays and epidemiology studies reported in the scientific literature and identify potential human health endpoints and toxicity benchmarks relevant to children and prospective parents;

2. Understand how different uses of the commercial octaBDE product in U.S. commerce may result in exposures to children and prospective parents;

3. Determine the plausible pathways by which children might come into contact with the commercial octaBDE product, and assess whether these pathways may result in potentially meaningful and relevant exposures to children and prospective parents;

4. Estimate child and adult exposures to the commercial octaBDE product for potentially meaningful and relevant situations using available screening-level approaches such as the use of predictive models and/or direct environmental measurements, and including assessment of the potential for aggregate exposure (whereby an individual child or adult may be exposed simultaneously to the commercial octaBDE product through more than one pathway);

5. Combine the findings from the hazard assessment with the results of the exposure assessment to derive qualitative and/or quantitative conclusions regarding the potential health risks to children and prospective parents; and,

6. Identify additional hazard and/or exposure information needed to reduce uncertainties surrounding the potential risks to children and, where relevant, to prospective parents.

Hazard Assessment

The commercial octaBDE product manufactured by GLCC is composed of a mixture of primarily pentaBDE (approximately 0.5%), hexaBDE (approximately 12%), heptaBDE (approximately 45%), octaBDE (approximately 33%), nonaBDE (approximately 10%), and decaBDE (approximately 1%) congeners (IUCLID, see Appendix III). At present, GLCC is the sole manufacturer of commercial octaBDE product in the United States and currently produces one formulation, Great Lakes DE-79™, where the number indicates the percent total bromine by weight in the product.

Commercial octaBDE product is produced by the direct bromination of diphenyl ether using a Friedel-Crafts catalyst. The commercial octaBDE product is characterized as an off-white to tan powder or flaked material. The commercial octaBDE product is not readily biodegradable. Volatilization of the commercial octaBDE product is minimal because low vapor pressures have been estimated or measured for the primary constituents. The main brominated diphenyl ether (BDE) constituents of the commercial octaBDE product are relatively immobile in soil and unlikely to leach into groundwater. There is almost no data in the literature describing the bioavailability of commercial octaBDE product or its constituents in either animals or humans. Based on the limited animal data, it is conservatively estimated that 50% and 3% of the predicted exposure concentrations of the commercial octaBDE product are absorbed by the oral and dermal routes of exposure, respectively. Inhalation absorption of the commercial mixture is assumed to be 75%.

The available toxicology data suggest that the commercial octaBDE product is not acutely toxic to humans or animals by the oral, dermal, or inhalation routes of exposure. It does not induce skin sensitization in guinea pigs, and has not been determined to be genotoxic in the Ames *Salmonella*, *Saccharomyces cerevisia* or in human lymphocytes *in vitro*. Among the chronic and subchronic toxicity studies available for the commercial octaBDE product, the most prominently observed endpoints in animal bioassays are in the liver and include induction of enzymes that function in xenobiotic metabolism and microscopic observations characteristic of adaptive responses to enzyme induction including increased liver weights and microscopic changes, such as increased size of the hepatocytes and cytoplasm described as “ground glass” in appearance. Disruption of thyroid hormone (3,3’,5,5’-tetraiodothyronine (T_4)) levels have been reported, and developmental toxicity studies in rats showed decreases in maternal and fetal body weights.

Toxicity values used in the Tier 1 assessment are summarized in Table ES-1. Theoretical exposures to children at different ages and prospective parents were evaluated by comparing the hypothetical daily intakes calculated using screening-level exposure models in the exposure assessment to toxicity threshold values developed to evaluate potential developmental effects and

the potential for thyroid hormone disruption. In the absence of data from laboratory animal models or humans, cancer was not included as an endpoint in this Tier 1 assessment. Liver enzyme induction, which was identified as the basis for an oral non-cancer reference dose by USEPA in 1980 (0.003 mg/kg/day), was not considered a relevant human health endpoint. There is no evidence to suggest that the alteration of liver enzyme function associated with exposure to the commercial octaBDE product will result in an adverse effect on reproduction in humans. However, this endpoint and the USEPA (2003) reference dose were used in the assessment to provide an upper-bound estimate of the potential health hazard to children.

The reproductive/developmental toxicity value was derived from the NOAEL for maternal toxicity, evidenced by decreased in fetal body weight observed in rodent studies reported by WIL Research (1986a). The application of uncertainty factors for inter- and intraspecies extrapolation to the BMDL5 calculated from benchmark dose modeling (8.7 mg/kg/day) resulted in a toxicity benchmark value of 0.09 mg/kg/day. The thyroid has been identified as a target tissue in subacute and subchronic rodent studies. Benchmark doses for potential effects on the thyroid were derived using data from Zhou (2001), WIL Research (2001), and IRDC (1978a). The BMDL10s derived from different studies ranged from 3.3 mg/kg/day in males and 1.7 mg/kg/day in females to 76 mg/kg/day in either sex. For estimates of intake primarily by the oral route, the BMDL10 derived from Zhou et al. (2001) was used in conjunction with the application of a 100-fold uncertainty factor. These findings are likely relevant for human health assessment, especially in children and differences in sensitivity to small changes in thyroid hormone levels should be considered quantitatively. The resulting benchmark toxicity value, 0.09 mg/kg/day, was used in this Tier 1 assessment.

Table ES-1. Human health endpoints and toxicity values used in the Tier 1 assessment

Potential Human Health Endpoint	Toxicity Value	Relevant Study
Reproductive/ Developmental effects: Change in maternal or fetal body weight	0.09 mg/kg/day	WIL Research (1986a)
Thyroid effects: Disruption of T_4 homeostasis	0.09 mg/kg/day	Zhou et al. (2001)
Liver enzyme induction	0.003 mg/kg/day	USEPA Reference Dose (RfD) for the commercial octaBDE product based on Carlson (1980b)

Exposure Assessment

The commercial octaBDE product is used almost exclusively to flame retard ABS plastics used in the plastic industries to manufacture casings for computers and electronic equipment. It is unknown what fraction of ABS plastics produced annually in the United States use the commercial octaBDE product as a flame retardant additive. According to the limited data provided by companies that purchase the commercial octaBDE product, ABS plastics contain

approximately 12-15% flame retardant, a proportion of which is the commercial octaBDE product.

Few environmental data describing levels of the commercial octaBDE product in the United States are available in the literature. The available data reported in the scientific literature typically describe the occurrence and levels of one or more specific BDE congeners. A review of the scientific literature suggests that the predominant BDEs reported in ambient air, soil, sediment, and biota are BDE-28, BDE-47, BDE-66, BDE-85, BDE-99, BDE-100, BDE-153, BDE-154, BDE-183, and BDE-209. With the exception of BDE-28 and BDE-47, these BDEs occur in the commercial octaBDE product. The current understanding of BDE levels in human breast milk is based primarily on the results of monitoring studies reported during the past decade in Sweden. Information from the published literature indicate that tri- to only heptaBDE congeners have been detected in human tissue and breast milk. At present, limited data on the levels of BDEs in breast milk from women in North America are available; among the few data reported, BDE levels in North American women are generally higher than BDE levels in breast milk reported in women from Sweden and Finland.

The populations that are potentially exposed to the commercial octaBDE product include primary product manufacturers, plastic pellet manufacturers, end-product manufacturers (i.e., casing and equipment manufacturers), equipment distributors, equipment recyclers, and adults and children in the indoor home/school/office environments, as well as ambient environment. The entire population of workers engaged in primary manufacturing of the commercial octaBDE product is fewer than 100 persons employed at one chemical plant located in Arkansas and are typically male workers ranging in age from 20-45 years old. The population of workers engaged in secondary, or chain-of-commerce, activities involving the use of the commercial octaBDE product as an additive in the manufacture of plastic casings for electronic equipment is large, and cannot be estimated with any degree of accuracy. Similarly, the population of children and adult men and women potentially exposed to ambient levels of BDEs in the environment and through contact with consumer products containing the commercial octaBDE product (primarily computers and monitors) in the indoor home/school/office environment also is large, and cannot be estimated with any degree of accuracy.

Three scenarios were evaluated in the Tier 1 exposure assessment of children and prospective parents: exposures in the workplace, exposures in the indoor home/school/office environment, and exposures associated with the ambient environment (e.g., via the diet, both direct and indirect contact with soil, and particulates in air). The major sources of worker exposures during the manufacture of plastic casings are likely to be associated with handling of the commercial octaBDE product prior to mixing with other ingredients; volatilization of BDEs from casings during the mixing and moulding processes; and, handling of residues during the cleaning of moulding equipment. The potential exposure routes for workers engaged in plastic casing manufacturing activities in the United States include inhalation of vapors originating from primary product or manufactured plastic casings, dermal contact with the primary product or

manufactured plastic casings, and incidental ingestion via hand-to-mouth contact. In addition to direct exposures, two indirect exposure pathways involving dirty laundry and indoor floors in worker houses also were considered as plausible exposure scenarios. Workers' children also were evaluated for these last two pathways.

The potential exposure routes evaluated for children and adults in the indoor home/school/office environment in the United States included inhalation of respirable particulates, ingestion of particulates inhaled and swallowed, dermal contact with indoor dust, incidental ingestion by young children mouthing plastic surfaces, and incidental ingestion of indoor dust via hand-to-mouth contact. The potential exposure routes evaluated for children and adults associated with ambient levels in the United States environment included inhalation of respirable particulates in ambient air, ingestion of particulates inhaled and swallowed, dermal contact with outdoor soil, incidental ingestion of outdoor soil via hand-to-mouth contact; ingestion of meat, dairy, and vegetable food products, ingestion of recreationally caught fish, and ingestion of human breast milk by infants.

Risk Assessment

A summary of exposure pathways in each of the three scenarios for which theoretical exposures to children and prospective parents resulted in non-cancer hazard indices greater than or less than a value of one is presented in Table ES-2. A hazard index greater than a value of one suggests that the theoretical level of exposure calculated in the screening-level Tier 1 model for a particular exposure pathway and receptor group exceeds one or more of the three toxicity values for the non-cancer human health endpoints indicated in Table ES-1. With the exception of prospective parents engaged in primary product production, ABS plastic production, and chain-of-commerce manufacturing activities, hypothetical exposures to the commercial octaBDE product did not result in levels above screening toxicity benchmark values. Theoretical exposures to the commercial octaBDE product by children did not result in levels that exceeded screening toxicity benchmark values. For prospective parents, hypothetical exposures to workers engaged in primary production or chain-of-commerce manufacturing activities resulted in levels that exceeded the screening toxicity benchmark for liver enzyme induction. This benchmark, however, is not considered relevant and was used to provide an upper-bound estimate of the potential health hazard. There is no evidence to suggest that the alteration of liver enzyme function associated with exposure to the commercial octaBDE product will result in an adverse effect on reproduction in humans.

Table ES-2. Summary of exposure pathways considered in the Tier 1 assessment associated with a theoretical non-cancer hazard index greater than one for one or more of the three non-cancer health endpoints included in the screening-level exposure assessment

Exposure Pathway	Receptor Group	Non-Cancer Endpoint with a Hazard Index Greater Than 1 [1]
Scenario #1 - Workplace Exposures for Adults		
Inhalation of Respirable Particulate	▪ Primary Production Workers ▪ Chain of Commerce Workers	+, Liver enzyme induction
Dermal Contact	- none -	- none -
Incidental Ingestion via Hand to Mouth Contact	- none -	- none -
Dermal Contact with Dirty Laundry	- none -	- none -
Incidental Ingestion via Hand to Mouth Contact with Dirty Laundry	- none -	- none -
Dermal Contact with Dirty Floors	- none -	- none -
Incidental Ingestion of Dust from Dirty Floors	- none -	- none -
Scenario #1 – Exposures for Worker's Children		
Dermal Contact with Floor Surfaces at Home	- none -	- none -
Incidental Ingestion via Hand to Mouth Contact with Floor Surfaces at Home	- none -	- none -
Scenario #2 – Home Exposure		
Inhalation of Respirable Particulate	- none -	- none -
Ingestion of Particles Inhaled and Swallowed	- none -	- none -
Dermal Contact	- none -	- none -
Incidental Ingestion via Hand to Mouth Contact	- none -	- none -
Ingestion from Mouthing a Cushion	- none -	- none -
Scenario #2 - School and Office Exposures		
Inhalation of Respirable Particulate	- none -	- none -
Ingestion of Particles Inhaled and Swallowed	- none -	- none -
Dermal Contact	- none -	- none -
Incidental Ingestion via Hand to Mouth Contact	- none -	- none -
Scenario #3 – Ambient Environment Exposures		
Inhalation of Respirable Particulate	- none -	- none -
Ingestion of Particles Inhaled and Swallowed	- none -	- none -
Dermal Contact	- none -	- none -
Incidental Ingestion via Hand to Mouth Contact	- none -	- none -
Fish Consumption	- none -	- none -
Meat Consumption	- none -	- none -
Dairy Consumption	- none -	- none -
Fats & Oil Consumption	- none -	- none -

Exposure Pathway	Receptor Group	Non-Cancer Endpoint with a Hazard Index Greater Than 1 [1]
Egg Consumption	- none -	- none -
Vegetable Consumption	- none -	- none -
Breast Milk Consumption	- none -	- none -

[1]. A "+" indicates that the theoretical dose predicted by the screening-level exposure models resulted in a hazard index (HI) greater than 1 for the non-cancer health endpoint and receptor noted in the table. Conversely, a "- none - " indicates that the calculated HI was less than 1 for all non-cancer health endpoints and receptors evaluated in the Tier 1 assessment.

Hazard indices for children, based on aggregate exposures to the commercial octaBDE product from all three exposure scenarios (i.e., worker's children, indoor home and school environments, and ambient environment) are summarized in Table ES-3. Aggregate exposures to children within different age groups were calculated by summing the theoretical daily intakes associated with exposure to the commercial octaBDE product in the indoor home, school, and ambient environment. For each age group, hazard indices were below a value of one. In addition, the theoretical daily intakes indicate that children in the age groups of <1 year and 1-2 years receive the highest exposure to the commercial octaBDE product. The results of the screening-level Tier 1 assessment indicated that the highest hypothetical exposures among children were associated with the ambient environmental exposures, specifically consumption of different foods by the <1 year old child, 1-2 year old child, and the 3-5 year old child.

Table ES-3. Aggregate theoretical exposure to the commercial octaBDE product by children associated with hypothetical exposures in a worker's house, in the indoor home/school, and ambient environments and associated hazard indices for three health endpoints

Receptor	Aggregate Total Theoretical Chronic Daily Intake [1]	Hazard Index		
		Comparison to Thyroid Effects Benchmark [2]	Comparison to Developmental Effects Benchmark [3]	Comparison to Liver Enzyme Induction Effects Benchmark [4]
	mg/kg/day	unitless	unitless	unitless
<1 yr Child	1.4E-04	0.002	0.002	0.05
1-2 yr Child	8.2E-05	0.0009	0.0009	0.03
3-5 yr Child	7.4E-05	0.0008	0.0008	0.02
6-8 yr Child	6.2E-05	0.0007	0.0007	0.02
9-11 yr Male Child	5.7E-05	0.0006	0.0006	0.02
9-11 yr Female Child	5.7E-05	0.0006	0.0006	0.02
12-14 yr Male Child	5.2E-05	0.0006	0.0006	0.02
12-14 yr Female Child	5.0E-05	0.0006	0.0006	0.02
15-18 yr Male Child	4.6E-05	0.0005	0.0005	0.02
15-18 yr Female Child	4.7E-05	0.0005	0.0005	0.02

[1]. Aggregate exposures to children were calculated by summing the theoretical total CDIs calculated for children hypothetically exposed to the commercial product in a worker home, in the home/school/office

environments, and ambient environment. This represents the highest level of total exposure developed in the Tier 1 screening-level assessment.

[2]. The benchmark used to evaluate the potential for thyroid effects is based on disruption of T_4 homeostasis observed in the Zhou et al. (2001) rodent bioassays. The no-effect level was extrapolated to humans using a 100-fold safety factor.

[3]. The benchmark used to evaluate the potential for developmental effects is based on benchmark modeling using fetal rodent pup body weight data from WIL Research (1986a) bioassays. The calculated BMDL was extrapolated to humans using a 100-fold uncertainty factor.

[4]. The benchmark used to evaluate the potential for liver enzyme induction is based on the Carlson (1980b) animal bioassay used by USEPA to derive the current RfD listed in IRIS (USEPA, 2003). It is not considered relevant but is provided to represent an upper-bound estimate of potential hazard.

A similar approach was used to evaluate aggregate exposures to the commercial octaBDE product in prospective parents. Table ES-4 summarizes hazard indices for adult prospective male and female parents based on aggregate exposures to the commercial octaBDE product from all three exposure scenarios (i.e., workplace, home/school/office environment, and ambient environment). Hazard indices were calculated in a similar manner to that described for children in Table ES-3. Aggregate exposures to prospective parents were calculated by summing the theoretical daily intake associated with each exposure pathway evaluated in each of the three hypothetical exposure scenarios. The hazard index associated with the aggregate total theoretical daily intake was calculated by summing the hazard indices associated with each of the three exposure scenarios included in the Tier 1 assessment. The results shown in Table ES-4 demonstrate that the exposure to the commercial octaBDE product via all plausible pathways and exposure scenarios did not result in a level of exposure that exceeds relevant screening toxicity benchmark values for thyroid or developmental effects. Although, the toxicity benchmark for liver enzyme induction was exceeded for workplace exposures, this benchmark is not considered a relevant human health endpoint based on more current data (see section 2.4). In addition, the more likely workplace exposure is 33-fold lower when reasonable industrial hygiene measures are assumed.

Table ES-4. Aggregate theoretical exposure to the commercial octaBDE product by adults associated with hypothetical exposures in the workplace, indoor home/school/office, and ambient environment and the associated hazard indices for three benchmarks

Hypothetical Exposure Scenario	Aggregate Total Theoretical Chronic Daily Intake	Hazard Index		
		Comparison to Thyroid Effects Benchmark [1]	Comparison to Developmental Effects Benchmark [2]	Comparison to Liver Enzyme Induction Effects Benchmark [3]
	mg/kg-day	unitless	unitless	unitless
Prospective Parent, Female				
Workplace Scenario [4]	0.04	0.5	0.5	14
Home, School, and Office Scenario [5]	3.4E-07	0.000004	0.000004	0.0001
Ambient Environment Scenario [6]	1.2E-05	0.0001	0.0001	0.004
Recreational Freshwater Fishing Scenario [7]	9.3E-06	0.0001	0.0001	0.003
SUM	**0.04**	**0.5**	**0.5**	**14**

Prospective Parent, Male				
Workplace Scenario [4]	0.04	0.4	0.4	12
Home, School, and Office Scenario [5]	3.7E-07	0.000004	0.000004	0.0001
Ambient Environment Scenario [6]	1.1E-05	0.0001	0.0001	0.003
Recreational Freshwater Fishing Scenario [7]	7.8E-06	0.00009	0.00009	0.003
SUM	**0.04**	**0.4**	**0.4**	**12**

[1]. The benchmark used to evaluate the potential for thyroid effects is based on disruption of T_4 homeostasis observed in the Zhou et al. (2001) rodent bioassays. The no-effect level was extrapolated to humans using a 100-fold safety factor.
[2]. The benchmark used to evaluate the potential for developmental effects is based on benchmark modeling using fetal rodent pup body weight data from WIL Research (1986a) bioassays. The calculated BMDL was extrapolated to humans using a 100-fold uncertainty factor.
[3]. The benchmark used to evaluate the potential for liver enzyme induction is based on the Carlson (1980b) animal bioassay used by USEPA to derive the current RfD listed in IRIS (USEPA, 2003). It is not considered relevant but is provided to represent an upper-bound estimate of potential hazard.
[4]. The theoretical highest exposed adult in the workplace scenario is associated with the primary production grinder and packager of the commercial octaBDE product.
[5]. The theoretical exposures in the home, office, and school environments were calculated by summing the total calculated daily intakes associated with oral, dermal, and inhalation exposures in each of the three environments.
[6]. The theoretical exposures in the ambient environment were calculated by summing the total calculated daily intakes associated with oral, dermal, and inhalation exposures to soil, air, and different food pathways.
[7]. The theoretical exposures in the ambient environment represent the prospective fishing person consuming freshwater fish caught in the United States.

Data Needs Assessment

Several factors contribute to uncertainty in the Tier 1 assessment. One of the largest sources of uncertainty in the Tier 1 assessment includes the limited environmental data on BDEs in the United States and levels of the commercial octaBDE product in different consumer products. At the present time, the lifecycle of the commercial octaBDE product is not well understood. The pathways by which the commercial mixture or its primary constituents are released to the environment and accumulate in different biotic and abiotic compartments are the subject of several on-going studies in the United States and elsewhere. The limited data available describing environmental levels in the U.S. provide, at present, a limited understanding of the potential for exposures to the commercial mixture or its primary constituents in the ambient environment. However, given the conservative nature of the exposure assessment, it seems unlikely that exposure would be higher than the upper-bound estimates presented in this Tier 1 assessment

In addition to the uncertainties associated with limited data on environmental levels in the U.S., other sources of uncertainty pertain to data gaps in the available hazard studies. In particular, further information to improve the understanding of the relevance of thyroid, developmental and neurobehavioral effects in animals to humans would be useful.

The risk estimates (hazard indices) for octaBDE identified in this Tier 1 assessment indicate that there is room for some uncertainty in both exposure and hazard. However, the limitations in the available hazard information included in this Tier 1 assessment could be addressed in future

VCCEPP activities. Among the data gaps that were identified: 1) a two-year cancer bioassay using the current formulation of the commercial octaBDE product; 2) additional studies to improve the understanding of the effects of BDE constituents of the commercial product on thyroid function, neurobehavorial responses and developmental effects in laboratory animals; 3) a multi-generation reproductive toxicity study; and 4) developmental neurotoxicity studies. It is anticipated that the results of various hazard and exposure studies currently underway or planned in the United States and Canada should improve the understanding of BDE levels in the environment and the potential hazards posed to children and prospective parents. However, given that conservative exposure assumptions were used and large margins of safety calculated in this Tier 1 assessment, it is difficult to characterize these additional studies as data needs.

1.0 Introduction

This assessment was conducted by ENVIRON International Corporation (ENVIRON) on behalf of Great Lakes Chemical Corporation (GLCC) to evaluate the potential health risks to children and prospective parents associated with exposure to the commercial octabromodiphenyl ether (octaBDE) product (CAS No. 32536-52-0). This work was performed in accordance with the framework for developing a Tier 1 assessment for the Voluntary Children's Chemical Evaluation Program Pilot (VCCEPP). The work described in this document meets both the specific policy and technical objectives of the United States Environmental Protection Agency Office of Pollution Prevention and Toxics (hereinafter referred to as USEPA or Agency) VCCEPP, and reflects both a state-of-the science evaluation and practical judgment.

1.1 VCCEPP Program

In the December 26, 2000, Federal Register (Vol. 65, No. 248, pp. 81699-81718), USEPA announced the VCCEPP. The USEPA asked companies that manufacture and/or import 23 chemicals (including the commercial octaBDE product) that have been found in human tissues or measured in the environment as part of various monitoring programs to volunteer to sponsor one or more of these chemicals in the first tier of a pilot of this program.

The stated purpose of the VCCEPP is to provide the Agency and the public with the means to understand the potential health risks to children and prospective parents associated with exposure to chemicals to which children and adults may be exposed. The VCCEPP is intended to ensure that human health effects and exposure data, which may include data not necessarily limited to assessment of children's health, are made available to USEPA and others for evaluation of the risks of these chemicals such that mitigation measures may be taken as appropriate. Additionally, the Agency also committed as part of the VCCEPP to consider animal welfare and to provide instructions on ways to reduce or, in some cases, eliminate animal testing, while at the same time ensuring that public health is adequately protected. The Agency views the VCCEPP as an important component of its overall Chemical Right-to-Know initiative.

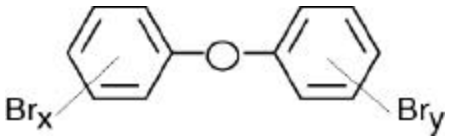
The Agency characterizes the VCCEPP as a pilot program focused only on 23 widely used industrial chemicals. The USEPA expects to refine the Program before its application to a larger range of chemicals in the future. Three tiers of assessment are envisioned in the Program. Tier 1 is characterized as a preliminary, screening evaluation (and is the focus of this report). If the results of the Tier 1 assessment indicate further evaluation is needed, then Tiers 2 and 3 will more fully characterize the risks that a chemical may pose to children, which would likely include laboratory toxicity bioassays and exposure studies to address data gaps or significant uncertainties in our understanding of the potential health risks.

In June 2001, GLCC agreed to participate in the VCCEPP and to sponsor Tier 1 assessments of the commercial octaBDE product. As part of Tier 1 sponsorship, GLCC committed to collect and/or develop information on health effects (hazard), exposure, and health risks. The hazard assessment requested by USEPA is nearly the same information requested in USEPA's High Production Volume (HPV) Challenge Program. GLCC also agreed to develop an exposure assessment and risk assessment. The assessment submitted as part of Tier 1 is intended to represent screening level information, with more detailed analyses submitted later as part of a future Tier 2 or Tier 3 commitment. In addition, GLCC agreed to perform a data needs assessment. This fourth item specifies whether additional environmental, exposure, and/or toxicology data are needed to address data gaps and reduce uncertainties either in the hazard assessment, exposure assessment, or risk assessment. The complete Tier 1 assessment (comprised of four components – hazard, exposure, risk, and data needs) will be submitted to USEPA's Peer Consultation Group for independent review.

1.2 Commercial Octabromodiphenyl Ether (octaBDE) Product

The focus of this VCCEPP Tier 1 assessment is the commercial octaBDE product:

IUPAC name:	Octabromodiphenyl ether (diphenyl ether, octabromo derivative)
CAS Number:	32536-52-0
EINECS Number:	251-087-9
Molecular formula:	$C_{12}H_5Br_8O$
Molecular weight:	801.38
Structural formula:	



The various synonyms and abbreviations used to denote individual polybrominated diphenyl ether (PBDE, or BDE) isomers, polybrominated biphenyls and diphenyls, and the commercial products often lead unintentionally to mis-identification of the commercial octaBDE product in the scientific literature. Synonyms and abbreviations include the following:

- polybrominated biphenyl ethers ≡ polybromobiphenyl ethers - PBBEs
- polybrominated biphenyl oxides ≡ polybromobiphenyl oxides - PBBOs
- polybrominated diphenyl ethers ≡ polybromodiphenyl ethers - PBDPEs
- polybrominated diphenyl oxides ≡ polybromodiphenyl oxides - PBDPOs

Unless otherwise stated, the term commercial octaBDE product will be used in this assessment to refer specifically to the commercially available product. When referring to a BDE congener group, the designation tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and deca- will be added to the beginning of the abbreviation to indicate the degree of bromination. Reference to one of the

209 individual polybrominated diphenyl ether (PBDE) isomers will be identified more specifically, where appropriate, using a numbering system similar to that used to distinguish the polychlorinated biphenyls (PCBs) (see Appendix I).

There are 175 different flame retardant chemicals, divided into four major groups: inorganic, halogenated organic, organophosphorus and nitrogen-based compounds and mixtures (EHC, 1997; Alaee and Wenning, 2002). Halogenated organic flame retardants are generally classified as either chlorinated or brominated flame retardants (BFRs). BFRs, which include the PBDEs, are further classified as either reactive or additive materials. The three PBDE flame retardants available commercially are additive materials and are referred to as the commercial penta-, octa-, and decabromodiphenyl ether products. Each of the commercial products is a mixture of brominated diphenyl ethers (BDEs) with varying degrees of bromination.

This VCCEPP assessment addresses only the commercial octaBDE product. The commercial octaBDE product is composed of a mixture of pentaBDE to decaBDE isomers. One commercial product is currently made in the U.S., and is referred to as Great Lakes DE-79™. The predominate congeners in the commercial octaBDE product are 2,2',3,4,4',5',6-heptaBDE (BDE-183) and 2,2',3,4,4,',5,5',6-octaBDE (BDE-203), followed by lesser amounts of, 2,2',3,4,4'-pentaBDE (BDE-85), 2,2',4,4',5- pentaBDE (BDE-99), 2,2',4,4',6-pentaBDE (BDE-100), 2,2',4,4',5,6-hexaBDE (BDE-154), and 2,2',4,4',5,5'-hexaBDE (BDE-153). Several chemical additives can be included in the commercial octaBDE product and comprise between 12 and 18 % by weight of the product formulation currently sold in the United States (i.e., Great Lakes DE-79™). The relative proportions of the different BDE constituents in the commercial octaBDE product sold in the United States and elsewhere, both past and present, are presented in Tables 1-1 and 1-2.

Table 1-1. Relative proportions by percent weight of different BDE homologue groups in the commercial pentaBDE, octaBDE, and decaBDE products.

BDE Homologue Group	Commercial pentaBDE product			Commercial octaBDE Product		Commercial DecaBDE Product
	2002	1985	late 1970's - early 1980's	2002	late 1970's - early 1980's	2002
	Great Lakes DE-71™ as currently produced [1]	Saytex 115 [2]	Great Lakes product [3]	Great Lakes DE-79™ as currently produced [4]	Great Lakes product [5]	Great Lakes DE-83R™ and DE-83™ as currently produced [6]
Decabromodiphenyl ether (1 congener)	--	--	0.8%	<0.70%	1.6%	>98%
Nonabromodiphenyl ether (3 congeners)	--	--	0.2%	<10%	13.0%	--
Octabromodiphenyl ether (12 congeners)	--	--	0.3%	<33%	30.7%	--
Heptabromodiphenyl ether (24 congeners)	--	--	2.6%	<45%	45.1%	--
Hexabromodiphenyl ether (42 congeners)	4-12%	--	13.3%	<12%	8.5%	--
Pentabromodiphenyl ether (46 congeners)	50-62%	--	58.1%	<0.50%	1.1%	--
Tetrabromodiphenyl ether (42 congeners)	24-38%	--	24.6%	--	--	--
Tribromodiphenyl ether (24 congeners)	0-1%	--	--	--	--	--

[1]. Data reported in the IUCLID document (see Appendix III) and GLCC MSDS for this product (see Appendix II). Other Great Lakes pentaBDE products (DE-60F Special™, DE-61™ and DE-62™) contain, by weight, 75% BDEs and 25% aromatic phosphates. Great Lakes DE-71™ was used in the study by Zhou et al. (2001) to evaluate the potential for effects on the thyroid in rodents.

[2]. Saytex 115 was used by Ethyl Corporation (1985a) to evaluate potential developmental effects in rodents. The composition of the product is unknown.

[3]. The GLCC commercial pentaBDE product produced in the late 1970's and early 1980's was used by Carlson (1980a) to evaluate potential effects on the liver in rodents, which is the basis for USEPA's current non-cancer Reference Dose toxicity value reported in IRIS (USEPA, 2003).

[4]. The composition of Great Lakes DE-79™ is reported in ECB (2002).

[5]. The GLCC commercial octaBDE product produced in the late 1970's and early 1980's was used by Carlson (1980a) to evaluate potential liver effects in rodents, which is the basis for USEPA's current non-cancer Reference Dose toxicity value reported in IRIS (USEPA, 2003).

[6]. MSDS (See Appendix II).

Table 1-2. Relative proportions by percent weight of different individual key BDE congeners in the commercial pentaBDE, octaBDE, and decaBDE product and occurrence in the manufacturing workplace, indoor buildings, and ambient environment

BDE Homologue Group	BDE Congener [1]	Formulations [2]				Indoor Buildings		Ambient Environment								
		Great Lakes DE-71 pentaBDE product	Great Lakes DE-79 octaBDE product	Bromkal 70-5DE pentaBDE product	Bromkal 79-8DE octaBDE product	Indoor Air [3]	Indoor Dust [4]	Ambient Air [5]	Soil [6]	Fish [7]	Meat [8]	Dairy [8]	Other Fats and Oils [8]	Eggs [8]	Vegetables [9]	Breast Milk [10]
Decabromodiphenyl Ether (1 congener)	**BDE-209**	**--**	**--**	**--**	**--**	11%	77%	1%	--	--	--	--	--	--	--	--
Nonabromodiphenyl Ether (3 congeners)	**--**	**--**	**--**	**--**	**--**	--	--	--	--	--	--	--	--	--	--	--
Octabromodiphenyl Ether (12 congeners)	**--**	**--**	**--**	**--**	**--**	--	--	--	--	--	--	--	--	--	--	--
Heptabromodiphenyl Ether (24 congeners)	**BDE-183**	**--**	**--**	**--**	**--**	1%	2%	--	--	--	--	--	--	--	--	✓
Hexabromodiphenyl Ether (42 congeners)	**BDE-154**	4%	2%	2%	4%	1%	1%	1%	--	✓	✓	✓	✓	✓	✓	✓
	BDE-153	6%	14%	3%	3%	2%	2%	1%	--	✓	✓	✓	✓	✓	✓	✓
Pentabromodiphenyl Ether (46 congeners)	**BDE-100**	8%	ND	7%	ND	4%	1%	4%	4%	✓	✓	✓	✓	✓	✓	✓
	BDE-99	43%	<1%	<1%	2%	27%	10%	31%	54%	✓	✓	✓	✓	✓	✓	✓
	BDE-85	--	--	--	--	1%	0%	--	--	--	--	--	--	--	--	--
Tetrabromodiphenyl Ether (42 congeners)	**BDE-66**	<1%	ND	<1%	ND	--	--	--	--	✓	--	--	--	--	--	**--**
	BDE-49	<1%	ND	<1%	ND	--	1%	--	--	--	--	--	--	--	--	**--**
	BDE-47	28%	<1%	30%	ND	53%	7%	63%	42%	✓	✓	✓	✓	✓	✓	✓
Tribromodiphenyl Ether (24 congeners)	**BDE-28/33**	**>1%**	**ND**	**>1%**	**ND**	--	--	--	--	--	--	--	--	--	--	✓

-- = data not reported

✓= congener reported relative percent occurrence not known

[1]. Numbering system adopted from PCBs (see Appendix I).

[2]. Data from Rayne and Ikonomou, 2002.

[3]. Sjödin et al., 2001a. Samples collected in a teaching hall with computers in Sweden.

[4]. Knoth et al., 2002. Samples collected from vacuum cleaner bags from German households.

[5]. Strandberg et al., 2001. Ambient air sampled in an urban location (Chicago) over three years (1997-1999).

[6]. Hale, 2002. Soil sampled near a foam production plant in mid-Atlantic region, USA.

[7]. Compilation of several studies. A total of 72 fish fillet samples from the USA.

[8]. Darnerud et al., 2000; unpublished. Swedish market basket study.

[9]. Ohta et al., 2002. Japanese market basket study.

[10]. Ryan et al., 2002. Twenty breast milk samples from Vancouver, Canada, collected in 2001-2002.

The commercial octaBDE product is used only for flame retardant purposes as an additive to certain types of plastics in consumer products manufactured by the electronics and plastic industries. It is used almost exclusively to flame retard acrylonitrile-butadiene-styrene (ABS) polymers used in computer casings (IPCS, 1994). According to the limited data provided by companies that purchase the commercial octaBDE product from GLCC for use as a flame retardant additive in ABS plastic, consumer products contain approximately 12-18% flame retardant. In the European Union (EU), approximately 95% of the total commercial octaBDE product supplied to industry is used in ABS plastic (IPCS, 1994). A similar level of use by the plastic and electronics industries is presumed in the United States.

PBDEs were the first group of BFRs to be detected in the environment. In 1979, the presence of BDE-209 was measured in soil and sludge samples collected from areas surrounding PBDE manufacturing facilities in the United States (de Carlo, 1979). Jansson and Asplund (1987) first suggested that PBDEs in tissue samples of fish-eating birds and marine mammals collected from the Baltic Sea, North Sea, and Arctic Ocean were similar to the commercial pentaBDE product, Bromkal 70-5. Similar reports confirmed the presence of BDE congeners in marine fish, shellfish, and sediments in the Pacific region and elsewhere (Watanabe et al., 1987a). Stafford (1983) confirmed the presence of BDEs in North America, reporting elevated concentrations of several congeners in the eggs and tissues of fish-eating birds from six U.S. states and Canada.

In recent years, increasing attention has been given to the presence of PBDEs in the environment. Nearly every environmental monitoring program conducted during the past decade has shown increasing levels of some BDE congeners in wildlife, particularly in Nordic countries where this trend contrasts with a general decline in the occurrence of dioxins, PCBs, and chlorinated pesticides in marine mammals and aquatic wildlife (Bergman, 2000; Hooper and MacDonald, 2000). More recently, evidence has begun to appear suggesting the presence of various PBDEs in humans. BDEs have been detected in human adipose tissue, blood serum, and in human breast milk (Stanley et. al., 1991; Klasson-Wheler et. al., 1997; Darnerud et al., 1998; Norén and Meironyté, 1998; Schroeter-Kermani et al., 2000; Päpke et. al., 2001; Petreas et. al., 2002; Ryan et. al., 2002; Thomsen et al., 2002; van Bavel et al., 2002; Choi et al., 2003; Meironyté-Guvenius et al., 2003; Petreas et al., 2003). The major route of human exposure appears to be through the diet, although data on this and other routes of exposure are limited at this time. A related concern is exposure of infants and young children to PBDEs and the potential risk for adverse health effects to the developing fetus and the breast-fed neonate.

To date, there have been few, if any, efforts by scientists to measure the commercial products in the environment or to correlate the occurrence of specific PBDE isomers and congeners with the commercial octaBDE product. Environmental monitoring data reported in the scientific literature typically describe the concentrations of total BDEs, homologue groups, or the concentrations only of certain BDE congeners (*cfg*, Alaee and Wenning, 2002; de Wit, 2002). Data reported in this manner hinder attempts to develop reliable exposure estimates describing plausible human exposure to the

commercial octaBDE product. Interpretation of the available environmental data is further complicated by the fact that much more extensive analysis has been performed for only certain congeners of the commercial octaBDE product.

The risk assessment of the commercial octaBDE product conducted by the European Chemicals Bureau (ECB) acknowledged that there are few data indicating adverse health effects in humans (Palm et al., 2002; Wenning, 2002; Darnerud et al., 2001, ECB, 2002). Available data indicate that the commercial octaBDE product is not mutagenic *in vitro*, and probably not genotoxic *in vivo* (ECB, 2002; Hardy, 2002a, 2002b). There are no data supporting carcinogenicity of the commercial octaBDE product. In general, the bioavailability and toxicity of the BDE molecule declines with increasing levels of bromination.

1.3 Purpose & Objectives

Conceptually, the VCCEPP Tier 1 assessment of the commercial octaBDE product is intended to address the following objectives:

1. Review and summarize the available animal bioassays and epidemiology studies reported in the scientific literature and identify potential human health endpoints and toxicity benchmarks relevant to children and prospective parents;

2. Understand how different uses of the commercial octaBDE product in U.S. commerce may result in exposures to children and prospective parents;

3. Determine the plausible pathways by which children and prospective parents might come into contact with the commercial octaBDE product, and assess whether these pathways may result in potentially meaningful and relevant exposures to children and prospective parents;

4. Estimate child exposures to the commercial octaBDE product for potentially meaningful and relevant situations using available screening-level approaches such as the use of predictive models and/or direct environmental measurements, and including assessment of the potential for aggregate exposure (whereby an individual may be exposed simultaneously to the commercial octaBDE product through more than one pathway);

5. Combine the findings from the hazard assessment with the results of the exposure assessment to derive qualitative and/or quantitative conclusions regarding the potential health risks to children and prospective parents; and,

6. Identify additional hazard and/or exposure information needed to further reduce uncertainties surrounding the potential risks to children and, where relevant, to prospective parents.

To achieve these objectives, the purpose of the VCCEPP Tier 1 assessment presented in this document is to report on the results of screening-level hazard, exposure, risk and data needs

assessments based on the available environmental and toxicological information, using the current paradigms for children's health risk assessment. The information and risk assessment modeling performed as part of this VCCEPP Tier 1 assessment have been fully documented and are consistent with federal guidance on risk assessment and children's health evaluation (USEPA, 1992, 1997, 2000a). The hazard, exposure, risk, and data needs assessments submitted to USEPA as part of this document are intended to be sufficient for independent third-party review by the Peer Consultation Group. This Tier 1 assessment represents the current state-of-the-science and addresses the adequacy of information associated with sources of exposure, populations at risk, environmental pathways, and routes of exposure to children.

1.4 Overall Tier 1 Approach & Scope

In accordance with available USEPA risk assessment guidance and recent guidance on preparing a Tier 1 assessment (Federal Register Notice, Vol 65, No. 248, 81699-81718, Sections I through K), this VCCEPP Tier 1 assessment includes four major components: (1) hazard assessment, (2) exposure assessment, (3) risk assessment, and (4) data needs assessment. Recent USEPA guidance on risk assessment of chemical mixtures also was consulted during the preparation of this assessment (USEPA, 2000c).

Hazard Assessment – A toxicology profile of the commercial octaBDE product, including abbreviated summaries of the available toxicology studies conducted to date, is presented in this section. A list of the chemical nomenclature used to define individual BDE congeners is included in Appendix I. A Material Safety Data Sheet (MSDS) for the Great Lakes DE-79™, as well as additional products produced by GLCC, is included in Appendix II. An International Uniform Chemical Information Database (IUCLID) data set containing hazard summaries describing each of the available relevant toxicology studies is included in Appendix III. In accordance with USEPA VCCEPP guidance, the IUCLID format is consistent with USEPA guidance for preparing Robust Summaries for the HPV Chemicals Challenge Program. This section concludes with a summary of available toxicity reference values used in the risk assessment for comparison to theoretical daily intake values and calculation of non-cancer hazard indices for various toxicity endpoints of concern.

Exposure Assessment – This section of the Tier 1 assessment includes four components. The first component describes the general substance information on the commercial octaBDE product including physical/chemical properties, the environmental fate, and exposure limits. The second component is a summary of primary manufacturing methods and chain-of-commerce uses for the commercial octaBDE product, including a description of primary manufacturing activities and highlighting those activities associated with the manufacture of the commercial octaBDE product that have the potential for significant worker exposures. Similarly, the predominant chain-of-commerce activities involving the use of the commercial octaBDE product as part of the manufacture of ABS plastic and its subsequent use in consumer products also highlights the potential for worker exposures. A description of chain-of-commerce uses of consumer products containing ABS plastic

that has been treated with the commercial octaBDE product, along with an assessment of the potential for general population exposure, is included.

The third component is a summary of available data describing environmental levels in the United States, North America, and elsewhere During data compilation, preference was given to data relevant to the ambient environment, home environment, and workplace in the United States. In the absence of U.S. data, secondary preference was given to data from Canada; third preference was given to data from the EU and elsewhere. All of the available U.S. data representing different environmental compartments (e.g., air, soil, sediment, fish, food products) are compared to data describing environmental levels in Europe and elsewhere. The data compiled from the literature are summarized in Appendix V. These comparisons provide a benchmark for establishing a general understanding of the occurrence of the commercial octaBDE product in the United States environment. In some cases, the compiled data are used to support exposure point concentrations in screening-level exposure models.

The fourth component of the exposure assessment includes a conceptual exposure model describing the possible routes of exposure to children and prospective parents. The conceptual exposure model for the commercial octaBDE product illustrates three different scenarios: exposures in the workplace, exposures in the indoor home/school/office environment, and exposures associated with the ambient environmental levels (e.g., via the diet, both direct and indirect contact with soil, and particulates in air). For direct and indirect exposure routes illustrated in the conceptual model, where exposures are plausible and quantifiable, screening-level exposure models developed in Microsoft® Excel are presented for children of different ages and adult men and women (prospective parents). The calculations used in the exposure assessment are presented in Appendix VI. Children exposures are evaluated for seven different age bins: <1 year, 1-2 years, 3-5 years, 6-8 years, 9-11 years, 12-14 years, and 15-18 years. Girls and boys are evaluated separately in the 9-11, 12-14, and 15-18 year age bins.

Consistent with the Tier 1 screening approach, exposure models rely on exposure point concentrations for different environmental compartments (e.g., air, soil, food stuffs, and in the workplace) representing the 95th upper confidence limit of the mean for robust data sets or the high end of the range of the available data, if the available data are limited. Exposure factors, based primarily on USEPA's *Exposure Factors Handbook* (USEPA, 1997) and *Child-Specific Exposure Factors Handbook* (USEPA, 2000a) are identified for each of the exposure scenarios and associated exposure pathways. All of the assumptions and parameter values used in the screening-level exposure calculations are specified and referenced to the relevant supporting technical information.

The exposure assessment section concludes with a summary of the results of the workplace, indoor home/school/office, and ambient environmental screening-level exposure models. Summary tables convey quantitative results relating sources of exposure to theoretical estimates of possible human exposure.

Risk Assessment – The results of the hazard and exposure assessments are used in the risk assessment section to convey both qualitative and quantitative conclusions about the likelihood that exposure to the commercial octaBDE product may pose a specific hazard to children or, where relevant, to prospective parents. Chronic intakes estimated using screening-level exposure models are compared to appropriate toxicity benchmarks believed to be relevant to children's health and the reproductive health of prospective parents. The same non-cancer RfD and other toxicity benchmark values are used to quantify non-cancer hazards to both children and adults. A key aspect of this effort is not to evaluate the toxicity of the individual chemical constituents in the commercial product; but, rather, to evaluate the toxicity of the commercial octaBDE product. Hazard information developed from studies of individual BDEs are not included in this Tier 1 assessment. The risk assessment section also includes a qualitative discussion of the various strengths and uncertainties inherent in the screening-level exposure models and their potential impact on the Tier 1 assessment.

Data Needs Assessment – This last section of the VCCEPP Tier 1 assessment addresses USEPA's specific requirement within the VCCEPP Tier 1 process for chemical sponsors to specify the additional hazard and/or exposure information that may be needed to further evaluate and understand the potential risks to children and, where relevant, prospective parents.

2.0 Hazard Assessment

A toxicology profile of the commercial octaBDE product, including abbreviated summaries of the available toxicology studies conducted to date, is presented in this section. An IUCLID data set prepared by GLCC containing hazard summaries describing each of the available relevant toxicology studies is included in Appendix III. The basis for USEPA's current non-cancer RfD for the commercial octaBDE product is included in Appendix IV. This section concludes with a summary of available toxicity reference values used in the exposure assessment for comparison to theoretical daily intake values and calculation of non-cancer hazard indices for various toxicity endpoints of concern.

2.1 Toxicokinetic Profile

Absorption and Metabolism

The few experimental data addressing bioavailability of the commercial octaBDE product in the scientific literature focus almost entirely on the oral route of exposure using rodents; inhalation and dermal bioavailability studies are not reported in the literature. Further, the majority of the limited available data focus on uptake, absorption rate, and distribution of specific congeners. With the exception of a single study of Great Lakes DE-71TM (a commercial pentaBDE product), information on the bioavailability of the commercial mixtures is lacking.

The available data on absorption, which are summarized in Tables 2-1 and 2-2, suggest significant differences based on the degree of bromination. Increasing the number of bromine atoms/molecule from 4 bromines in tetraBDE to 10 in decaBDE results in significantly reduced oral absorption, a shortened half-life, and a substantial increase in the percent of the total administered dose eliminated in feces and urine (Hardy, 2002a). Studies by Örn and Klasson-Wehler (1998) and Hakk et al. (1999) highlight the differences between lower and higher BDEs.

In a study conducted by Örn and Klasson-Wehler (1998), ^{14}C-labeled 2, 2', 4, 4'-tetraBDE (BDE- 47) was used to measure absorption and elimination in rats and mice. Animals were dosed orally with 30 µmol/kg of the tetraBDE dissolved in corn oil and their urine and feces collected daily for five days. Thereafter, the animals were sacrificed and their organs analyzed for chemical content. Örn and Klasson-Wehler (1998) reported that the rat excreted 14 % of the ^{14}C-labeled tetraBDE via feces and <0.5% via urine. Excretion rates in mice were higher; 20 % of the ^{14}C-labeled tetraBDE was excreted via feces and 33% via urine. If the amount of parent compound excreted at the end of the first day is assumed to be the non-absorbed dose, the rats and mice excreted about 5% and 7%, respectively, of the total administered oral dose (Örn and Klasson-Wehler, 1998). Without taking into consideration metabolism and other biochemical changes in the parent compound, the percent of the parent compound retained in rat and mice tissue after five days was 86% and 46%, respectively (Tables 2-1 and 2-2).

A similar study was conducted in Sprague-Dawley rats using ^{14}C-labeled 2, 2', 4, 4', 5-pentaBDE (BDE-99; Hakk et al., 1999). Animals were administered an oral dose of 2.2 mg/rat dissolved in peanut oil and their urine and feces were collected daily for 3 days. The major excretory pathway for the pentaBDE was through the feces. At the end of three days, 43% of the administered dose was excreted (Tables 2-1 and 2-2).

Table 2-1. Percent of the administered dose of a tetraBDE (BDE-47), a pentaBDE (BDE-99), decaBDE, the commercial pentaBDE product (Great Lakes DE-71Ô), and the commercial octaBDE product (Great Lakes DE-79Ô) excreted in the feces of rodents

Animal Species	PBDE Formulation	Route of Administration	Dose (mmol/kg bw)	Time (days)	BDE Congener Evaluated	% Excretion of Parent	% of Dose Retained	Reference
SD Rat	BDE-47 in corn oil	Oral	30	5	BDE-47	9	85.5	Klasson-Wehler et al. (2001)
SD Rat	BDE-47 in corn oil	Oral	30	1 2 3 4 5	BDE-47	5.7 5.4 1.2 0.9 0.5	94.3 88.9 87.7 86.8 86.3	Örn and Klasson-Wehler (1998)
SD Rat	BDE-99 in corn oil	Oral	15	1 3 12	BDE-99 (mean)	18 26 65	82 74 35	Klasson-Wehler et al. (2001)
SD Rat	BDE-99 in peanut oil	Oral	2.2 mg/rat	1 2 3	BDE-99	22.3 14.8 6	77.3 62.2 56**	Hakk et al. (1999)
SD Rat	DE-71 in peanut oil	Oral	672 ng/rat or 2.9 ppb	21	BDE-47 BDE-85 BDE-99 BDE-100 BDE-153 BDE-154	7.6 55.8 13.5 8.7 12.5 15.8	59.1 11.7 40.2 56.7 42.2 56.5	Hakk et al. (1999)
SD Rat	DE-79 in peanut oil	Oral	33 ng/day /rat or 3 ppb)	21	BDE-153 BDE-154 BDE-183 BDE-190 Hepta Octa Octa Octa	15.9 4.9 27.1 20.9 31.5 38.2 16.7 44.3	19.6 76.1 37.2 52.1 31.4 39 83.3 39.5	Huwe et al. (2002a)
SD Rat	DecaBDE in Lutrol F-127, soya phospholipid, water	Oral	3	7	DecaBDE	22	78	Klasson-Wehler et al. (2001)
Mouse*	BDE-47 in corn oil	Oral	30	1 2 3 4 5	BDE-47	7.6 6.4 2.2 2.2 1.7	92.4 86 83.8 81.6 79.9	Örn and Klasson-Wehler (1998)

SD Rat = Male Sprague-Dawley Rat
* Male C57B1 Mice used

** This value was calculated based on the reported percentages excreted for days 1 and 2 in the study. However, the authors reported this value as 43% (Hakk et al., 1999)

Table 2-2. Percent of the administered dose of tetraBDE (BDE-47), pentaBDE (BDE-99), and decaBDE (BDE-209) excreted in the urine of rodents

Animal Species	PBDE Formulation	Route of Administration	Dose (mmol/kg bw)	Time (days)	BDE Congener Evaluated	% Excreted of Parent Compound	% of Dose Retained	Reference
SD Rat	BDE-47 in corn oil	Oral	30	--	BDE-47	<0.5	85.5	Klasson-Wehler et al. (2001)
SD Rat	BDE-47 in corn oil	Oral	30	5	BDE-47	<0.5	86.3	Örn and Klasson-Wehler (1998)
SD Rat	BDE-99 in corn oil	Oral	15	--	BDE-99	<0.9	--	Klasson-Wehler et al. (2001)
SD Rat	BDE-99 in peanut oil	Oral	2.2 mg/rat	1 2 3	BDE-99	0.4 0.3 0.2	77.3 62.2 56	Hakk et al. (1999)
SD Rat	DecaBDE in Lutrol F-127, soya phospholipid, water	Oral	3	--	DecaBDE	<0.5	--	Klasson-Wehler et al. (2001)
Mouse*	BDE-47 in corn oil	Oral	30	5	BDE-47	33	46.8	Örn and Klasson-Wehler (1998)

SD Rat = Male Sprague-Dawley Rat
* Male C57B1 Mice used
-- Not Reported

Hakk et al. (2001a) also reported the results of a feeding study in which male Sprague-Dawley rats were administered 672 ng/rat (rats weighed between 258-288 grams) of the commercial pentaBDE product (Great Lakes DE-71™) in peanut oil for 21 days. The total concentration fed to the rats was approximately 2.9 ppb, which Hakk et. al. (2001a) believed to be representative of ambient environmental levels. Examining only the feces, livers and carcasses, Hakk et al. (2001a) reported that a pentaBDE (BDE-99) and a hexaBDE (BDE-153) were the most abundant congeners in the liver and carcass (see Table 2-5). Another pentaBDE (BDE-85) was highest in the feces, representing about 56% of the total administered dose (Tables 2-1 and 2-2). Hakk et al. (2001a) concluded that this study did not indicate any significant differences between the bioavailability and accumulation of lower brominated congeners, which are inferred from the study to refer to pentaBDE congeners and lower brominated congeners.

Huwe et al. (2002a) conducted a comparable study in which male Sprague-Dawley rats were fed 33 ng/day/rat (rats weighed between 250-300 grams) of the commercial product Great Lakes DE-79™ in peanut oil for 21 days. Animals were sacrificed 24 hours after the final feeding and their feces, liver, and carcass were analyzed. Huwe et al. (2002a) reported a hexaBDE (BDE-153) to be the dominant congener detected (see also Table 2-5). Feces excretion accounted for 20-40% of the dosed congeners with the exception of another hexaBDE (BDE-154) and one of the octaBDE congeners detected. These concentrations were near background levels and their percent recoveries ranged from

48-80%. Huwe et al. (2002a) believed that these recoveries indicated that higher BDE congeners were metabolized. However, they did not find any evidence to indicate conversion to lower congeners.

Klasson-Wehler et al. (2001) performed an experiment in which male Sprague-Dawley rats were separated into 3 groups and administered a dose of 30 µmol/kg body weight of a tetraBDE (BDE-47), 15 µmol/kg body weight of a pentaBDE (BDE-99), or 3 µmol/kg body weight of decaBDE (BDE-209). Both urine and feces were collected every 24 hours. Klasson-Wehler et al. (2001) reported that 9% of the tetraBDE was collected from the feces after 5 days. Additionally, an average of 65 % of the oral dose was excreted after 12 days as the pentaBDE (26% after 3 days, and 18% during the first 24 hours). By comparison, after 7 days, 22% of the administered decaBDE was excreted through the feces (Tables 2-1 and 2-2).

The only experimental study addressing dermal bioavailability is an unpublished study reported by GLCC (Inverest Research, 2001). In this study, human and rat dermis were examined for their ability to absorb tetraBDE using an *in vitro* tissue diffusion chamber system. This system allowed the skin samples to be maintained at a constant physiological temperature of 31.9 to 32.8°C and a dose of 10 mg/cm^2/day was administered. The receptor chamber for collection of absorbed material had a flow rate of 1.5 mL/hr, samples were collected hourly (0-6 hours post dose) and then at 2-hour fractions from 6-24 hours post dose. In the rat dermis model, the mean total unabsorbed material of applied dose was 82.27 %, and the mean dermal delivery of applied dose was 17.94%. The mean absorbed dose was 14.81 %. In the human dermis, the mean total unabsorbed material of applied dose was 97.56 %, the mean dermal delivery of applied dose was 3.13%, and the mean absorbed dose was 1.94%. The discrepancy between the permeability of rat dermis and human dermis was expected because rat dermis is typically 7 to 10 times more permeable than human dermis (Inverest Research, 2001).

Elimination Half-Life

Limited data are available in the scientific literature on elimination of the various congeners in the commercial octaBDE product. A study by von Meyerinck et al. (1990) tested the components of Bromkal 70™, a commercial pentaBDE product that is no longer produced in the United States but may be similar to Great Lakes DE-71™. Although the specific congeners were not identified, seven congeners in the mixture were detected by gas chromatography-mass spectroscopy (GC-MS) analysis. The mixture was found to contain one tetra-, three penta-, and three hexaBDE congeners. The results demonstrated that the degree of bromination directly influences the rate of elimination. Lower brominated compounds, such as tetraBDE, were eliminated more quickly than higher brominated compounds, such as hexaBDE. In addition, there was no significant variation in elimination between male and female rats for penta- and hexaBDE congeners. However, there was a sex difference for tetraBDE, with females having faster elimination. The elimination rates for the various BDE congeners are presented in Table2-3.

Table 2-3. Elimination rates for various BDE congeners found in Bromkal 70™ [1]

Component of Bromkal 70™	Half-Life in female rats (days)	Half-Life in male rats (days)
tetraBDE	29.9	19.1
pentaBDE	47.4	36.8
pentaBDE	25.4	24.9
penta- and hexaBDE	44.6	55.1
hexaBDE	90.9	119.1

[1]. von Meyerinck et al., 1990.

In addition to bioassays, scientists have used biological models to predict the uptake and half-life of BDEs based on chemical structure. For example, Palm et al. (2002) used Estimation Program Interface for Windows (EPIWIN) software to estimate the half-lives of BDE-47 (a tetraBDE), BDE-99 (a pentaBDE), and BDE-209 (a decaBDE) in various environmental media. EPIWIN predicts physical, chemical, and environmental fate properties of a chemical. Palm et al. (2002) reported the predicted half-life of the tetraBDE (BDE-47) to be around 11 days; a longer half-life, 318 days, was predicted for decaBDE (BDE- 209). The predicted half-life of the tetraBDE and pentaBDE (BDE-47 and BDE-99) in water, soil, and sediment was about the same (150 days); the half-life of decaBDE (BDE-209) was approximately 4-times longer (600 days).

Distribution in Biological Tissues

Only five studies describe the distribution of congeners in different biological tissues in rodents exposed orally to the commercial octaBDE product, commercial pentaBDE product(s) and one of the congeners, a pentaBDE (BDE-99). The results are presented in Tables 2-4 and 2-5.

Örn and Klasson-Wehler (1998) also reported that after 5 days, 86% of a tetraBDE (BDE-47) was retained in the adipose tissue of male Sprague-Dawley rats, and 47% of a tetraBDE was retained in the adipose tissue of male C57B1 mice (see Table 2-4). Similar results were reported by Klasson-Wehler et al. (2001) in a study where male Sprague-Dawley rats were fed a combination of 30 µmol/kg bw of a tetraBDE (BDE-47), 15 µmol/kg bw of a pentaBDE (BDE-99) and 3 µmol/kg bw of decaBDE.

Hakk et al. (1999) reported that a pentaBDE (BDE-99) preferentially accumulated in the carcass, gastrointestinal tract, adipose tissue and blood of male Sprague-Dawley rats after 3 days. Hakk et al. (2001a) reported that on average, after 21 days, 0.3-1.2% of dose of commercial pentaBDE product (Great Lakes DE-71™) remained in the liver and 17.9-42.7% remained in the carcass. Furthermore, Huwe et al. (2002a) reported that of the dose administered of the commercial octaBDE product, Great Lakes DE-79™, 0.9-1.7% was detected in the liver and 16.2-62.9% was detected in the carcass also after 21 days.

In a study reported by Hakk et al. (2001b), 672 ng/rat of the commercial product Great Lakes DE-73 (sic.) was fed to male Sprague-Dawley rats (weighing between 258-288 grams) in peanut oil for 21

days, and only the livers and carcasses were analyzed.[1] The total concentration fed to the rats was equal to approximately 2.9 ppb, which Hakk et al. (2001b) believed to be representative of ambient environmental levels. Hakk et al. (2001b) suspected that most of the congeners that were consumed, but not detected, were eliminated through the feces. Retention of the most prevalent BDE congeners ranged from 0.15-0.59 % of the administered dose in the liver and 11-56% in the carcass. A tetraBDE (BDE-47) had the greatest bioaccumulation in both tissues; 0.69% in the liver and 55.9% in the carcass. Hakk et al. (2001b) reported that the retention of the BDE congeners in the tissues seemed to decrease with increasing amounts of bromination, except for a pentaBDE (BDE-85) (Table 2-5).

Table 2-4. BDE congener concentrations in nmol/g lipid weight in different tissues of male Sprague-Dawley rats [1]

Tissue	BDE-47 nmol/g/w	BDE-99 nmol/g/w	DecaBDE nmol/g/w
Adrenal	ND	157	4.4
Adipose Tissue	693	116	0.17
Kidney	203	19	1.9
Liver	128	224	13.9
Lungs	134	80.5	2

ND = Not Determined

[1]. Dose was orally administered 30 µmol/kg body weight (bw) of BDE-47, 15 µmol/kg bw of BDE-99, and 3 µmol/kg bw of BDE-209 in corn oil for a maximum of 12 days (Klasson-Wehler et al., 2001).

[1] . It is necessary to note that according to GLCC (personal communication) the commercial Great Lakes DE-73 has never been produced and does not exist; it is not known whether Hakk et al. (2001b) are aware of a printing error in their study.

Table 2-5. The percentage of various BDEs in different tissues of male Sprague-Dawley rats [1]

Tissue	Hakk et al., 1999	Hakk et al., 2001a						Hakk et al., 2001b					
		Great Lakes DE-71™						Great Lakes DE-73™					
	% of BDE-99	% of BDE-47	% of BDE-85	% of BDE-99	% of BDE-100	% of BDE-153	% of BDE-154	% of BDE-47	% of BDE-85	% of BDE-99	% of BDE-100	% of BDE-153	% of BDE-154
Adrenals	0.1	--	--	--	--	--	--	--	--	--	--	--	--
Adipose Tissue	3.8	--	--	--	--	--	--	--	--	--	--	--	--
Blood	1.4	--	--	--	--	--	--	--	--	--	--	--	--
Carcass	38.8	32.6	32	45.2	33.9	44.1	27.4	55.9	10.62	30.6	56.09	49.78	21.07
G.I. tract	6.1	--	--	--	--	--	--	--	--	--	--	--	--
Heart	0.03	--	--	--	--	--	--	--	--	--	--	--	--
Kidney	0.1	--	--	--	--	--	--	--	--	--	--	--	--
Liver	0.9	0.7	0.6	1	0.7	1.2	0.4	0.69	0.15	0.46	0.4	0.25	0.16
Lungs	0.1	--	--	--	--	--	--	--	--	--	--	--	--
Testes	0.06	--	--	--	--	--	--	--	--	--	--	--	--
Thymus	0.06	--	--	--	--	--	--	--	--	--	--	--	--
Total % retained	***51.45***	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

-- = the tissue was not analyzed

N/A = Not Applicable. Because the carcass and the liver were the only tissues analyzed, it is not appropriate to calculate the total percent retained in the male Sprague-Dawley rats.

[1]. Dose was orally 2.2 mg/rat of BDE-99 in peanut oil for three days; 672 ng/rat of commercial pentaBDE product DE-71 for 21 days; and 672 ng/rat of commercial pentaBDE product DE-73 for 21 days.

2.2 Systemic Toxicity

2.2.1 Systemic Toxicity Studies

The acute, subacute, subchronic, and chronic effects of the commercial octaBDE product or its main BDE constituents via oral and inhalation pathways have been evaluated and summarized in the following sections.

Acute Studies

The acute effects of octaBDE have been evaluated in two studies at doses ranging from 126 mg/kg to 5000 mg/kg. The acute effects via inhalation exposure of octaBDE have been evaluated at only two concentrations.

Oral

Studies evaluating the potential acute oral effects of octaBDE have been conducted in both male and female rats (Norris et al., 1973; IRDC, 1975). No effects were observed in male rats given a single dose of 50, 500, or 5000 mg/kg via gavage during a 14-day observation period (IRDC, 1975). IRDC (1975) concluded that octaBDE would not be considered toxic by the oral route. In addition, Norris et al. (1973) reported no clinical signs of toxicity and no deaths following a single oral dose ranging from 126 to 2000 mg/kg in female rats. These studies are summarized in Table 2-6.

Table 2-6. Summary of acute oral studies with octaBDE

Species/ Strain	Duration / Dosing Method	Doses (mg/kg)	Endpoints Evaluated	NOAEL (mg/kg)	LOAEL (mg/kg)	Reference
Rats/male/ Charles River CD (Sprague Dawley); 5 per dose group	1day/ gavage	50, 500, 5000 in corn oil [1]	Body weight; mortality	5000	--	IRDC (1975)
Rat/female/ Sprague Dawley; number per dose group not specified	1day/ gavage	0, 126, 252, 500, 1000, 2000 (vehicle not specified)	Clinical signs; body weight; mortality	2000	--	Norris et al. (1973)

[1]. No control group was reported for this study.
-- Could not be determined.

Inhalation

Only one study was located that evaluated the potential acute effects of octaBDE following inhalation exposure (IRDC, 1975). The study exposed male and female rats to 2,000 or 60,000 mg/m^3 octaBDE

in the form of a dust for 1 hour in a closed glass chamber (Table 2-7). The physical properties of octaBDE precluded the administration higher atmospheric concentrations. The rats were examined immediately following exposure and at 14 days after exposure. All animals in both dose groups survived the 14-day observation period. No treatment-related clinical signs were reported and at the end of the 14-day study period; all rats in both dose groups appeared normal and exhibited normal body weight gains.

Table 2-7. Summary of acute inhalation studies with octaBDE

Species/ Strain	Duration/ Dosing method	Doses (mg/m^3)	Endpoints Evaluated	NOAEL (mg/m^3)	LOAEL (mg/m^3)	Reference
Rats/male and female/ Charles River CD (Sprague Dawley); 10 per sex per dose group	1 hour	2000, 60000 [1] (administered as dust particles)	Weight gain; clinical signs; mortality	60,000	--	IRDC (1975)

[1]. No control group was reported for this study.
-- Could not be determined.

Subacute, Subchronic, and Chronic Studies

The toxicity of octaBDE has been evaluated in longer-term repeated dose studies (e.g., 14 to 90 days) following oral or inhalation exposure. Exposures to octaBDE were associated with changes in the liver and thyroid gland. In the liver, octaBDE resulted in enzyme induction accompanied by increases in liver weights and hepatocellular size. Thyroid effects included decreases in thyroid hormone (T_4) levels and increases in the incidence of thyroid hyperplasia. The results of these studies are summarized in the text below and in Tables 2-8 (oral route) and 2-9 (inhalation route).

Oral

In a study designed to examine thyroid hormone concentrations and hepatic enzyme activity, Zhou et al. (2001) administered Great Lakes DE-79 ™ (a mixture of octaBDE, pentaBDE, hexaBDE, and tetraBDE, with octaBDE comprising 30.7% of the mixture) orally to 28-day old female rats at doses of 0, 0.3, 1, 3, 10, 30, 60, or 100 mg/kg/day for 4 days. A dose-dependent decrease in serum T_4 concentrations was noted with significant decreases reported at doses of 10 mg/kg/day and higher. A dose-dependent induction of hepatic microsomal ethoxy-resorufin-O-deethylase (EROD), pentoxyoxy-resorufin-O-deethylase (PROD), and uridinediphospahte-glucuronosyltransferase (UDPGT) activity was also reported, which was statistically significant at 10 mg/kg/day for PROD activity and 30 mg/kg/day for EROD and UDPGT activity.

In rats, octaBDE was administered at a concentration of 0.1 mmol/kg/day (77 mg/kg/day) in corn oil for 14 days (Carlson, 1980a). Administration of octaBDE resulted in increases in the following metabolic enzymes: NADPH cytochrome C reductase, cytochrome P-450, EPN detoxification, and benzo(a)pyrene hydroxylase, all of which are indicative of enhanced endogenous and xenobiotic metabolism. Sorbitol dehydrogenase levels, an indicator of liver damage, were not increased.

In a 28-day subacute dietary toxicity study, no treatment-related effects on general behavior, body weight or food consumption were noted in male or female rats fed diets containing 0, 100, or 1,000 ppm octaBDE (IRDC, 1976). While not measured directly, liver effects characteristic of enzyme induction were seen. Statistically significant increases in absolute and relative liver weights were observed at 1,000 ppm (81 mg/kg/day) for males and at 100 ppm (8 mg/kg/day) and 1,000 ppm (88 mg/kg/day) for female rats. Mean relative, but not absolute, liver weight was also increased in males in the 100 ppm dose group. An increased incidence of enlarged liver cells (centrilobular and mid-zonal hepatocytes) was seen in male rats in the low dose group and in males and females in the high dose group. These liver cells had granular "ground glass" appearance also characteristic of an enzyme induction (Popp and Cattley, 1991). No other histopathological changes in the livers of treated rats were noted. Thyroid hyperplasia was observed in 3 of 5 of the high-dose male rats but not female rats. No other treatment related findings were reported. In the absence of histopathological changes, observations in the liver were considered adaptive and not adverse. The NOAEL for males was 100 ppm, and for females the NOAEL was 1,000 ppm.

There were no changes in body weight, food consumption, hematology, clinical chemistry parameters (including measures of hepatic enzymes indicative of cellular damage, such as serum glutamic oxaloacetic transaminase (SGOT) or serum glutamic pyruvic transaminase (SGPT) in male or female rats that received up to 10,000 ppm octaBDE in the diet (approximately 577 mg/kg/day for males and 751 mg/kg/day for females) for 28 days (IRDC, 1978a). Signs of liver enzyme induction were noted. Absolute and relative liver weights were statistically significantly increased in the high-dose (10,000 ppm) group. In the mid-dose group (1,000 ppm or approximately 52 mg/kg/day for males and 71 mg/kg/day for females), relative liver weights were significantly increased in the males and absolute and relative liver weights were increased in the females. In the liver, the only tissue examined microscopically, hepatocytes with "ground glass" cytoplasm were reported in all male and female rats in the mid- and high-dose groups and in 4/5 males in the low-dose group (100 ppm or 5 mg/kg/day). Vacuolation of centrilobular hepatocytes was reported in 4/5 males in the high-dose group. With the exception of "ground glass" cytoplasm in all of the high-dose males, there were no treatment-related microscopic changes observed in the liver after a 4-week recovery period. A NOAEL for this study would be 52 mg/kg/day for males and 751 mg/kg/day for females based on the presence of vacuolation in high-dose males at the end of treatment.

In a 30-day feeding study, male Sprague Dawley rats received 0, 8, 80, or 800 mg octaBDE/kg/day (Norris et al., 1973). The findings were a decreased packed cell volume (PCV) and red blood cell counts (800 mg/kg/day), increased liver weights (8, 80, or 800 mg/kg/day), increased kidney weights

(80 and 800 mg/kg/day), centrilobular cytoplasmic enlargement in the liver (all dose groups) and hyaline degenerative changes in the kidney (all dose groups) (Norris et al., 1973). However, Norris et al. (1973) provided only brief information regarding the study protocol. Statistical significance was only stated in the text, results of statistical comparisons and the type of tests used were not provided.

Two 90-day studies have been conducted with octaBDE (Carlson 1980b; IRDC, 1978a). Carlson (1980b) only evaluated the potential for octaBDE to induce metabolism. In this study groups of Sprague Dawley rats were administered a high-dose series (0, 4.8, 9.6, or 19.15 mg/kg/day) and a low dose series (0, 0.6, 1.2, 2.4 mg/kg/day). Exposures to an octaBDE commercial product induced metabolism based on increases in EPN detoxification and p-nitroanisole demethylation in all dose groups (Carlson, 1980b). In the high-dose series, increases in cytochrome c reductase activity and cytochrome P-450 activity, were also noted. The effects on EPN detoxification, p-nitroanisole demethylation, cytochrome c reductase, and cytochrome P-450 were persistent and remained elevated at 30 and 60 days after cessation of dosing in both the low and high dose series. However, there were no microscopic changes reported in the livers of rats that received the low-dose series (the livers in the high-dose groups were not examined microscopically). The NOAEL for this study was at least 2.4 mg/kg/day (the highest dose in the low-dose series) and may have been higher. A LOAEL could not be determined.

Rats were fed octaBDE at 0, 100, 1,000 or 10,000 ppm (0, 5, 52, 577 mg/kg/day for males and 0, 7, 71, 751 mg/kg/day for females) for 13 weeks with an 8-week recovery period for some animals in each dose group (IRDC, 1978a). No treatment-related effects were reported on survival, clinical chemistry parameters, serum liver enzymes, or hematological parameters in any dose group. Decreases in mean body weight in females in the mid-dose group and males and females in the in the highest dose group were accompanied by decreases in food consumption. Statistically significant increases in thyroid, liver, and kidney weights, primarily reported at 1,000 and 10,000 ppm in males and females, seen at 13 weeks were comparable to controls following the 8-week recovery period. The only statistically significantly[2] increased histopathological changes seen were increases in the number of liver cells with the granular "ground glass" appearance in males (all doses) and females (two highest doses); increases in liver cells with vacuoles only in females in the high-dose group; increased in kidney tubule regeneration in high dose males only (which may be consistent with hydrocarbon nephropathy); and thyroid changes reported as tall columnar epithelium lining the follicles of the thyroid glands in high-dose males only. The hepatic microscopic effects noted in males and females, homogenous "ground glass" cytoplasm in the hepatocytes, were not considered adverse. However, the effects noted in females in the high dose group, vacuolation of periportal hepatocytes, were considered treatment-related adverse effects. Thyroid histopathological changes returned to base line conditions following the 8-week recovery period. The NOAEL for male and female rats was 52 and 71 mg/kg/day, respectively.

[2] Statistical analyses conducted by ENVIRON and consisted of pair-wise comparisons using the Fisher's Exact test.

Only one study, with a very limited amount of information reported, was located that assessed the potential chronic toxicity of octaBDE (the authors did not identify the compound tested as commercial product or congener) (Norris et al., 1973, 1975). Norris et al. (1973) reported that over the first 180 days of a two-year feeding study in male and female Sprague Dawley rats, there were no overt signs of toxicity and food consumption and body weights in the treated rats were comparable to the controls. Rats received diets that provided doses of 0, 0.01, 0.1, or 1 mg/kg/day. After 8 months of treatment, there were no overt signs of toxicity and the only effect reported was an increase in liver weights in the 1 mg/kg/day group (Norris et al., 1975).

Table 2-8. Summary of subacute, subchronic, and chronic oral studies with the commercial octaBDE product

Species/ Sex/Strain	Protocol	Endpoints Evaluated	Dose (mg/kg/day)	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Reference
Rats/female/ Long-Evans; 8 per dose group	Great Lakes DE-79 was administered orally in corn oil once a day for 4 days	T_3, T_4; EROD; PROD; UDPGT	0, 0.3, 1, 3, 10, 30, 60, 100	3	10 (decreased T_4)[1]	Zhou et al. (2001)
Rats/male/ Sprague-Dawley; 4 per dose group	octaBDE was administered orally once a day for 14 days	*O*-ethyl *O*-*p*-nitrophenyl phenylphosphonothioate (EPN) detoxification; p-nitroanisol demethylation; NADPH-cytochrome c reductase; cytochrom P-450; liver weight; UDP-glucuronyltransferase; benzo(a)pyrene hydroxylase; serum sorbitol dehydrogenase	0.1 mmol/kg/day (approximately 77 mg/kg/day based on a molecular weight of 766 g/mol)	77		Carlson (1980b)
Rat/male and female/ CD (Sprague Dawley); 10 per sex per dose group	octaBDE was administered in the diet for 28 days	Food consumption, body weight, organ weights, liver bromine analysis, macroscopic pathology and microscopic pathology (liver, kidneys and thyroid)	0, 100 or 1000 ppm (approximately 0, 8 or 81 mg/kg/day in males and 0, 8, or 88 mg/kg/day in females	8 (males and females)	81 (males – thyroid hyperplasia) 88 (females – thyroid hyperplasia)	IRDC (1976)
Rats/male and female/Sprague Dawley; 10/sex/group	octaBDE was administered in the diet for 28 days	Food consumption, body weight, hematology, clinical chemistry, organ weights, macroscopic pathology and microscopic pathology (liver only)	0, 100, 1000, or 10000 ppm (approximately 0, 5, 52, 577 for males and 0, 7, 71, 751 mg/kg/day)	52 (males) 751 (females)	577 (males – hepatocyte vacuolation)	IRDC (1978a)
Rats/male/ Sprague-Dawley; number per dose group not specified	octaBDE was administered in the diet for 30 days	Hematology; urinalysis; organ weights; histopathology	0, 8, 80, 800	8	80	Norris et al. (1973, 1975)

Species/ Sex/Strain	Protocol	Endpoints Evaluated	Dose (mg/kg/day)	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	Reference
Rat/male/ Sprague Dawley; number per dose group not specified	octaBDE was administered via corn oil gavage for 13 weeks with sacrifices at 1, 30 or 60 days after the last dose.	EPN detoxification, p-nitroanisole demethylation, cytochrome c reductase, and cytochrome P-450 protein	Low dose series : 0, 0.6, 1.2, 2.4 High dose series 0, 4.8, 9.6, or 19.15	2.4 [2]	[2] –	Carlson (1980a)
Rat/male and female/ Charles River CD (Sprague Dawley); 35 per sex per dose group	octaBDE was administered in the feed for 13 weeks.	Bromide analysis; hemoglobin; hematocrit; erythrocyte count; total and differential leukocyte counts; glucose urea nitrogen; serum glutamic oxalacetic and pyruvic transaminase activity; serum alkaline phosphatase activity; urinalysis; complete histopathlogy of all major organs.	0, 100, 1000, or 10000 ppm (approximately 0, 5, 52, 577 for males and 0, 7, 71, 751 mg/kg/day)	52 (males) 71 (females)	577 (males – thyroid tall columnar follicular epithelium) 751 (females – vacuolation of periportal hepatocytes)	IRDC (1978a)
Rats/male and female/Sprague Dawley; number per dose group not specified	octaBDE administered in feed for 180 days	Overt signs of toxicity; body weight; organ weights [3]	0, 0.01, 0.1, 1 mg/kg/day	1 mg/kg/day	--	Norris et al. (1973, 1975)

-- Not determined

[1]. In the absence of histopathological evaluation, liver enzyme changes (EROD, PROD, and UDPGT) were not considered adverse.

[2]. In this study groups of Sprague Dawley rats were administered a high dose series and a low dose series. Enzyme induction was increased in all dose groups in both series; however, this effect was not considered adverse. There were no microscopic changes in the livers of the rats in the low dose series (the high dose series was not examined microscopically). Consequently, the NOAEL for this study was 2.4 mg/kg/day (the highest dose tested in the low dose series) and a LOAEL could not be determined

[3]. A study summary and abstract were the only information available for this study.

Inhalation

Rats exposed to dust particles of octaBDE at concentrations of 1.2, 12, 120, or 1200 mg/m^3 for 8 hours a days for 14 days showed compound-related histopathological effects in the liver (IRDC, 1978b). At concentrations of 12 mg/m^3 and above, slight focal to multifocal cytoplasmic enlargement of the hepatocytes was reported. At concentrations of 120 mg/m^3 and greater, focal acidophilic degeneration of liver cells was observed. Histopathological changes were similar in the higher dose groups and increased in severity. Hepatocellular necrosis was also present at the 1200 mg/m^3 in male and female rats. The NOAEL for this study was 1.2 mg/m^3.

OctaBDE was evaluated in 14-day (WIL Research, 2000) and a 90-day nose-only inhalation toxicity studies (WIL Research, 2001), both in rats. In the 14-day study, the only treatment-related effects reported were consistent with enzyme induction (increases in absolute and relative liver weights and increases in the incidence of hepatocyte hypertrophy) and localized irritation (minimal increases in hypertrophy/hyperplasia of goblet cells in the nasal tissues) . The effects on the goblet cells were confined to specific area of the nasal cavity and there were no effects in underlying or adjacent structures in the nasal cavity. Based on these observations, the study authors concluded that the localized goblet cell hypertrophy/hyperplasia were not adverse effects and a reaction to the dust. A NOAEL for this study was 250 mg/m^3, the highest concentration tested.

In a 90-day study, rats were exposed to 1, 15, or 200 mg/m^3 (WIL Research, 2001) . The major findings in this study were statistically significantly decreased T_4 levels in the mid- and high-concentration males and high-concentration females and increased thyroid stimulating hormone (TSH) in the high-concentration females. However, there were no changes in T_3 levels or thyroid weights and no microscopic changes that were attributable to exposure to octaBDE were reported in the thyroid glands of treated rats. Increases in hepatocellular hypertrophy were observed in the mid- (3/10 males and 3/10 females) and high-concentration males (10/10) and females (3/10). In the lungs of the high-concentration male and female rats, alveolar histiocytosis and chronic active inflammation were noted in all animals. The ovaries of 3/10 female rats exposed to 200 mg/m^3 for 90-days had no corpora lutea visible, while corpora lutea were present in the ovaries of control rats. The incidence of this finding was not significantly increased; however, the authors concluded that this effect may have been related to treatment. A NOAEL for this study was 1 mg/m^3 based on the decreased T_4 levels.

Summary of Liver and Thyroid Effects

Following subacute and subchronic exposures to octaBDE, effects have been observed in the liver and the thyroid. In the liver, the only consistent effect noted was induction of enzymes involved in xenobiotic metabolism along with other observations characteristic of adaptive responses to enzyme induction to include increased liver weights and microscopic changes, such as increased size of the hepatocytes (hypertrophy) and cytoplasm described as “ground glass” in appearance. Enzymes characteristic of liver injury or damage, such as SGOT, SGPT, or sorbitol dehydrogenase, were not increased in any study measured at any dose tested. The pattern of enzyme induction in rats suggests

a phenobarbital-like metabolic pattern. According to Carlson (1980a), the large increases in three enzyme markers (EPN detoxification, p-nitroanisole demethylation, and NADPH cytochrome c reductase) compared to two other enzymes (UDPGT and benzo[a]pyrene hydroxylase) resemble phenobarbital-like induction. Further, induction of PROD occurred at lower doses than either EROD or UDPGT (Zhou et al., 2001).

With the exception of Carlson (1980a), studies of longer exposure duration did not assess the potential for octaBDE to induce enzyme systems; however, these studies did measure organ weights and examine the livers microscopically. In studies of 28- to 30-days duration, liver weights were increased in rats that received doses of 8 mg/kg/day or higher, and these increases were accompanied by increases in hepatocyte size (IRDC, 1976; Norris et al., 1973). In another 90-day study, increases in liver weights and microscopic changes in the liver also consistent with enzyme induction were reported at doses of 5 mg/kg or greater in males (IRDC, 1978a).

Collectively, as might be expected, the results of the subacute and subchronic oral toxicity studies with octaBDE indicate that exposures of longer duration result in enzyme induction at lower doses than shorter-term exposures. Carlson (1980a) did not examine the livers of rats in the high-dose series microscopically, while IRDC (1978a) did not evaluate induction of metabolic enzymes. However, no histological signs, other than those associated with enzyme induction, were seen in the IRDC (1978a) study. Consequently, the results of the IRDC (1978a) study, where rats were exposed to doses more than 2-fold higher (approximately 50 mg/kg/day) than the highest dose administered in the Carlson (1980a) study, suggest that the only microscopic changes that might have been observed in rats in the higher dose series in the Carlson (1980b) study would be changes consistent with enzyme induction. Microscopic changes in the liver that occur as a result of enzyme induction are considered adaptive changes (Popp and Cattley, 1991), and were not considered adverse. Consequently, the NOAEL of 2.4 mg/kg/day, as identified by the USEPA (USEPA, 2003), would likely be higher based on the findings in the IRDC (1978a) study.

Changes in thyroid hormone levels and thyroid morphology were also observed following exposures to octaBDE. Thyroid hormone levels were only evaluated following 4 days of exposure where significantly decreased levels of T_4 were reported at doses of 10 mg/kg/day or greater (Zhou et al., 2001). In this same study, levels of T_3 were unaffected. The thyroid glands were not examined microscopically. In 28-day and 90-day studies, thyroid glands were examined microscopically, although thyroid hormone levels were not determined. Following exposures for 28 days, thyroid hyperplasia was reported at doses of 81 or 88 mg/kg/day for male and female rats, respectively (IRDC, 1976). This effect was not observed at a dose of 8 mg/kg/day. After 90 days of exposure, the only microscopic effects reported were an increase in the incidence of follicular columnar epithelium height in the high-dose males (approximately 575 mg/kg/day) (IRDC, 1978a). This change had resolved following 8 weeks of recovery. There were no microscopic changes observed in male rats that received 5 or 52 mg/kg/day or in female rats that received doses of up to approximately 750 mg/kg/day.

In a 90-day inhalation study (WIL Research, 2000), rats were exposed to atmospheric concentrations (0, 1, 15, or 200 mg/m^3) that resulted in doses of approximately 0, 0.1, 2, or 27 mg/kg/day, assuming 75% absorption (ECB, 2002). TSH was significantly increased in the high-concentration females and T_4 was significantly decreased in the mid- and high-concentration females and in the high-concentration males. These changes occurred in the absence of changes in thyroid gland weights or thyroid histopathology. Collectively the results of these studies suggest that following exposures to the commercial octaBDE product, changes in T_4 levels occur before changes in thyroid gland weights or morphology.

Table 2-9. Summary of subacute and subchronic inhalation studies with the commercial octaBDE product

Species/ Sex/ Strain	Duration/ Dosing method	Doses (mg/m^3)	Endpoints Evaluated	NOAEL (mg/m^3)	LOAEL (mg/m^3)	Reference
Rats/ male and female/ Charles River CD (Sprague Dawley); 5 per sex per group	8 hours/day for 14 consecutive days via closed exposure chamber	1.2, 12, 120, or 1200 [1]	Body weight, hematology, clinical chemistry, urinalysis, bromine in the lung, liver, and fat tissues; histopathology of all major organs.	1.2	12 (cytoplasmic enlargement of hepatocytes accompanied by focal acidophilic degeneration)	IRDC (1978b)
Rats/male and female/ Charles River CD (Sprague Dawley); 5 per sex per group	6 hours/day, 5 days/week, for two consecutive weeks via nose-only inhalation	0, 1, 10, 100 or 250	Survival, clinical signs, food consumption, body weights, hematology, clinical chemistries, TSH, T_3, T_4, ophthalmologic examinations, macroscopic and microscopic pathology	250	– [2]	WIL Research (2000)
Rats/male and female/Charles River CD (Sprague Dawley); 10 per sex per group	6 hours/day, 5 days/week, for 13 consecutive weeks via nose-only inhalation	0, 1, 15 or 200	Survival, clinical signs, food consumption, body weights, macroscopic and microscopic pathology	1	15 (lung inflammation, increased TSH and decreased T_4)	WIL Research (2001)

[1]. The study did not include a control group. Rats were exposed to octaBDE in the form of dust particles.

[2]. Increases in absolute (100 and 250 mg/m^3 males and females) and relative (10, 100 and 250 mg/m^3 males and 100 and 250 mg/m^3 females) liver weights, increases in the incidence of hepatocyte hypertrophy (5/5 in the 10, 100 and 250 mg/m^3 males and 4/5 in the 100 and 250 mg/m^3 females), and hypertrophy/hyperplasia of goblet cells in the nasal tissues (all treated males and the 10, 100 and 250 mg/m^3 females). However, these effects were not considered adverse and a LOAEL was not identified.

2.2.2 Genotoxicity

The genotoxic potential of octaBDE has been evaluated *in vitro* in reverse mutation assays (Litton Bionetics, 1976; Microbiological Associates, 1996), an unscheduled DNA synthesis assay (Hazelton, 1983), a sister chromatid exchange assay and a chromosomal aberration assay (BioReliance, 1999) (Table 2-10). OctaBDE was not mutagenic when tested in reverse mutation assays with *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537, and TA1538 or *Saccharomyces cerevisiae* strain D4, with or without Aroclor 1254-induced rat liver S9 metabolic activation at doses up to 5000 µg/plate (Litton Bionetics, 1976; Microbiological Associates, 1996). OctaBDE did not induce unscheduled DNA synthesis (UDS) when tested in human WI-38 fibroblasts *in vitro* at doses up to 300 µg/ml (Hazelton, 1983), and did not increase the frequency of sister chromatid exchanges in Chinese hamster ovary (CHO) cells, when tested with or without metabolic activation at doses up to 750 µg/ml (Hazelton, 1982).

The potential for octaBDE to induce chromosomal aberrations was evaluated in human peripheral blood lymphocytes (BioReliance, 1999). OctaBDE did not increase the frequency of chromosomal aberration assays in human peripheral blood lymphocytes *in vitro*, when compared with the negative controls at doses up to 1000 µg/ml (Table 2-10). In these chromosomal aberration assays, octaBDE was tested at concentrations that induced cytotoxicity in the presence or absence of Aroclor-1254 induced rat liver metabolic activation.

2.2.3 Reproductive/Developmental Effects

Developmental toxicity studies of commercial mixtures containing octaBDE have been conducted in rats (Ethyl Corporation, 1985a; Life Sciences Research Israel Ltd., 1987; WIL Research, 1986a, 1986b) and in rabbits (Breslin et al., 1989) (Table 2-11)[3]. In each of these studies, pregnant rats or rabbits received daily gavage doses during the critical period of organogenesis during gestation and sacrificed just prior to parturition. The potential for both maternal toxicity and fetotoxicity was evaluated. Maternal toxicity, evidenced by decreases in maternal body weight and body weight gain, was seen in the higher dose groups in rabbits (15 mg/kg/day, the highest dose tested) (Breslin et al., 1989) and in rats (at doses equal to or greater than 50 mg/kg/day) (WIL Research, 1986a; Ethyl Corporation, 1985a). No evidence of maternal toxicity was noted in rats treated at doses equal to or less than 25 mg/kg/day (WIL Research, 1986a; Ethyl Corporation, 1985a; Life Sciences Research Israel Ltd., 1987). Although a screening study reported evidence of maternal and fetal toxicity in rats at <5 mg/kg/day, this study used an octaBDE product that is no longer made by a producer that is no longer in business in the U.S. (WIL Research, 1986b). Based on all of the applicable studies (Ethyl Corporation, 1985a; Life Sciences Research Israel Ltd., 1987; WIL Research, 1986a), the NOAEL for maternal toxicity in the rabbit is 5 mg/kg/day, while that in the rat is 25 mg/kg/day.

[3] Complete reports were only available for the Breslin et al. (1989) and WIL (1986a) studies.

Table 2-10. Summary of genotoxicity studies with the commercial octaBDE product

Assay Type	Strains Tested	Protocol	Results	Reference
Reverse Mutation Assay (overlay method)	*Salmonella typhimurium* strains TA-1535, TA-1537, TA-1538, TA-98, TA-100 and *Saccharomyces cerevisiae* strain D4	0, 0.25, 0.5, 5 and 50 µg/plate with and without Aroclor 1254-induced rat liver S9 metabolic activation	Negative	Litton Bionetics (1976)
Reverse Mutation Assay (plate incorporation method)	*Salmonella typhimurium* strains TATA-98, TA-100, TA-1535 and TA-1537	0, 33, 100, 333, 1000 or 5000 µg/plate with or without Aroclor 1254-induced rat liver S9 metabolic activation	Negative	Microbiological Associates (1996)
Unscheduled DNA Synthesis	Human WI-38 fibroblasts	0, 60, 90, 134, 201, 300 µg/ml, with or without Aroclor 1254-induced rat liver S9 metabolic activation [1]	Negative	Hazelton (1983)
Sister Chromatid Exchange	Chinese hamster ovary (CHO) cells	0, 7.5, 25, 75, 250, or 750 µg/ml, with or without Aroclor 1254-induced rat liver S9 metabolic activation [1]	Negative	Hazelton (1982)
Chromosomal Aberration Assay	Human peripheral blood lymphocytes	0, 8, 16, 32, 63, 125, 250, 500 or 1000 µg/ml for 4 hours with a 16 hour recovery time without metabolic activation	Negative	BioReliance (1999)
		0, 8, 16, 32, 63, 125, 250, 500 or 1000 µg/ml for 20 hours without metabolic activation	Negative	
		0, 32, 63, 125, 250, 500 or 1000 µg/ml for 4 hours with a 16 hour recovery time with Aroclor 1254-induced rat liver S9 metabolic activation	Negative	

[1]. The highest concentrations tested induced cytotoxicity.

Reproductive/developmental parameters evaluated, include the number of corpora lutea, fetal survival (the number of live fetuses per dam), litter size, number of implantations/resorptions, and sex ratio, and were unaffected by treatment at any dose in either rats or rabbits (WIL Research, 1986a; Life Sciences Research Israel Ltd., 1987; Breslin et al., 1989)[4]. A statistically significant decrease in mean fetal body weight was observed in the 50 mg/kg/day group in rats (WIL Research, 1986a). In contrast, a significant increase in early resorptions was noted in rats treated with Saytex 111 (a commercial mixture containing 35.5% octaBDE and 45% hexaBDE) at a dose of 25 mg/kg/day (Ethyl Corporation, 1985a). However, no detail/data were available for review for this study. OctaBDE did not produce skeletal or soft tissue malformations in either rats or rabbits at any dose tested with the exception of slight delays in ossification in both species noted only at doses that also produced maternal toxicity, that is, at doses equal to or greater than 5 mg/kg/day in the rabbit and 50 mg/kg/day in the rat. Whether this response (delayed ossification) represents a true adverse effect of the compound is questionable; therefore, the NOAEL for fetotoxity is 15 mg/kg/day in the rabbit (Breslin et al., 1989) and 25 mg/kg/day in the rat (WIL Research, 1986a). A NOAEL as low as 10 mg/kg/day is a possibility pending review of the details of the Ethyl Corporation study.

2.2.4 Immunotoxicity

Studies that specifically evaluated the potential effects of the commercial octaBDE product on the immune system have not been conducted. However, immune system organs (e.g., spleen, lymph nodes, thymus, bone marrow, etc.) have been evaluated microscopically as part of 90-day dietary and inhalation studies in rats (IRDC, 1978a; WIL Research, 2000). There were no treatment-related microscopic changes in any of the organs listed above.

2.2.5 Carcinogenicity

The commercial octaBDE product has not been evaluated in a two-year animal bioassay, and the potential for octaBDE to induce cancer in animals is not known. Apparently one two-year study was started; however, only preliminary results of this study have been reported in the literature (Norris et al., 1973, 1975). The genotoxicity studies conducted with octaBDE have been negative, and based on the results of these studies, octaBDE would not be expected to induce cancer by a genotoxic mode of action. Other nongenotoxic modes of action deserve further investigation.

[4] Mean post-implantation losses were increased in the 50 mg/kg/day dose group (1.6%) and, although this was above the historical control range for WIL Research (1986a; 1.2%) it was not significantly different from concurrent controls.

Table 2-11. Summary of reproductive and developmental effects with octaBDE

Species/ Sex/ Strain	Exposure/duration/ frequency	Dose (mg/kg/day)	Endpoints	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	References
Rabbits/Female/ New Zealand White 26 per dose group	Daily gavage doses to pregnant rabbits on GDs 7 to 19; Does sacrificed on GD 28 and fetuses examined [1]	0, 2, 5, or 15 mg/kg/day of Saytex 111 (a commercial mixture containing 33.5% octa-BDE)	Maternal body weight (GD 28)	5	15	Breslin et al. (1989)
			Maternal body weight gain (GD 7 to 28)	5	15	
			Maternal liver weight (absolute and relative)	5	15	
			Maternal kidney weight (absolute and relative)	15	-	
			Reproductive/ developmental parameters (percent pregnant, number of litters with viable pups, corpora lutea/doe, implantations/doe, live fetuses/litter, percent implantations absorbed, fetal body weight, fetal sex ratio)	15	-	
Rats/Female/ Strain not specified 25 per dose group	Daily gavage doses on GDs 6 to 15; dams sacrificed prior to parturition; fetuses examined	0, 2.5, 10 or 25 mg/kg/day of Saytex 111 (a commercial mixture containing 33.5% octa-BDE)	Fetal body weight	10	25	Ethyl Corporation (1985a)
			Live fetuses per litter (decrease in live fetuses per litter)	10	25	

Species/ Sex/ Strain	Exposure/duration/ frequency	Dose (mg/kg/day)	Endpoints	NOAEL (mg/kg/day)	LOAEL (mg/kg/day)	References
Rats/Female/ Sprague-Dawley COBS CD 10 per dose group	Daily gavage doses on GD 6 to 15; dams sacrificed on GD 20; fetuses examined	0, 2.5, 10, 15, 25, or 50 mg/kg/day of DE-79 (a commercial octa-BDE mixture) in corn oil	Maternal survival	50	-	WIL Research (1986a)
			Maternal body weight (GDs 0, 6, 9, 12, 16, and 20)	25	50	
			Maternal body weight gain (GD 6 to 16)	50	-	
			Maternal body weight gain (GD 16 to 20)	25	50	
			Maternal clinical chemistry (GD 20)	50	-	
			Maternal serum bromide levels	15	25	
			Maternal liver and thyroid weight	50	-	
			Maternal liver and thyroid histopathology	50	-	
			Reproductive/developmental parameters (numbers of corpora lutea, number of viable fetuses, early and late resorptions, sex ratio, and total number of implantation sites)	50	-	
			Fetal weight (decreased)	25	50	
			Skeletal and soft tissue malformations (delayed ossification)	25	50	

[1]. Three pregnant rabbits, one each from 0, 2, and 5 mg/kg/day dose groups died during the study. Two rabbits in the 15 mg/kg/day dose group delivered their litters prior to GD 28. Following gross pathological examination, cause of death or early delivery was not determined.

2.3 Robust Toxicological Summaries

An IUCLID data set prepared by GLCC containing hazard summaries describing each of the available relevant toxicology studies is included in Appendix III. In accordance with USEPA VCCEPP guidance, the IUCLID format is consistent with USEPA guidance for preparing Robust Summaries for the HPV Chemicals Challenge Program (USEPA, 1999a)

2.4 Tier 1 Assessment Absorption Factors and Toxicity Values

There is limited data in the scientific literature describing the absorption or metabolism of the commercial octaBDE product and its constituents. For the purpose of this Tier I assessment, oral, dermal, and inhalation absorption factors values were determined based on the results of five recent studies (Hakk et al., 2001a; Hakk et al., 1999; Örn and Klasson-Wehler, 1998; Klasson-Wehler et al., 2001; and Inverest Research, 2001). These studies assumed that the difference between the administered dose and the amount of the parent compound excreted in both the feces and urine represented the absorbed fraction. In these studies, bioavailability was based on the lowest brominated BDE isomer retained in exposed animals, which in all cases was BDE-100 (a pentaBDE.

Oral, dermal, and inhalation absorption factors used in the Tier 1 exposure assessment are summarized in Table 2-12. For oral routes of exposure, 50% of the predicted exposure concentration of the commercial octaBDE product was assumed due to the lack of available data (ECB, 2002). The dermal absorption value was assumed to be 3.13 % (absorption value for tetraBDE) based on the results of an unpublished human skin absorption study conducted by Inveresk Research (2001), which is a conservative value based on the differences in the potential absorption for tetra-BDE vs. higher brominates congeners. In the absence of any data describing inhalation absorption of BDEs, the default value of 75% used by ECB (2002) was adopted to represent the bioavailable fraction of commercial octaBDE product absorbed by inhalation routes.

Table 2-12. Oral, dermal, and inhalation bioavailability values used in the Tier 1 exposure assessment

Substance	Exposure Route	% Absorption	Reference
Commercial octaPDE Product	Oral	50	ECB (2002)
	Dermal	3.13	Inveresk Research (2001)
	Inhalation	75	ECB (2002)

For the purposes of this Tier 1 assessment, which represents a screening-level evaluation based on the currently available scientific information, the relevant toxicity endpoints for evaluating exposures to children and prospective parents based on the available data are: (1) thyroid hormone disruption, and (2) change in maternal or fetal body weight, and (3) liver enzyme induction. In the absence of any data, cancer is not included as a human health endpoint in the

Tier 1 assessment. Toxicity values used in the Tier 1 exposure assessment are summarized in Table 2-13.

Table 2-13. Human health endpoints and toxicity values used in the Tier 1 exposure assessment

Potential Human Health Endpoint	Toxicity Value	Relevant Study
Reproductive/ Developmental effects: change in maternal or fetal body weight	0.09 mg/kg/day	WIL Research (1986a)
Thyroid effects: disruption of T_4 homeostasis	0.09 mg/kg/day	Zhou et al. (2001)
Liver enzyme induction	0.003 mg/kg/day	USEPA Reference Dose (RfD) for the commercial octaBDE product based on Carlson (1980b)

Reproductive/Developmental Effects

Effects on reproduction and/or development would be relevant for children and prospective parents. Developmental toxicity has been evaluated in rabbits (Breslin et al., 1989) and in rats administered the commercial octaBDE product to dams during gestation and through lactation (Ethyl Corporation, 1985a; Life Sciences Research Israel Ltd., 1987; WIL Research 1986a). Maternal toxicity, evidenced by decreases in maternal body weight and body weight gain, were seen in the higher dose groups in rabbits (15 mg/kg/day, the highest dose tested) (Breslin et al., 1989) and in rats (at doses equal to or greater than 50 mg/kg/day) (WIL Research,1986a; Life Sciences Research Israel Ltd., 1987; Ethyl Corporation, 1985a). No signs of maternal toxicity were seen in rats at doses equal to or lower than 25 mg/kg/day. Also, no adverse fetotoxic effects were noted in the rabbit at doses up to 15 mg/kg/day (Breslin et al., 1989) or in the rat at doses up to 25 mg/kg/day (WIL Research, 1986a). In the WIL Research (1986a) study, the only fetal effect noted was a decrease in mean fetal weight in the highest dose tested, 50 mg/kg/day. No effects on indices of pregnancy (e.g., gestation length, live litter size, sex ratio) were observed in rabbits (Breslin et al., 1989) or in rats (WIL Research, 1986a; Life Sciences Research Israel Ltd., 1987) at any dose tested. An increase in late resorptions was reported in a summary of the study conducted by Ethyl Corporation (1985a). The Ethyl study evaluated a commercial octaBDE mixture, Saytex 111, that, unlike DE-79 used in the WIL Research (1986a) study is no longer commercially available. No data or details were available for either the Ethyl study or the study conducted by Life Sciences Research Israel Ltd. (1987). Therefore, the Ethyl and Life Sciences studies were not considered quantitatively in the development of toxicity values.

The NOAEL in the rat for both maternal and fetotoxicity was 25 mg/kg/day and represents a value that could be used to derive toxicity values. The results of a screening study by WIL Research (1986b) were not considered because the study involved an octaBDE product that is no longer made in the U.S. Note that a NOAEL as low as 10 mg/kg/day is a possibility pending

review of the details of the Ethyl Corporation study. However, benchmark dose modeling (BMD) was conducted using the fetal body weight data from the WIL Research (1986a) study, thereby avoiding the need to identify an experiment-specific NOAEL. Using K-power hybrid model, the benchmark model dose level indicating a 5% statistical increase in response above background ($BMDL_5$) was estimated to be 8.7 mg/kg/day. A $BMDL_5$ was selected for continuous reproductive/developmental effects based on the studies conducted by Allen et al. (1994a, b). Application of uncertainty factors for inter- and intraspecies extrapolation would result in a toxicity value 0.09 mg/kg/day that would be compared estimates of exposure for woman of child-bearing years and children 2 or 3 years of age. For this screening level Tier 1 assessment all age groups and both male and females were considered.

Thyroid Effects

The thyroid has been identified as a target tissue in subacute and subchronic rat studies. Significant decreases in T_4 levels were seen in rats exposed to doses of 10 mg/kg/day and higher for 4 days (Zhou et al., 2001) and 250 mg/m^3 (equivalent to 25 mg/kg/day, assuming 75% absorption, a default breathing rate for the rat, and averaging over exposure days) (WIL Research, 2001). Neither histopathological lesions nor functional changes were seen in the 25 mg/kg/day (estimated) dose group in the WIL Research study (2000). Histopathological changes (changes in follicle lining) were noted in a 90-day feeding study but only in males and only at the highest dose tested (575 mg/kg/day) (IRDC, 1978a).

It is known that pregnant women and the developing fetus are sensitive to thyroid hormone disruption (McDonald, 2002). Key stages of development, which may involve thyroid hormone homeostatis, e.g., neurobehavioral development, in humans are most likely during the last trimester (brain development begins in the first trimester) and post-natally for the first 1 to 2 years (McDonald, 2002). Consequently, *in utero* exposure should be considered. No data were available for octaBDE on levels of T_4 in the fetus; however, evidence of decreased fetal T_4 levels in rats exposed to pentaBDE at doses at which maternal T_4 levels were also reduced have been reported (Zhou et al., 2002). It is not known if the decreases in the fetus were the direct action of pentaBDE in the fetus or the decrease in maternal transfer of T_4. Maternal transfer is the major source of T_4 during the first two trimesters (McDonald, 2002).

The potential for fetal thyroid effects is likely dependent on the mode of action for decreases in T_4 levels. If these fetal changes are due to enzyme induction, in particular UDPGT, which would increase conjugation of T_4, or by inducing its metabolism to the metabolite that competes with T_4, the differences in the presence and activity of enzymes *in utero* and in the neonate and young child in the rat and human should be considered. If the key enzyme is UDPGT, this particular enzyme is not thought to be active in the human *in utero (*Clewell et al., 2002) and consequently exposures that do not result in decreases in maternal transfer of T_4 would be of interest. However, UDPGT become active soon after birth and, therefore, the potential for thyroid effects should be considered for children of all ages. If the mode of action for disruption of thyroid

homeostatis in the fetus is similar to that of other hydroxyl-chlorinated biphenyls, which is associated with the transport protein transthyretin (TTR), then octaBDE could be transported from the mother to the fetus (McDonald, 2002).

PentaBDEs required metabolic activation to competitively bind with TTR and then only when metabolized by phenobarbital-type microsomes (CYP2B) (Meerts et al., 2000). However, while the octaBDE isomer was not tested in this competitive binding assay, the hexa- and heptaBDEs tested did not compete with T_4, with or without metabolic activation (Meerts et al., 2000). Moreover, in humans, the key transport protein for T_4 is thyroid-binding globulin (TBG) and may transport T_4 from the mother to the fetus (McDonald, 2002). The affinity of PBDEs for TBG is not known but the affinity of hydroxyl-chlorinated biphenyls is low (McDonald, 2002). It is unlikely that BDEs act directly on the hypothalamic-pituitary axis as there are no changes in TSH levels in response to BDE exposure (Zhou et al., 2001). While not known with certainty, effects on the fetus from maternal exposure to octaBDE may only occur at doses that produce sufficient hypothyroidism in the mother, such that maternal transfer of T_4 is sufficiently impaired.

Children older than 1 to 2 years of age may be more sensitive to these changes in thyroid hormone levels than adults, Therefore, children are evaluated in this assessment. As with enzyme induction, effects on thyroid levels may be proportional to the body burden and in children for a given exposure, the body burden would be expected to be greater than in an adult. The exposure assessment accounts for this in that children would have a higher amount of intake for the same level of exposure as adults.

The biological significance of subtle changes in T_4 levels in the rat should be considered. Changes in T_4 levels occurred at approximately the same estimated doses in both the 4-day study (Zhou et al., 2001) and the 90-day (WIL Research, 2000) study. Significant changes did not occur at 3 mg/kg/day or less (next highest dose was 10 mg/kg/day) in the 4-day study or at 1.5 mg/kg/day or less (estimated based on absorption, breathing rate, body weight) (next highest dose was estimated to be 25 mg/kg/day) in the 90-day study. Further, changes in T_4 levels in the 90-day study were not accompanied by changes in thyroid weight or histopathology even at the highest concentration (WIL Research, 1986a). Further, unlike the potential for enzyme induction, which may happen at the same blood concentration in rats and humans, there is considerable evidence that humans are more resistant to changes in circulating thyroid hormone levels because of the significant buffering capacity of binding proteins and are less sensitivity to alterations in those changes (USEPA, 1998a). Even if it is assumed that these findings in rats may be qualitatively relevant for human health assessment, differences in sensitivity to small changes in thyroid hormone levels in the rat, should be considered quantitatively.

In this screening level Tier 1 assessment, changes in thyroid hormone levels in the rat were used to develop toxicity values to compare to levels of exposure to children of all ages and prospective parents. However, the biological significance of changes in T_4 in the rat relative to human development is uncertain.

Benchmark doses ($BMDL_{10}$s) were derived using the data from Zhou et al. (2001), WIL Research (2000), and IRDC (1978a) and were evaluated using BMDWin software. The $BMDL_{10}$ for the 4-day study was higher, 8.5 mg/kg/day in males (the only sex tested). The $BMDL_{10}$s derived from the 90-day inhalation study were 3.3 mg/kg/day in males and 1.7 mg/kg/day for females. The $BMDL_{10}$ based on thyroid follicular columnar epithelium changes in the 90-day feeding study was 73 mg/kg/day. While BMDLs from the inhalation study are lower, there is considerable uncertainty in the route extrapolation and the resulting estimates of intake or body burden. An inhalation RfC could be developed for comparison to estimates of air exposure. However, for estimates of intake primarily by the oral route, the $BMDL_{10}$ derived from the Zhou et al. (2001) study is recommended with an uncertainty factor of 100 (3 for intraspecies extrapolation, 10 for human variability, and 3 for duration of dosing). Each of these factors takes into account both the differences in kinetics and sensitivity (USEPA, 1994). For intraspecies extrapolation, a factor of 3 was used in the absence of data on the kinetic differences in the rat and human. A factor of 1 was used for sensitivity, as it is expected that humans would be less sensitive to thyroid effects than the rodent (USEPA, 1998a). A factor of 3 was used for duration of dosing because the doses at which changes in thyroid hormone levels occurred were similar in the 4-day and 90-day studies. Also, while at initial exposure decreases in thyroid hormone levels may be manifested, both rats and humans have significant capacity to compensate for these small changes and maintain homeostatis. For this screening level Tier 1 assessment, the resulting toxicity value of 0.09 mg/kg/day was compared to estimates of exposure for children of all ages and prospective parents.

Enzyme Induction

The USEPA (2003) has derived an RfD for octaBDE based on increased hepatic enzyme induction in rats reported by Carlson (1980b). In this study, groups of Sprague Dawley rats were administered a high dose series (series 0, 4.8, 9.6, or 19.15 mg/kg/day) and a low dose series (0, 0.6, 1.2, 2.4 mg/kg/day). Enzyme induction was increased in all dose groups in both series. There were no microscopic changes in the livers of the rats in the low dose series; however, the high dose series was not examined microscopically. Consequently, the USEPA considered the highest dose tested in the low-dose series (2.4 mg/kg/day) a NOAEL. To derive the RfD, a composite uncertainty factor of 1000 (10 for interspecies extrapolation, 10 for intraspecies variation and 10 for study duration) was applied, resulting in an RfD of 3×10^{-3} mg/kg/day. This value is still reported on IRIS (USEPA, 2003); however, it has not been revised since developed in the early 1980s. In this Tier 1 assessment, this RfD was used to provide an upper-bound estimate of hazard for children and adults.

Carlson (1980b) did not examine the livers of rats in the high-dose series microscopically. However, in the IRDC (1978a) study, where rats were exposed to much higher doses (up to 575 and 751 mg/kg/day for males and females, respectively), the microscopic changes observed in livers of these rats were considered adaptive changes, consistent with enzyme induction (Popp and Cattley, 1991), with the exception of vacuolation observed in the 751 mg/kg/day females. It

is likely that only these adaptive changes would have been observed in the livers of the high-dose series in the Carlson (1980b) study if those tissues had been examined microscopically. As a result, a higher NOAEL may have been identified for the Carlson (1980b) study. Therefore, the RfD would also be higher.

It is important to note that the enzymes induced in the Carlson study were those potentially involved in endogenous and xenobiotic metabolism, not enzymes indicative of cellular damage in the liver (SGOT, SGPT, sorbital dehydrogenase were unchanged even at high doses) and other tissues. If enzyme induction by octaBDE in the rat were also to occur in humans, the pattern of xenobiotic metabolism may be altered, although the potential consequences of enzyme induction, particularly for children, are unclear. Depending on the enzyme induced and the endogenous or exogenous substrate metabolized, it can not be said *a priori* that enzyme induction is an adverse effect.

Enzyme induction in the fetus by the commercial octaBDE product has not been evaluated. There are data that suggest that *in utero* exposures to pentaBDE resulted in significant increases in EROD induction in the rat fetus at the highest dose tested (30 mg/kg/day) (Zhou et al., 2002). Levels of the enzyme, UDPGT, which is involved in T_4 conjugation, were not detected in the fetus (Crofton, 2003). Many of the types of enzymes present in the fetus and neonate in the rat and human are heterogeneousand some are homogeneous, the timing of their activation (and likely the inducibility) differ (Clewell et al., 2002). Nevertheless, it is possible that for children and adults of child-bearing age, key metabolic enzymes could be induced resulting in changes in metabolic patterns in children. If induction of key enzymes is linked to other biological consequences not limited to liver toxicity, then while enzyme induction in itself is not an adverse effect, it would be health protective to limit exposure to levels that would not result in enzyme induction.

$BMDL_{10}$s were estimated based on continuous data for markers of induction for 4 different enzymes following 90 days of exposure of up to 19 mg/kg/day in Carlson (1980a) study using the K-power hybrid model. These BMDLs ranged from 0.32 to 8.96 mg/kg/day. BMDLs reported by Zhou et al. (2001) for enzyme induction of 3 separate enzymes ranged from 0.4 to 11 mg/kg/day following 4 days of exposure of up to 100 mg/kg/day. Although different enzymes were evaluated by Carlson (1980b) and Zhou et al. (2001), the magnitude of the increases, based on the BMDL) was comparable for the two studies, suggesting that increasing exposure duration did not result in an increased magnitude of response.

For this screening level assessment, rather than focus on a specific enzyme evaluated in the rat that may not be present or be active in humans, the 4 BMDLs developed from the Carlson (1980b) study and the 3 $BMDL_{10}$s reported by Zhou et al. (2001) for enzyme induction (ranging from (0.4 to 11 mg/kg/day) were averaged to develop a mean $BMDL_{10}$ for enzyme induction. The resulting mean BMDL was 3.8 mg/kg/day. This value would represent a baseline for enzyme induction in the rat. The application of uncertainty factors to this mean BMDL is not

recommended as enzyme induction does not represent an adverse effect and the relevance of the resulting number would be questionable and highly uncertain.

However, it is possible that enzymes present in humans could be induced by exposure to octaBDE potentially resulting in some adverse effect due to altered metabolic patterns. Further, in general, induction of liver enzymes in the rat may be quantitatively similar in humans for the same body burden, although different isozymes may be induced in rats and humans. Toxicity benchmarks for the other endpoints observed in the octaBDE studies (e.g., thyroid and developmental effects) were 0.09 mg/kg/day, as discussed above. If enzyme induction in the rat and human is comparable at comparable body burdens, significant enzyme induction would not be expected in the human at these toxicity benchmarks for other endpoints.

3.0 Exposure Assessment

This section of the Tier 1 assessment includes four components: (1) a summary of general substance information (2) primary manufacturing methods and chain-of-commerce uses for the commercial octaBDE product; (3) a summary of available data describing environmental levels in the U.S. and North America; and, (4) the conceptual model and exposure calculations describing the scenarios and exposure pathways used to predict children and prospective parent exposures to the commercial octaBDE product. The first component summarizes general substance information on the commercial octaBDE product including physical/chemical properties, the environmental fate, and exposure limits. The second component summarizes the primary manufacturing methods and chain-of-commerce uses for the commercial octaBDE product and includes a description of primary manufacturing activities; highlighting those activities associated with the manufacture of the commercial octaBDE product that have the potential for significant worker exposures. Similarly, the predominant chain-of-commerce activities involving the use of the commercial octaBDE product as part of the manufacture of consumer products also highlights the potential for significant worker exposures. A description of downstream uses of consumer products containing the commercial octaBDE product, along with an assessment of the potential for general population exposure, is described. The third component is a compilation of U.S. and North American environmental data that are used to identify exposure point concentrations in the screening-level exposure models. The fourth component is the conceptual exposure model describing the possible routes of exposure to children and prospective parents. The conceptual exposure model for the commercial octaBDE product illustrates three different scenarios: exposures in the workplace, exposures in the indoor home/school/office environment, and exposures associated with ambient environmental levels (including food).

3.1 General Substance Information

The commercial octaBDE product made in the U.S. by GLCC (Great Lakes DE-79™) is composed of a mixture of primarily pentaBDE (approximately 0.5%), hexaBDE (approximately 12%), heptaBDE (approximately 45%), octaBDE (approximately 33%), nonaBDE (approximately 10%), and decaBDE (approximately 0.7%) congeners (IUCLID, see Appendix III). The predominate congeners in the commercial octaBDE product are 2,2',3,4,4',5',6-heptaBDE (BDE-183) and 2,2',3,4,4,',5,5',6-octaBDE (BDE-203), followed by lesser amounts of 2,2',3,4,4'-pentaBDE (BDE-85), 2,2',4,4',5- pentaBDE (BDE-99), 2,2',4,4',6-pentaBDE (BDE-100), 2,2',4,4',5,6-hexaBDE (BDE-154), and 2,2',4,4',5,5'-hexaBDE (BDE-153). One commercial product is currently made in the United States, and is referred to as Great Lakes DE-79™. The relative proportions of the different BDE constituents in commercial octaBDE products sold in the United States and elsewhere are presented in Tables 1-1 and 1-2.

At present, GLCC is the sole manufacturer in the United States of the commercial octaBDE product and currently produces one formulation, Great Lakes DE-79™, where the number

indicates the percent total bromine by weight. A MSDS for the commercial octaBDE product is included in Appendix II.

Physical and Chemical Properties

The physical-chemical properties of the commercial octaBDE product are presented in Table 3-1. The information in Table 3-1 is pertinent to the commercial product, as indicated, unless stated otherwise.

Table 3-1. Physical and chemical properties of the commercial octaBDE product

Physical / Chemical Property	Characteristic
Chemical formula	$C_{12}H_5Br_8O$
Molecular weight	801.38 (80% bromine by weight)
Melting point	85-89 ^oC (commercial product)
Boiling point	Decomposes at >330^oC (commercial product)
Relative density	2.8 (commercial product)
Vapor pressure	6.59×10^{-6} Pa at 21°C (commercial product)
Water solubility	<1 ppb at 25 °C (commercial product) heptabromodiphenyl ether component = 1.98 µg/L
Log octanol-water partition coefficient (K_{ow})	6.29 (measured; commercial product)
Flammability	Not applicable - flame retardant
Autoflammability	Decomposes above 330^oC (commercial product)
Explosive properties	None
Oxidizing properties	None
Viscosity	None

With the application of heat, the commercial octaBDE product decomposes more quickly than the host polymer matrix, hindering the formation of flammable gases. The release of bromine molecules upon heating interferes with the radical chain mechanism that typically takes place in the gas phase during combustion. High-energy hydroxyl and hydrogen radicals formed during combustion are removed by bromine (Rahman et al., 2001).

Physical Appearance

The commercial octaBDE product is characterized as an off-white to tan powder or flaked material (ECB, 2002) or sticky, solid particulates (GLCC, personal communication). In contrast, pure octaBDE is characterized as a white crystalline solid (ECB, 2002).

Melting Point

The melting point of the commercial octaBDE product has been reported by GLCC as 85-89 °C. However, ranges such as 130-155°C, 70-150°C, and 167-257°C have also been reported (ECB, 2002).

Boiling Point

A boiling point is not available for the commercial octaBDE product. Technical-grade octaBDE decomposes approximately 2% at 330°C and 40% at 395°C (Dead Sea Bromine Corp, 1993). Because the commercial product is a mixture, it is expected to exhibit a wide temperature range for decomposition (WHO, 1994).

Density

The relative density of the commercial octaBDE product is reported as 2.8 at 25 degree Celsius (^{0}C; Great Lakes DE-79™ MSDS; see Appendix II).

Vapor Pressure

The vapor pressure for the substance analyzed by the ECB (2002) has been measured as 6.59 x 10^{-6} Pa at 21°C using a spinning rotor gauge in a Good Laboratory Practice (GLP) study (Stenzel and Nixon, 1997). According to Watanabe and Tatsukawa (1990), vapor pressure increases as the degree of bromination decreases. According to ECB (2002), the material tested was a composite sample from three manufacturers and had the following composition: 5.5% hexabromodiphenyl ether, 42.3% heptabromodiphenyl ether, 36.1% octabromodiphenyl ether, 13.9% nonbromodiphenyl ether, and 2.1% decabromodiphenyl ether. The method used was not able to separate the contributions of the individual components to the total vapor pressure; results likely represent only the vapor pressures of the more volatile constituents of the commercial mixture and, thus, the value likely represents the upper limit of the vapor pressure of the commercial mixture. GLCC has reported the same value at 21°C for the Great Lakes DE-79™ commercial product.

Solubility

According to GLCC, the water solubility of the commercial octaBDE product is <1 ppb at 25°C (MSDS for Great Lakes DE-79™; see Appendix II). The ECB (2002) reported a value of 0.5 µg/L at 25°C.

Octanol-Water Partition Coefficient

According to Watanabe and Tatsukawa (1990), the octanol-water partition coefficient (log K_{ow}) for the commercial octaBDE product is 8.35-8.90 using a high-performance liquid chromatography method. GLCC and MacGregor and Nixon (1997) have reported a similar value, 6.29 at 25°C.

Flash Point

The commercial octaBDE product does not have a flash point because it is used as a flame retardant, making this parameter irrelevant.

Autoignition

The commercial octaBDE product does not undergo autoignition and, instead, decomposes at elevated temperatures (>330^{0}C). The decomposition properties are consistent with the use of this material as a flame retardant.

Explosivity

Explosive properties are not expected on the basis of chemical structure and physical properties. The commercial octaBDE product is not known to exhibit explosive properties with other materials.

Oxidation

Testing for this property is not applicable due to the physical nature of the substance. The commercial octaBDE product does not contain any substance with structural alerts for oxidizing effects and, therefore, is not considered to be an oxidizer.

Surface Tension

This property is not applicable due to the physical nature of the substance.

Hazardous Chemical Reactions

According to ECB (2002), pyrolysis of the commercial octaBDE product between 400°C and 900°C can result in the formation of brominated dibenzofurans and brominated dibenzo-p-dioxins.

3.2 Environmental Fate

Abiotic Degradation

No information is currently available on abiotic degradation of the commercial octaBDE product. However, the limited information on abiotic degradation of octaBDE indicates that it may behave similarly to decaBDE. Eriksson et al. (2001) recently reported, that when in a solution of methanol (80%) and water (20%), photolysis of 2,2',3,4,',5,5',6-octabromodiphenyl ether (BDE-203) does occur and that as the degree of bromination increases so does the amount of photodegradation. Ericksson et al. (2001) reported a half-life of 5 hours for BDE-203.

One of the major concerns associated with the higher brominated diphenyl ethers (e.g. decaBDE and octaBDE), is the formation of lower brominated derivatives (e.g. tetraBDE and pentaBDE), since these compounds occur extensively in the environment and are believed to be more

bioaccumulative in comparison to other congeners. According to the ECB (2002), data on the direct photolysis of decaBDE in water shows that it does photodegrade but that lower brominated congeners (heptaBDE to nonaBDE) are formed only in small amounts and are not the major degradation products. The limited information reported in the scientific literature suggests that photodegration is possible. However, estimates of the rate of accumulation of octaBDE and degraded products, as well as their breakdown rates, are not known (ECB, 2002).

A rate constant for the reaction of octaBDE with hydroxyl radicals has been estimated as 2.1 x 10^{-13} cm^3 $molecule^{-1}$ s^{-1} using the Syracuse Research Corporation AOP estimation program. Assuming an atmospheric concentration of hydroxyl radicals of 5 x 10^5 molecules/cm^3, an atmospheric half-life of around 76 days has been estimated for this reaction (ECB, 2002).

Biodegradation

The commercial octaBDE product is not readily biodegradable (ECB, 2002). Information is not available regarding anaerobic biodegradation.

Volatilization

Volatilization of the commercial octaBDE product is likely minimal because low vapor pressures have been estimated or measured for many PBDEs. However, once in the atmosphere, PBDEs are likely to adsorb strongly onto atmospheric particles and subsequently removed by wet or dry deposition.

Adsorption

Octanol-water partition coefficients (log K_{ow}) have been measured for several constituents of the commercial octaBDE product (Watanabe and Tatsukawa, 1990). The values obtained are shown in Table 3-2. In general, higher brominated BDE compounds tend to adsorb more tightly to soil and sediment than lower brominated compounds. The results indicate that all components of commercial octabromodiphenyl ether are expected to adsorb strongly onto soil and sediment since they have high octanol-water partition coefficients (ECB, 2002).

Using the Technical Guidance Document, ECB (2002) calculated a log organic carbon (K_{oc}) value for octaBDE of 156,640 L/kg. Similarly, EPIWIN predicted the K_{oc} to be 144,544 L/kg. No measured value is available for octaBDE. However, based on the measured adsorption coefficient values for commercial decaBDE and pentaBDE products, a value of 1,363,040 L/kg would be expected for commercial octaBDE product.

Table 3-2 Octanol-water partition coefficients [1]

Polybrominated diphenyl ether	Log Kow
Dibromodiphenyl ether	5.03
Tribromodiphenyl ether	5.47-5.58
Tetrabromodiphenyl ether	5.87-6.16
Pentabromodiphenyl ether	6.46-6.97
Hexabromodiphenyl ether	6.86-7.92
Octabromodiphenyl ether	8.35-8.90
Decabromodiphenyl ether	9.97

[1]. Watanabe and Tatsukawa, 1990

From the above information, the main BDE constituents of the commercial octaBDE product can be characterized as relatively immobile in soil and unlikely to leach into groundwater. Using the Surface Area Modeling System (SAMS) model for tetraBDE, pentaBDE, and octaBDE congeners, ECB (2002) concluded that releases of tetra-, penta-, and octaBDEs to the soil surface would result in contamination of only the top few centimeters of soil, with an insignificant amount, if any, leaching into groundwater.

Bioaccumulation Potential

Bioaccumulation has been reported to increase with decreasing bromination (Darnerud et al., 2001). According to ECB (2002), octaBDE congeners are expected to be bioaccumulative.

3.3 Occupational Exposure Limits

Occupational exposure limits for the commercial octaBDE product have not been established in the U.S. by the Occupational Safety and Health Administration. However, GLCC has established a Worker Exposure Limit (WEL) of 0.14 mg/m^3 (GLCC, personal communication).

3.4 Environmental Limits

Environmental limits for the commercial octaBDE product have not been established in the United States.

3.5 Production Volumes

Historically, there may have been as many as 9 different manufacturers of commercial BDE products worldwide (WHO, 1994). At present, 3 commercial BDE formulations are produced and sold in the United States and elsewhere. Of these, the commercial decaBDE product constitutes 82% of the reported global BDE demand, the commercial octaBDE product contributes an additional 5-7% of global demand, and the commercial pentaBDE products contributes the remaining 11-13% (Hale et al., 2002). The annual worldwide production of all three commercial BDE products was estimated in 1994 as 40,000 metric tons/year (KEMI, 1994). The majority of worldwide production is associated with the commercial decaBDE product

(30,000 metric tons/year), followed by the commercial octaBDE product (6,000 metric tons/year) and the commercial pentaBDE product(4,000 metric tons/year ; KEMI, 1994).

In 1999, overall annual global demand for these products increased to 67,125 metric tons/year with North America accounting for approximately 50% of the increased demand (Hale et al., 2002). Other major markets for the commercial BDE products in 1999 included Asia (37%) and Europe (12%) (Hale et al., 2001). At present, North America accounts for nearly 36% of the total global demand for the commercial octaBDE product (Hale et al., 2002; Renner, 2000). GLCC is currently the sole manufacturer of the commercial octaBDE product in the United States.

3.6 Production Methods

Commercial octaBDE product is produced by the direct bromination of diphenyl ether using a Friedel-Crafts catalyst. The molten liquid produced is placed into pans to solidify. Once solid, the mixture is broken out of the pans and ground and supplied bagged as either the pure product or blended with a synergist (ECB, 2002). In some applications, the flame retardant is compounded with the polymer to produce pellets (referred to as masterbatch). The pellets are used in the polymer-processing process method used to manufacture products such as the hard plastic housings of several commonly used office equipment and business machines such as typewriters, computer central processing units (CPUs), video terminals, and photocopiers. In the United States, only one chemical manufacturing plant, owned and operated by GLCC and located in Arkansas, manufacture and distribute the commercial octaBDE product.

The amount of flame retardant used in different plastic casing products depends on the flame retardancy required of the finished product, the effectiveness of the flame retardant and synergist within a given polymer, the physical properties of the end product (e.g. color, density, stability etc.) and the use to which the end product will be put. Typically, the flame-retardants are added at concentrations between 5 and 30% by weight (WHO, 1994; ECB, 2002).

3.7 Chain-of-Commerce Product Uses

Primary Use

The commercial octaBDE product is used only for flame retardant purposes as an additive in manufactured products by the plastics industry. It is used almost exclusively to flame retard ABS polymers used in computer casings and monitors (IPCS, 1994). According to the limited data provided by companies who purchase the commercial octaBDE product from GLCC for use as a flame retardant additive in ABS, products contain approximately 12-18% flame retardant. In the EU, approximately 95% of the total commercial octaBDE product sold to the electronics and plastic industries is used in ABS. Although data are not available, GLCC estimates similar volumes in the United States.

Other Possible Uses

In the United States, 80-90% of the commercial BDE products are used high impact polystyrene (HIPS), flexible polyurethane foam, textile coatings, wire and cable insulation, electrical and electronic connectors and other interior parts (Rahman et al., 2001). Other minor uses may include polybutylene terephthalate (PBT) and polyamide polymers, at typical loadings of 12-18% by weight in the final product. Currently, GLCC recommends a typical loading of 16% by weight of the commercial octaBDE product in the final product.

3.8 Summary of Available Environmental Data

This section summarizes the available data describing environmental levels in the United States and North America. Additional supporting information, including the environmental sampling data from different studies used to develop media-specific data summaries is provided in Appendix V.

Environmental Data Compilation Strategy

During data compilation, preference was given to data relevant to the ambient environment, indoor home/school/office environment, and workplace in the United States. In the absence of United States data, secondary preference was given to data from Canada; third preference was given to data from the EU and elsewhere. The environmental data presented in this Tier 1 assessment was collected from both primary and secondary literature sources and may not represent an exhaustive survey of the published scientific literature. In most cases, the values published in different documents were not verified, nor was the quality of the study considered during the selection and compilation of data.

Among the challenges in the Tier 1 assessment of the commercial octaBDE product is discerning the differences in environmental and hazard information pertaining to a specific BDE isomer, a congener group, and the product. Although the commercial octaBDE product clearly represents a mixture of many chemicals, it is not uncommon to find both environmental and hazard studies imprecisely describing their purpose, methods, and results as pertinent to the commercial product when, in fact, the study is pertinent to only one or more constituents of the commercial product. For example, environmental monitoring data reported in the scientific literature typically describe the occurrence of PBDEs in one of several ways: the concentrations of one or more congener groups (e.g., tetra- through nona- BDEs), total PBDE concentrations, or the concentrations of certain BDE isomers. It is not uncommon for studies to inadequately specify or inconsistently use chemical terms to describe congener groups, isomers, and the commercial product.

In general, 10-12 individual BDE isomers are commonly reported (although not consistently) by different researchers involved in environmental investigations or human exposure studies. Data reported in this manner greatly hinder efforts to develop reliable exposure estimates describing plausible human exposure. Interpretation of the available environmental data also is complicated by the fact that much more extensive analysis has been performed only in recent years and for

only a few constituents of the commercial products. In addition, the chemical composition of the commercial octaBDE product has been shown to vary between commercial products and over the past 10-20 years (e.g., see Table 1-1).

To address these limitations in the scientific literature, a chemical profile of the commercial octaBDE product is used to identify the dominant individual BDEs that can reasonably be associated with the commercial product (yet, recognizing that a few BDE isomers may occur in both the commercial pentaBDE and the commercial octaBDE products). In studies that do not report specifically on the occurrence of the commercial octaBDE product and, instead, report on the occurrence of specific BDEs, the concentrations of one or more of the individual BDEs will be assumed to be representative of the occurrence of the entire commercial octaBDE product. If more than one BDE associated with the commercial octaBDE product is reported in a study, the results are assumed to be additive and the occurrence of the commercial octaBDE product is assumed to be the sum of the concentrations of each of the individual BDEs. In cases where the same BDE may occur in both the commercial pentaBDE and the commercial octaBDE products, the same study used to support the commercial octaBDE product Tier 1 assessment also may be used to support the commercial pentaBDE product Tier 1 assessment. In general, however, the technical limitations associated with changes in the chemical composition of the commercial product over time and the occurrence of BDE constituents in the environment that originate from the commercial product cannot be resolved in Tier 1; instead, both of these challenges are identified as sources of uncertainty in the Tier 1 assessment.

Levels in Ambient Air

In general, few environmental data describing levels of BDEs in U.S. ambient air are available in the scientific literature. Table 3-3 summarizes the available U.S. data, as well as the results of ambient air monitoring conducted elsewhere in the world. None of the studies found in the scientific literature attribute the occurrence of individual BDEs to the commercial octaBDE product. The predominant BDEs reported in ambient air, BDE-47, BDE-99, BDE-100, and BDE-153, are found in the commercial octaBDE product with the exception of BDE-47.

Few ambient air studies have been conducted in the United States. Air samples collected in the United States from urban, rural, and remote shorelines of the Great Lakes all contained measurable levels of BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154 (Dodder et al., 2000). Strandberg et al. (2001) reported a total BDE concentration range in air of between 5.5 in rural environments and 52 pg/m^3 in urban air in Chicago, Illinois. The concentrations of total BDEs represent the sum of six congeners: BDE-47, BDE-99, BDE-100, BDE-153, BDE-154, and BDE-209. Air measurements were averaged over a three-year period between 1997 and 1999. The results of ambient air data collected by Hale et al. (2001) report the levels of different BDEs as a percentage of total PBDE levels. The occurrence and relative distributions of BDEs reported by Hale et al. (2001) are consistent with the data reported by both Dodder et al. (2000) and Strandberg et al. (2001), as well as by scientists in other countries (see Table 3-1).

With the exception of Canada, the occurrence and relative distributions of BDEs reported in the United States are consistent with data reported by scientists in other countries (see Table 3-1). The highest ambient air levels have been reported in Canada. By comparison, the levels of BDE-47, BDE-99, BDE-100, BDE-153 in ambient air in Sweden, where considerable environmental monitoring has been conducted in recent years, range from 0.7-100 pg/m^3, 0.35-60.0 pg/m^3, 0.07-9.0 pg/m^3, 2.94-4.0 pg/m^3, respectively (Bergman et al., 1999; Sjödin, 2001a; Palm et al., 2002).

Levels in Sediment

In general, few environmental data describing levels of BDEs in sediments in U.S. waterways are available in the scientific literature. Table 3-4 summarizes the available U.S. data, as well as the results of environmental sampling conducted elsewhere in the world describing the concentrations of BDEs found in sediment from rivers, estuaries, and lakes near industrial plants, sewage plants, and landfills. Only one study found in the scientific literature (conducted in the United Kingdom) attributes the occurrence of individual BDEs to the commercial octaBDE product (Allchin et al., 1999).

In two U.S studies conducted by Hale et al. (2002) and Dodder et al. (2002), total BDE concentrations in sediment ranged from non-detect to 132 ng/g dry weight (dw). The highest value was reported in sediment immediately downstream from a flexible polyurethane foam (FPUF) manufacturing plant. The predominant BDEs reported in sediment (BDE-47, BDE-99, and BDE-100) are found in the commercial octaBDE product.

Table 3-3. Levels of BDEs reported in ambient air in the United States and other countries

Location	BDE-47	BDE-49	BDE-85	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	BDE-209	Total PBDE	Reference and Comments
Levels Reported in the United States											
Chicago, USA (urban)	33.00	--	--	16.00	2.00	0.53	0.41	--	0.30	52.00	Strandberg et al, 2001; values averaged over a three year period (1997, 1998, and 1999)
Eagle Harbor, USA (rural)	2.90	--	--	2.10	0.29	0.13	0.09	--	<0.10	5.50	
Sturgeon Point, USA (rural)	3.80	--	--	2.80	0.39	0.19	0.11	--	<0.10	7.20	
Sleeping Bear Dunes, USA (rural)	8.40	--	--	5.30	0.80	0.25	0.15	--	<0.10	15.00	
Virginia, USA (rural) (% of total PBDE)	53%	ND	--	38%	4%	3%	2%	--	--	--	Hale et al, 2001; values were extrapolated from a graph, rural air
Levels Reported in Other Countries											
Sweden	<100	--	<6	<60	<9	<4	<2	<0.7	<40	--	Sjödin, 2001a; total air concentration of particle-associated and semivolatile BFR, two samples collected
Sweden	<100	--	--	<50	--	<3	--	<0.4	<20	--	Bergman et al, 1999; ambient outdoor air, two samples, limit of quantification = 10 times the blank sample amount
Ammarnos, Sweden	6.3	--	--	1.6	0.4	--	--	--	ND	--	Bergander et al. (1995) as cited in Palm et al. (2002)
Hoburgen, Sweden	0.7	--	--	0.35	0.07	--	--	--	ND	--	
Japan & Taiwan	--	--	--	--	--	--	--	--	--	7.1-53 (tri-hexa)	Watanabe, et al. (1992) as cited in Palm et al. (2002)
Osaka, Japan	--	--	--	--	--	--	--	--	83-3060	--	
Dunai, Russia	--	--	--	--	--	--	--	--	--	5	Alaee et al. (1999) as cited in Palm et al. (2002)
Tagish, Yukon, Canada	<10-210	--	--	<10-490	--	--	--	--	--	2000	
Alert, Canada	--	--	--	--	--	--	--	--	--	1.0-28.0	
Bobcaygeon, Ontario, Canada	47-650	--	--	<10-330	--	--	--	--	--	88-1300	Gouin et al. (in review) as cited in Palm et al. (2002)
Stoke Ferry, UK	4.0-50.0	--	--	5.5-13	1.1-3.9	--	--	--	--	--	Peters et al. (1999) as cited in Palm et al. (2002)
Hazelrigg, UK	3.2-61.0	--	--	3.1-22.0	0.62-5.4	--	--	--	--	--	

ND = Not Detected
< = did not meet the limit of quantification

Table 3-4. Levels of BDEs reported in sediments (ng/g dw) in the United States and other countries

Location	BDE-47	BDE-49	BDE-99	BDE-100	BDE-153	BDE-154	BDE-190	BDE-209	Total PBDE	DE-71	DE-79	DE-83	Reference and Comments
Levels Reported in the United States													
Virginia and North Carolina, Sediment from stream leaving foam plant, Sample 1	6.37	--	9.47	1.34	--	--	--	--	17.2	--	--	--	Hale et al. (2002)
Virginia and North Carolina, Sediment from stream leaving foam plant, Sample 2	<0.1	--	<0.1	<0.1	--	--	--	--	ND	--	--	--	
Virginia and North Carolina, Sediment from stream leaving foam plant, Sample 3	36.2	--	86.3	9.01	--	--	--	--	132	--	--	--	
Virginia and North Carolina, Pond Sediment, Sample 1	0.5	--	<0.1	<0.1	--	--	--	--	0.5	--	--	--	
Virginia and North Carolina, Pond Sediment, Sample 2	<0.1	--	<0.1	<0.1	--	--	--	--	ND	--	--	--	
Hadley Lake, IN - West	4.7	--	22	2.5	4.2	4.2	<0.02	33	71	--	--	--	Dodder et al. (2002)
Hadley Lake, IN -Middle	0.85	--	1.6	0.32	1.4	1	<0.03	36	41	--	--	--	
Hadley Lake, IN - Middle	1.1	--	2.8	0.53	1.5	1.2	<0.03	27	34	--	--	--	
Hadley Lak, IN - East	0.8	--	1.9	0.36	1.1	1.3	<0.03	19	24	--	--	--	
Virginia, USA (% of total PBDE)	55	0	35	7	2	1	--	--	--	--	--	--	Hale et al. (2001)
Levels Reported in Other Countries													
Sweden - Core depth mm/(date):		--											Nylund, K. et al. (1992); Sediment core from the Baltic Sea
Sweden - Core depth mm/(date): 0-5 (1987)	1.6	--	0.98	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 5-10 (1986)	0.76	--	0.2	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 10-15 (1984)	0.68	--	0.36	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 15-20 (1982)	0.5	--	0.13	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 20-25 (1980)	0.35	--	0.09	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 25-30 (1978)	0.28	--	0.1	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 30-35 (1976)	0.24	--	0.07	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 35-40 (1974)	ND	--	0.05	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 40-45 (1971	0.13	--	0.06	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 45-50 (1967)	ND	--	0.03	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 50-60 (1961)	0.21	--	ND	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 60-70 (1953)	0.15	--	0.07	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 70-80 (1946)	0.12	--	ND	--	--	--	--	--	--	--	--	--	
Sweden - Core depth mm/(date): 80-90 (1939	0.06	--	ND	--	--	--	--	--	--	--	--	--	
Baltic Sea, surface sediment	--	--	--	--	--	--	--	--	0.5	--	--	--	Nylund et al. (1992) as cited in Manchester-Neesvig et al. (2001)
Osaka, Japan, surface sediment	--	--	--	--	--	--	--	--	57.2	--	--	--	Watanabe et al., 1987 as cited in Manchester-Neesvig et al. (2001)

Location	BDE-47	BDE-49	BDE-99	BDE-100	BDE-153	BDE-154	BDE-190	BDE-209	Total PBDE	DE-71	DE-79	DE-83	Reference and Comments
UK, River Tweed	<0.3-0.4	--	<0.6	--	--	--	--	--	--	<0.38	<0.44	<0.6	Allchin et al. (1999) as cited in de Wit (2002)
UK, River Tees, upstream a suspected source/manufacturing plant	<0.3	--	<0.6	--	--	--	--	--	--	<0.38	<0.44	<0.6	
UK, River Tees/Skerne, downstream of confluence	8.0-51.0	--	11.0-85.0	--	--	--	--	--	--	34.0-35.0	25.0-129.0	<0.6-7	
UK, River Skerne, Newton Ayecliffe	239	--	319	--	--	--	--	--	--	130	397	64	
UK, River Skerne, downstream Newton Ayecliffe	68-112	--	111-159	--	--	--	--	--	--	45-68	264-1405	23-294	
UK, Tees estuary	8.9-368	--	16-898	--	--	--	--	--	--	19-366	29-1348	<0.6-9	
UK, River Calder, upstream of sewage plant	2.3-7.6	--	0.6-16	--	--	--	--	--	--	<0.38-6.1	3.0-9.0	<0.6-399	
UK, River Calder, downstream of sewage plant	24	--	46	--	--	--	--	--	--	18	17	3190	
UK, River Ribble	1.2	--	1.7	--	--	--	--	--	--	<0.38	4.4	111	
UK, River Nith	<0.3-1.7	--	<0.6-3.5	--	--	--	--	--	--	<0.38-0.6	<0.44-2	<0.6	
UK, Avonmouth	2.4-3.6	--	2.9-4.7	--	--	--	--	--	--	0.6-1.0	<0.44	<0.6-7	
UK, Great Ouse and Elstow Landfill	0.4-4.2	--	<0.6-5.7	--	--	--	--	--	--	<0.38-1.5	<0.44-13	<0.6	
UK, River Humber	21	--	36	--	--	--	--	--	--	6.6	29	17	
Sweden, Upstream a plastics industry	3.7	--	8.8	1.6	--	--	--	--	14.1	--	--	--	Sellstrom (1996, 1999), as cited in de Wit (2002); Ignition loss % = 67
Sweden, Downstream a plastics industry	780	--	1200	270	--	--	--	--	2250	--	--	--	Sellstrom (1996, 1999), as cited in de Wit (2002); Ignition loss % = 62.5

< = did not meet the limit of quantification

In general, sediment levels reported in Sweden and the United Kingdom appear to be higher than in the United States, ranging from 0.5 to 2250 ng/g dw. BDE-47, BDE-99, and BDE-100 are the predominant analytes in the studies reviewed. The study conducted by Nyland et al. (1992) has been cited extensively as evidence of the increased global use of the commercial products during the past decade. In the United Kingdom, Allchin et al. (1999) reported sediment concentrations of the commercial products Great Lakes DE-71™ (a commercial pentaBDE product), DE-79™ (an octaBDE commercial product), and DE-83™ (an decaBDE commercial product). The concentrations of the commercial products ranged from 0.38 to 366 ng/g dw, 0.44 to 1405 ng/g dw, and 0.6 to 3190 ng/g dw, respectively.

Levels in Water

It is highly unlikely that BDEs will be detected in water because of the hydrophobic nature of this class of compounds. The environmental data reported in the literature are from studies conducted in Japan. A summary of the results, indicating the inability to detect BDEs in water, is presented in Table 3-5. Additional studies (data not reported) in Japan between 1977 and 1989 reported non-detect levels of BDEs in 75-200 water samples (IPCS, 1994). All of the analyses reported hexaBDE, octaBDE, and decaBDE as not detected in water (IPCS, 1994; Darnerud, 2001).

Table 3-5. Levels of BDEs reported in water (µg/L) in Japan [1]

Location	BDE-209	Reference and Comments
Japan, 1977	<0.2-<2.5	EU (2000) as cited in Palm (2002)
Japan, 1987	<0.1	Watanabe and Tatsukawa (1989) as cited in Palm (2002)
Japan, Kino River	<0.1	EU (2000) as cited in Palm (2002)
Japan, 1988	<0.06	EU (2000) as cited in Palm (2002)

< = did not meet the limit of quantification
[1] Data were reported only for BDE-209, and not for other BDE congeners

Levels in Soil

In general, few environmental data describing levels of BDEs in soil in the United States are available in the scientific literature. Table 3-6 summarizes the available U.S. data. Environmental data on levels in soil from other countries were not found in the scientific literature. Soil samples collected by Hale et al. (2002) adjacent to a FPUF manufacturing plant likely represent higher levels than are reasonably expected in rural and, possibly, urban areas in the United States and elsewhere. Total PBDE levels ranged from non-detect to 76 µg/kg dw. BDE-99 was the predominant isomer detected in soil, followed by BDE-47 and BDE-100. The Hale et al. (2002) study does not attribute the occurrence of individual BDEs to the commercial octaBDE product.

Table 3-6. Levels of BDEs reported in soil (µg/kg dw) in the United States

Location	BDE-47	BDE-99	BDE-100	Total PBDE	Reference and Comments
Mid-Atlantic region, USA					Hale et al. (2002)
Soil near FPUF production building	31.6	41.2	3.15	76	
Soil downwind of FPUF plant, Sample 1	8.11	4.75	0.77	13.6	
Soil downwind of FPUF plant, Sample 2	<0.10	<0.10	<0.10	ND	

< = did not meet the limit of quantification
ND = Not Detected

Levels in Food

Two studies conducted in Japan and Sweden report levels of BDEs in different food products as part of market basket surveys of contaminant levels in consumer food products. The only study of food in the United States was conducted by Huwe et al. (2002b), who reported total BDE levels in farm chickens raised in two different regions of the United States. A summary of the available data describing the levels of BDEs found in food in the United States, Japan, and Sweden is presented in Table 3-7. None of the studies found in the literature attribute the occurrence of BDE congeners to the commercial octaBDE product. The predominant BDEs reported in these studies included BDE-28, BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154.

In the study by Huwe et al. (2002b), the concentration of total BDEs in one composite sample of chickens raised in North Dakota was reported as 1.7 ng/g on a whole weight basis. The total BDE concentration in 13 chickens raised in Arkansas and tested individually was 39.4 ng/g on a whole weight basis. The range of results reported by Huwe et al. (2002b), though limited, are 1-2 orders of magnitude higher than the level reported in chickens in Japan (0.00625 ng/g fresh weight, fw) reported by Ohta et al. (2002). In an earlier study by Huwe et al. (2000), chickens fed ball clay and chicken bought at a grocery store were analyzed for total BDEs (i.e., BDE-47, BDE-99, BDE-100, BDE-153, BDE-154, and BDE-183). BDE-99 was the dominant congener in all samples; total BDE concentrations ranged between 4 and 35 ng/g lipid weight in chickens fed ball clay, and 0.5 ng/g lipid weight in store-bought chicken.

The only studies describing the levels of BDEs in other meats and food products available in the scientific literature were conducted in Japan by Ohta et al. (2002) and in Sweden by Darnerud et al. (2000; unpublished). Ohta et al. (2002) reported measurable levels of BDEs in spinach (0.134 ng/g fw), potato (0.048 ng/g fw), carrot (0.038 ng/g fw), pork (0.064 ng/g fw), and beef (0.016 ng/g fw). In general, BDE-47 and BDE-99 were the predominant isomers, followed by BDE-28, BDE-153, BDE-154, and BDE-100. Darnerud et al. (2000; unpublished) reported comparable levels of total BDEs (on a whole weight basis) in Swedish foods. BDEs included in the data reported by Darnerud et al. (2000; unpublished) included BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154 which, with the exception of BDE-47, are found in the commercial octaBDE product.

Table 3-7. Levels of BDEs reported in different foods in the United States and other countries

Location	BDE-28	BDE-47	BDE-99	BDE-100	BDE-153	BDE-154	Total PBDE	Reference and Comments
USA - (ng/g whole weight)								Huwe et al. (2002b); data represent 13 discrete samples of chickens raised in Arkansas and one composite sample of chickens raised in North Dakota.
North Dakota, chicken	--	--	--	--	--	--	1.7	
Arkansas, chicken	--	--	--	--	--	--	39.4	
USA - (ng/g lipid weight)								Huwe et al. (2000)
Chickens fed ball clay	--	--	--	--	--	--	3.6 - 35	
Store-bought chicken	--	--	--	--	--	--	0.5	
Japan - (ng/g fresh weight)								Ohta et al. (2002); data represent approximations derived from graphs.
Spinach	0.023	0.04	0.012	0.003	0.01	0.002	0.134	
Potato	0.004	0.006	0.004	0.001	0.03	0.003	0.0476	
Carrot	0.006	0.012	0.005	0.002	0.012	0.002	0.0384	
Pork	0.002	0.025	0.025	0.006	0.003	0.002	0.0636	
Beef	0.002	0.006	0.002	0.002	ND	0.001	0.0162	
Chicken	ND	0.002	0.002	0.002	ND	0.0005	0.00625	
Sweden (ng/g whole weight)								Darnerud et al. (2000); congeners included BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154; data are unpublished by Darnerud et al.
Fish	--	--	--	--	--	--	0.634	
Meat	--	--	--	--	--	--	0.0458	
Dairy	--	--	--	--	--	--	0.0182	
Egg	--	--	--	--	--	--	0.0425	
Fat/Oil	--	--	--	--	--	--	0.158	
Pastry	--	--	--	--	--	--	0.0925	

ND = Not Detected

Levels in Fish

Relative to other environmental matrices, the levels of BDEs in fish have been studied extensively in the United States and in other countries. Table 3-8 summarizes the available U.S. and Canadian data. The levels of BDEs reported in fish from elsewhere in the world are summarized in Table 3-9. With the exception of one study conducted in the United Kingdom (Allchin et al., 1999), none of the studies found in the scientific literature measure the commercial octaBDE product or attribute the occurrence of individual BDEs to the commercial octaBDE product. The predominant BDEs reported in fish, BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154 are found in the commercial octaBDE product, with the exception of BDE-47.

Table 3-8. Levels of BDEs reported in fish (ng/g ww) in the United States and Canada

Location	BDE-28/33	BDE-47	BDE-49	BDE-66	BDE-99	BDE-100	BDE-153	BDE-154	BDE-181	BDE-183	BDE-155	BDE-190	BDE-209	Total PBDE	Reference and Comments
Levels Reported in the U.S.															
Laurentian Great Lakes, USA - (ng/g wet)															Luross et al. (2002); values are mean concentrations in freshwater fish and represent whole fish tissues
Lake Ontario lake trout	--	58 ± 15	--	1.3 ± 0.38	14 ± 3.5	5.7 ± 1.1	4.9 ± 1.6	--	--	--	--	--	--	--	
Lake Erie lake trout	--	16 ± 4.2	--	0.18 ± 0.12	2.0 ± 0.48	2.5 ± 0.89	0.89 ± 0.14	--	--	--	--	--	--	--	
Lake Huron lake trout	--	27 ± 8.6	--	0.82 ± 0.38	7.7 ± 3.8	3.8 ± 1.8	2.3 ± 0.98	--	--	--	--	--	--	--	
Lake Superior lake trout	--	29 ± 9.8	--	0.89 ± 0.31	12 ± 5.9	4.1 ± 1.3	1.5 ± 0.54	--	--	--	--	--	--	--	
Lake Michigan, USA															Manchester-Neesvig et al. (2001); n = 21 coho and chinook salmon; the average % lipid was 3.89 with a range b/w 1.83-7.19; a 100g "steak" was tested which included skin, muscle, bone, and organ tissues and then blended - the steak was extracted from in front of the dorsal fin.
Salmon - Range	--	26.0-95.1	--	1.2-2.5	5.9-18.9	5.2-18.8	1.8-4.8	2.8-8.5	--	--	--	--	--	44.6-148.0	
Mean	--	52.1	--	1.7	9.3	9.7	2.7	4.5	--	--	--	--	--	80.1	
Washington State, USA															Johnson and Olson (2001); Samples were collected at different times between 9/94 - 7/99; Total PBDEs can be broken down into TBDEs, PeBDEs, and HxBDEs
Rock Island Creek - Rainbow trout (whole)	--	--	--	--	--	--	--	--	--	--	--	--	--	ND	
RI - Rainbow trout (whole)	--	--	--	--	--	--	--	--	--	--	--	--	--	ND	
SR - Rainbow trout (whole)	--	--	--	--	--	--	--	--	--	--	--	--	--	297.0	
Douglas Creek - Rainbow trout (whole)	--	--	--	--	--	--	--	--	--	--	--	--	--	1.4	
Douglas Creek - Rainbow trout (split)	--	--	--	--	--	--	--	--	--	--	--	--	--	1.5	
Douglas Creek - Rainbow trout (split)	--	--	--	--	--	--	--	--	--	--	--	--	--	1.4	
Spokane River - Raibow trout (fillet)	--	--	--	--	--	--	--	--	--	--	--	--	--	20.0	
Spokane River - Raibow trout (fillet)	--	--	--	--	--	--	--	--	--	--	--	--	--	119.0	
Spokane River - Raibow trout (fillet)	--	--	--	--	--	--	--	--	--	--	--	--	--	166.0	
Spokane River - Raibow trout (fillet)	--	--	--	--	--	--	--	--	--	--	--	--	--	174.0	
Sole Duck River - Moutain whitefish (fillet)	--	--	--	--	--	--	--	--	--	--	--	--	--	ND	
Spokane River - Mountain whitefish (whole)	--	--	--	--	--	--	--	--	--	--	--	--	--	1250.0	
Spokane River - Largescale sucker (fillet)	--	--	--	--	--	--	--	--	--	--	--	--	--	120.0	
Spokane River - Largescale sucker (whole)	--	--	--	--	--	--	--	--	--	--	--	--	--	105.0	
Yakima River - Largescale sucker (whole)	--	--	--	--	--	--	--	--	--	--	--	--	--	64.0	
Yakima River - Carp (fillet)	--	--	--	--	--	--	--	--	--	--	--	--	--	22.0	
Snake River - Channel catfish (fillet)	--	--	--	--	--	--	--	--	--	--	--	--	--	8.0	
Columbia River - Starry flounder (whole)	--	--	--	--	--	--	--	--	--	--	--	--	--	30.0	
USA															Dodder et al. (2002); This lake is in close proximity(1.3 km) to a suspected PBDE manufacturing facility
Hadley Lake - white crappie and bluegill	--	13 ± 2	--	--	16 ± 2	7.4 ± 1	15 ± 2	13 ± 1	--	--	--	<0.007	<1.4	65 ± 8	
Hadley Lake - Carp	--	3.20	--	--	0.07	0.89	0.10	2.10	--	--	--	<0.007	<1.4	6.2	
Hadley Lake - Carp	--	9.80	--	--	0.12	3.60	0.04	6.90	--	--	--	<0.006	<1.3	20.0	
Lake of the Ozarks - white crappie and bluegill	--	3.5 ± 0.7	--	--	1.9 ± 0.4	1.1 ± 0.2	0.27 ± 0.11	0.2 ± 0.05	--	--	--	<0.007	<1.4	6.9 ± 1.4	
Lake Superior - Smelt	--	5.7 ± 0.3	--	--	1.8 ± 0.2	0.98 ± 0.09	0.2 ± 0.02	0.45 ± 0.03	--	--	--	<0.007	<1.5	9.1 ± 0.6	
Lake Ontario - Smelt	--	10 ± 1	--	--	5.3 ± 0.7	1.6 ± 0.1	0.49 ± 0.02	0.9 ± 0.05	--	--	--	<0.007	<1.6	18 ± 1	
Michigan, USA															Rice et al. (2002); n=12, composite subsamples of whole fish
Detroit River - Large mouth bass - Mean	--	2.80	--	--	0.48	0.45	0.44	0.43	0.26	0.26	--	--	--	5.25	
Detroit River - Carp - Mean	--	3.00	--	--	0.50	0.48	0.47	0.45	0.24	0.25	--	NA	--	5.39	Rice et al. (2002); n=10, composite subsamples of whole fish

Table 3-8. Levels of BDEs reported in fish (ng/g ww) in the United States and Canada

Location	BDE-28/33	BDE-47	BDE-49	BDE-66	BDE-99	BDE-100	BDE-153	BDE-154	BDE-181	BDE-183	BDE-155	BDE-190	BDE-209	Total PBDE	Reference and Comments
Illinois, USA															Rice et al. (2002); results may be attributable to industrial discharges
Des Plaines River (lower) Carp - Mean	--	2.54	--	--	0.50	0.44	1.01	1.89	3.28	2.99	--	1.75	--	14.40	Rice et al. (2002); n=10, composite subsamples of whole fish
Des Plaines River (upper) Carp - Mean	--	1.34	--	--	0.50	0.49	0.66	0.98	1.44	1.31	--	0.97	--	7.68	Rice et al. (2002); n=4, composite subsamples of whole fish
Buffalo, New York															Loganathan et al. (1995) as cited in Manchester-Neesvig et al. (2001)
Carp Muscle	--	--	--	--	--	--	--	--	--	--	--	--	--	18.7	
Virginia, USA															Hale et al. (2001); values were extracted and approximated from a graph, Number of samples: Catfish n=15; flathead n=20; carp n=5; stiped n=24; white n=6
Range (% of total PBDEs)	--	45-74%	2-12.0%	--	0-27%	16-24%	0-7%	3.0-5.0%	--	--	--	--	--	--	
Levels Reported in Canada.															
Canada															Easton et al. (2002); NDR = Peak detected, but did not meet quantification criteria, ND = Not detected
Salmon feed	0.061; 0.067	0.84; 1.1	0.19; 0.17	--	0.13; 0.18	0.17; 0.23	0.065; 0.038	0.17; 0.48	--	--	0.14; 0.019	--	--	1.875; 1.902	Easton et al. (2002); NDR = Peak detected, but did not meet quantification criteria, ND = Not detected; 2 samples were taken at different locations. Both are listed.
Farmed Salmon	0.036; 0.11	0.69; 2.6	0.11; 0.21	--	0.14; 0.39	0.13; 0.47	NDR; 0.08	0.041; 0.13	--	--	0.016; 0.067	--	--	1.187; 4.147	
Wild Salmon	NDR-0.019	0.029-0.28	NDR-0.029	--	NDR-0.097	0.004-0.043	ND-0.003	ND-0.005	--	--	NDR-0.003	--	--	0.039-0.485	Easton et al. (2002); NDR = Peak detected, but did not meet quantification criteria, ND = Not detected; 4 samples were taken at different locations. Both are listed.

ND = Not Detected
NDR = Peak detected, but did not meet quantification criteria (Easton et al., 2002)
NA = Not Applicable
< = did not meet the limit of quantification

Table 3-9. Levels of BDEs reported in fish in other countries.

Location	BDE-28	BDE-47	BDE-99	BDE-100	BDE-153	BDE-154	DE-71	DE-79	Total PBDE	Reference and Comments
Levels Reported in Other Countries										
(ng/g ww)										
Klosterfjorden, Sweden										Andersson et al. (1981) as cited in Manchester-Neesvig et al. (2001)
Sea Trout	--	--	--	--	--	--	--	--	15.0	
Viskan River, Sweden										
pike muscle	--	--	--	--	--	--	--	--	124.0	
pike liver	--	--	--	--	--	--	--	--	9680.0	
Baltic Sea										
Salmon muscle	--	--	--	--	--	--	--	--	14.0	Asplund et al. (1999) as cited in Manchester-Neesvig et al. (2001)
Salmon egg	--	--	--	--	--	--	--	--	9.0	
Salmon blood	--	--	--	--	--	--	--	--	6.0	
Wakayama, Japan										
Sardine	--	--	--	--	--	--	--	--	0.8	Watanabe et al. (1987) as cited in Manchester Neesvig et al. (2001)
North Sea										
North - cod liver	--	--	--	--	--	--	--	--	26.0	de Boer. (1989) as cited in Manchester-Neesvig et al. (2001)
Central - cod liver	--	--	--	--	--	--	--	--	54.0	
South - cod liver		--	--	--	--	--	--	--	170.0	
(ng/g lipid weight)										
North Sea										Boon et al. (2002)
Herring filet - Mean	1.9	37	12	9.2	0.9	1.5	--	--	--	
Range	1.2-2.4	23-47	9.9-17	6.3-12	0.6-1.3	1.3-1.9	--	--	--	
Cod filet - Mean	2.7	43	6.3	13	<LOD	3.9	--	--	--	
Range	1.5-4.5	26-74	3.1-16	5.9-21	<LOD	3.9-3.9	--	--	--	
Whiting filet - Mean	1.8	26	9	8.6	<LOD	3.3	--	--	--	
Range	1.3-2.4	7.1-40	5.3-14	4.2-12	<LOD	2.2-4.4	--	--	--	
Herring liver - Mean	2.1	30	13	9.1	2.1	2.6	--	--	--	
Range	1.6-2.5	19-52	8.0-21	5.6-17	1.1-3.9	1.5-4.4	--	--	--	
Cod liver - Mean	6.7	133	15	40	0.7	6.4	--	--	--	
Range	2.0-12	63-307	1.4-53	18-93	0.5-1.3	4.3-12	--	--	--	
Whiting liver - Mean	3.6	70	15	16	1.4	4.5	--	--	--	
Range	0.7-6.3	7.6-132	1.9-34	1.7-31	0.3-3.1	0.6-11	--	--	--	
Baltic Sea										Bergman et al., Cambridge Isotope Labs
Salmon muscle	--	200.0	54.0	47.0	--	--	--	--	--	
Sweden - Baltic Sea										Bergman et al. (1999)
Salmon - Mean	--	201	55	--	--	--	--	--	--	
Range	--	103-411	26-74	--	--	--	--	--	--	
Herring - Homogenate	--	83	27	--	--	--	--	--	--	

Table 3-9. Levels of BDEs reported in fish in other countries.

Location	BDE-28	BDE-47	BDE-99	BDE-100	BDE-153	BDE-154	DE-71	DE-79	Total PBDE	Reference and Comments
Sweden										Sellstrom et al. (1993)
Herring Muscle-Bothnian Sea Nov. 1986	--	82	27	--	--	--	--	--	--	
Baltic Proper June 1987	--	450	46	--	--	--	--	--	--	
Skagerrak April 1987	--	59	9.8	--	--	--	--	--	--	
Fladen Nov. 1987	--	12	3.4	--	--	--	--	--	--	
Utlangan Sept. 1987	--	38	17	--	--	--	--	--	--	
Landsort Oct. 1987	--	35	9.2	--	--	--	--	--	--	
Angskarsklubb Sept.-Oct 1987	--	27	17	--	--	--	--	--	--	
Harufjarden Oct 1987	--	19	7.8	--	--	--	--	--	--	
Bream muscle - River Viskan Spring 1987	--	250	2.3	--	--	--	--	--	--	
Bream muscle - River Viskan Spring 1987	--	750	2.4	--	--	--	--	--	--	
Pike muscle - River Haggen Spring 1987	--	6500	1100	--	--	--	--	--	--	
Pike muscle - River Viskan Spring 1987	--	2000	78	--	--	--	--	--	--	
Perch muscle - River Viskan Spring 1987	--	24000	9400	--	--	--	--	--	--	
Perch muscle - River Viskan Spring 1987	--	2200	380	--	--	--	--	--	--	
Trout muscle - Kesnacksalven Fall 1988	--	460	590	--	--	--	--	--	--	
Trout muscle - Bengtsbroholjen Fall 1988	--	120	130	--	--	--	--	--	--	
Trout muscle - Kesnacksalven Fall 1988	--	140	130	--	--	--	--	--	--	
Trout muscle - Bengtsbroholjen Fall 1988	--	250	220	--	--	--	--	--	--	
Trout muscle - Skifors Fall 1988	--	190	64	--	--	--	--	--	--	
Pike muscle - Kesnacksalven Fall 1988	--	98	79	--	--	--	--	--	--	
Pike muscle - Bengtsfors Fall 1988	--	94	60	--	--	--	--	--	--	
Arctic char muscle - Lake Vattern May 1987	--	400	64	--	--	--	--	--	--	
Whitefish muscle - Lake Storvindeln Nov. 1986	--	15	7.2	--	--	--	--	--	--	
Great Britain										Allchin et al. (1999) as cited in de Wit (2002)
Tees Bay - plaice, flounder and dab	--	520-9500	83-370	--	--	--	920-1200	500-1200	--	
Lune/Wyre (off River Calder) - flounder	--	400	54	--	--	--	100	120	--	
Nith estuary - flounder	--	73-120	ND-19	--	--	--	47-120	ND-83	--	
Bay (off Avonmouth) - paice, flounder, and dab	--	ND-370	ND-100	--	--	--	94-120	ND-970	--	
The Wash (off Great Ouse) - dab	--	380	74	--	--	--	110	58	--	
Off River Humber	--	1600	160	--	--	--	110	900	--	

ND = Not Detected

< LOD = did not meet the limit of quantification

Based on seven U.S studies reported in the scientific literature, total BDEs in either whole or fillet fish tissues range from non-detect to 1250 ng/g wet weight (ww) in the United States. As shown in Table 3-10, the 95th percent upper confidence limit of the mean was 71.67 ng/g ww using the few available U.S. fish data reporting BDEs in edible fish tissues. By comparison, total BDEs in either whole or fillet fish tissues ranged between 6-9680 ng/g ww in Sweden, including fish from the Baltic Sea, and from 0.04 to 4 ng/g ww in Canada. Fish data reported in other measurement units are not included in this data range.

In general, fish levels in the United States are comparable to those reported in Europe and higher than levels reported in Canada. However, data in the U.S. and Canada are limited, and results reported in the published literature describe different BDEs and use different units of measure (e.g., whole wt., wet wt., and lipid wt.) that make direct comparisons difficult. A Swedish market basket study reported the total concentration of five BDE congeners as 0.0634 ng/g whole weight in store-bought fish. A similar market basket study has not been conducted in the United States. For the purposes of the Tier 1 assessment, the exposure model addressing fish consumption uses the calculated 95th percent upper confidence limit of the mean, 71.67 ng/g ww, for fish fillets found in the United States. This assumption is substantially higher than the levels of BDEs reported in most fish studies in the United States. The only study to report on levels of the commercial octaBDE product was conducted by Allchin et al. (1999) in the United Kingdom. The concentrations of Great Lakes DE-79™ ranged between non-detect and 1200 ng/g lipid weight.

Table 3-10. Levels of BDEs in the edible tissues of fish in the United States

Fish	Sample Size	Total BDE (ng/g ww) [1]	Reference and Comments
Spokane River - Rainbow trout (fillet)	n=1	20.0	Johnson and Olson (2001)
Spokane River - Rainbow trout (fillet)	n=1	119.0	
Spokane River - Rainbow trout (fillet)	n=1	166.0	
Spokane River - Rainbow trout (fillet)	n=1	174.0	
Soleduck River - Mountain whitefish (fillet)	n=5	1.05*	
Spokane River - Largescale sucker (fillet)	n=1	120.0	
Yakima River - Carp (fillet)	n=5	22.0	
Snake River - Channel catfish (fillet)	n=5	8.0	
Columbia River - Starry flounder (whole)	n=5	30.0	
Hadley Lake -Carp	n=1	6.2	Dodder et al. (2002)
Hadley Lake -Carp	n=1	20.0	
Buffalo, NY - Carp Muscle	n=45, mean	18.7	Loganathan et al (1995) as cited in Manchester-Neesvig et al. (2001)
95th % UCL of the mean	**--**	**71.67**	Calculated by ENVIRON

*Value represents half the detection limit

[1]. Where sample size is greater than one, total BDE levels represent the authentic mean

Levels in Indoor Air

Data on indoor air levels of BDEs in U.S. homes, schools, or the workplace are not reported in the scientific literature. The few available data describing levels in the workplace are summarized in Table 3-11 from studies conducted in Sweden and Norway by Sjödin et al. (2001a), Bergman et al. (1999) and Thomsen et al. (2001a). Indoor air concentrations of BDE-47, BDE-85, BDE-99, BDE-100, BDE-153, BDE-154, BDE-183, and BDE-209 were tested in an electronics dismantling hall, computer repair facilities, laboratory, around electronics shredders, and during the assembly of circuit boards. The levels of BDE-183 and BDE-209 were more prevalent than other BDEs, with the exception of laboratory air where these BDEs were not analyzed. The mean concentrations of seven BDEs in 12 samples from a dismantling hall ranged from 1.2 to 19 ng/m^3; at the shredder, BDE concentrations in two samples ranged from 0.4 to 87 ng/m^3 (Sjödin et al. 2001a). In the dismantling hall, BDE-183 and BDE-209 concentrations ranged from 6 and 44 ng/m^3 and between 12 and 71 ng/m^3, respectively. Air concentrations were highest in samples collected by Sjödin et al. (2001a) adjacent to the electronic equipment shredder.

Indoor air levels are also reported in teaching halls and offices in Sweden (Sjödin et al., 2001a). Only two samples were taken in the teaching hall that contained 20 computers. Both results are listed on Table 3-11. Congeners contained within the commercial octaBDE product such as BDE-85, BDE-99, BDE-100, BDE-153, and BDE-154 were detected and found at levels of 0.05 ng/m^3, 0.4 ng/m^3, 0.01 ng/m^3, 0.01 ng/m^3, and 0.02 ng/m^3 respectively. However, the levels of these congeners did not meet the level of quantification in the office samples. These were the only data reported in the literature.

Levels in Indoor Dust

Two studies report levels of BDEs in indoor dust. Knoth et al. (2002) collected dust samples from household vacuum cleaners in Germany. Santillo et al. (2001) collected 13 samples from vacuum cleaners used in European parliament buildings plus 3 similar samples from the offices of an Internet provider in the Netherlands. None of the studies attribute the occurrence of BDE congeners to the commercial octaBDE product. Congeners BDE-47, BDE-99, BDE-100, BDE-153, BDE-154, and BDE-209 predominated in all of the indoor dust samples. The data are presented in Tables 3-12 and 3-13.

Table 3-11. Levels of BDEs in indoor air (ng/m^3)

Location	BDE-47	BDE-85	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	BDE-209	Reference and Comments
Sweden									
In a dismantling hall	1.2	0.17	2.6	0.25	3.9	0.57	19	36	Sjodin (2001a); total air concentration of particle-associated and semivolatile BFR, Only two samples (n=2) were collected around shredder - both concentrations are listed
Range	0.35-2.1	0.10-0.24	0.54-5.5	0.063-0.52	0.88-11	0.13-1.0	6.3-44	12.0-70	
Around a shredder	2.0;2.1	0.42;0.52	4.0;4.5	0.56;0.61	14;15	2.4;2.4	84;87	150;200	
During the assembly of circuit boards	0.35	--	0.15	0.041	0.019	0.0069	0.053	0.22	
Range	<0.1-0.39	<0.006	<0.06-0.15	<0.009-0.058	<0.004-0.033	<0.002-0.013	0.014-0.11	<0.04-0.32	
In a computer repair facility	<0.10	<0.006	<0.06	<0.009	<0.004;0.014	<0.002;0.0043	0.048;0.063	<0.04;0.093	
Sweden - Dismantling of Electronics									Bergman et al. (1999); (n=12); limit of quantification was determined as 10 times the blank sample amount
Mean	1.225	--	2.621	--	3.963	--	18.967	36.83	
Range	0.358-2.106	--	0.57-5.583	--	0.909-11.043	--	6.347-43.77	11.63-70.752	
Sweden									Sjodin (2001a); total air concentration of particle-associated and semivolatile BFR, Only two samples (n=2) were collected in the teaching hall - both concentrations are listed
- In an office with computers									
Mean	--	--	--	--	--	--	0.0082	0.083	
Range	<0.1	<0.009	<0.06	<0.006	<0.002	<0.004	0.0046-0.012	<0.04-0.087	
- In a taching hall									
sample 1	0.72	0.053	0.35	0.0085	0.012	0.022	0.011	<0.04	
sample 2	0.8	0.059	0.41	0.011	0.013	0.023	0.012	0.17	
Norway									Thomsen et al. (2001a);samples were taken by adsorption to glass at ordinary laboratory circulation and by pumping air through a SPE column.
Laboratory Air - Range	0.012-0.059	--	0.007-0.02	<0.0045	<0.0045	<0.0045	--	--	

Table 3-12. Levels of BDEs in indoor dust from homes in Germany (ng/g)

Sample Number	Total PBDEs	Reference and Comments
S040	2002	Knoth et al. (2002)
S041	1328.9	
S042	4302.1	
S043	204.6	
S045	490.2	
S046	943.8	
S047	309.9	
S048	666.8	
S049	145	
S050	208.1	
S051	837.2	
S052	451.2	
S053	286.6	
S054	271.2	
S055	282.2	
S056	694	
S057	451.1	
S058	19124.2	
S059	624.7	
S060	16.3	
S061	302.5	
S062	6774.2	
S063	2707.8	
S064	233.9	
S065	309	
95 % UCL on the mean	**410**	**Calculated by ENVIRON**

Table 3-13. Levels of BDEs in indoor dust from European Parliament buildings (ug/g)

Location	BDE-28	BDE-47	BDE-66	BDE-71	BDE-75	BDE-77	BDE-85	BDE-99	BDE-100	BDE-119	BDE-138	BDE-153	BDE-154	BDE-190	BDE-209	Reference and Comments
Netherlands	<0.16	97	<0.16	<0.16	0.49	<0.17	7.4	130	30	<0.16	3.5	48	11	<0.16	800	Santillo et al. (2001); samples were taken from vacuum cleaner bags after cleaning offices or parliament buildings
Finland	1.6	180	2.4	<0.60	<0.60	<0.62	7.5	160	36	<0.59	1.9	22	9.4	<0.60	1100	
Sweden	0.95	78	1.6	<0.36	<0.36	<0.38	3.1	68	19	<0.36	<0.36	9.8	5	<0.36	700	
Italy 1	2.8	89	2.3	<0.26	<0.26	<0.27	3.3	59	15	<0.26	2.3	21	5.4	<0.26	6900	
Italy 2	1.5	110	2.7	<0.20	<0.20	<0.21	11.2	170	23	<0.20	4.7	59	9.2	<.20	4600	
Denmark 1	0.47	21	0.48	<0.29	<0.29	<0.30	1.6	27	5	<0.28	<0.28	6.1	5.2	<0.29	470	
Denmark 2	0.91	39	0.88	<0.26	<0.26	<0.27	2.2	40	8.3	<0.26	0.76	8.5	3	<0.26	330	
Netherlands (Office) 1	<0.16	15	0.58	<0.16	<0.16	<0.17	0.9	15	3.9	<0.16	0.89	17	2.3	<0.16	490	
Netherlands (Office) 2	<0.17	10	0.5	<0.17	<0.17	<0.18	<0.06	10	2.5	<0.17	<0.17	6.3	<0.43	<0.17	330	
Netherlands (Office) 3	<0.12	17	0.69	<0.12	<0.12	<0.12	0.99	14	3.4	<0.12	0.49	13	1	<0.12	260	
Austria 1	2.8	66	2.3	<0.03	<0.04	<0.04	5.5	68	26	<0.03	1.6	26	11	<0.08	340	
Austria 2	3	64	<0.04	<0.04	<0.04	<0.04	5.5	72	23	<0.04	<0.04	18	9.4	<0.09	510	
Germany 1	6.9	80	17	<0.04	<0.04	<0.04	2.9	50	14	<0.04	<0.04	17	6.3	<0.09	1500	
Germany 2	<0.03	8.6	<0.03	<0.03	<0.03	<0.03	0.87	12	3.4	<0.03	<0.03	4.8	<0.07	<0.06	290	
UK 1	16	320	36	<0.09	<0.09	<0.10	6.6	92	22	<0.09	<0.09	31	8.9	<0.2	4500	
UK 2	<0.08	19	<0.08	<0.08	<0.08	<0.08	2.2	29	8.2	<0.08	<0.08	11	<0.19	<0.17	550	

< = did not meet the limit of quantification

Natural Sources of BDEs in the Environment

The information presented is further described in ECB (2000), which summarizes the available information presented in this section regarding natural sources of BDEs in the environment. Several brominated compounds that are structurally similar to the BDEs have been found in some marine species, especially marine sponges (Faulkner, 1988; Gribble, 2000). The compounds identified by Faulkner (1988) and Gribble (2000) are characterized by the diphenyl ether ring structure and contain an additional hydroxyl and methoxy groups on one or both of the aromatic rings. Many of the compounds have been shown to possess anti-microbial properties (Sharma et al, 1969). Carte and Faulkner (1981) isolated several substituted BDE compounds from marine sponges *(Dysidea herbacea, Dysidea chlorea* and *Phyllospongiafoliascens).* The compounds identified were:

from *D.heracea*

- 2-(2',4'-dibromophenoxy)-3,4,5-tribromophenol,
- 2-(2',4'-dibromophenoxy)-4,5,6-tribromophenol, and
- 2-(2',4'-dibromophenoxy)-3,5-dibromophenol;

from *D.chlorea*

- 2-(2',4'-dibromophenoxy)-4,6-dibromophenol;

from *P. foliascens*

- 2-(3',5'-dibromo-2'-methoxy-phenoxy)-3,5-dibromoanisole,
- 2-(3',5'-dibromo-2'-hydroxyphenoxy)-3,5,6-tribromophenol, and
- 2-(3',5'-dibromo-2'-hydroxyphenoxy)-3,4,5,6-tetrabromophenol.

Similar compounds have been isolated from *Dysidea* species by Salva and Faulkner (1990), Norton and Wells (1980), Norton et al. (1981), Fu et al. (1995), Llîn et al. (1996) and Anjaneyulu et al. (1996). Generally, compounds with between 4 and 6 bromine atoms/molecule have been detected. Salva and Faulkner (1990) found that the brominated compounds appeared to be found only in the tropical species of *Dysidea* that also contained large populations of cyanophytes in their tissues. Unson et al. (1994) demonstrated that the presence of 2-(2',4'-dibromophenyl)-4,6-dibromophenol in *Dysidea her-bacea* was associated with the symbiotic filamentous cyanobacterium (similar to *Oscillatoria spongeliae)* present within the organism, rather than the sponge cells, and concluded that the brominated compounds are biosynthesized by the cyanobacterium.

Similar compounds are produced by the acorn worm *Ptychodera flava laysanica* from Hawaii (Higa and Scheuer, 1977) and the green alga *Cladophora fascicularis* (Kuniyoshi et al. 1985) in

marine waters around Japan. Species of the green algal genus *Cladophora* are known to occur in a variety of marine and freshwaters, including the Baltic Sea (Dodds and Gudder, 1992).

It is possible that some of these naturally occurring brominated compounds may cause interferences in analytical methods used to detect BDEs in the environment. Since the natural brominated compounds generally have between 4 and 6-bromine atoms/molecule, this interference is likely to be a consideration only in the determination of the levels of pentaBDEs. For example, in a recent paper by Haglund et al. (1997) both pentaBDEs and methoxy-BDEs were detected in biotic samples using GC-MS. The study confirmed the presence of 2,2',4,4'-tetrabromodiphenyl ether, 2,2',4,4',5- and 2,2',3,4,4'-pentabromodiphenyl ether and 2,2',3,4,4',5'-hexabromodiphenyl ether in samples of ringed and grey seals and herring from along the Swedish coast, and also salmon, fish oil and human adipose tissue by comparison of the mass spectra with that from reference material. The source of the methoxy-derivatives found in the environment was not identified, but the possibility of a natural source of these compounds could not be ruled out.

3.9 Summary of Available Human Data

Levels in Human Breast Milk

The available data reported in the scientific literature describing the levels of various BDEs in breast milk from women in the United States and other countries are summarized in Table 3-14. The current understanding of BDE levels in human breast milk is based primarily on the results of monitoring studies reported during the past decade in Sweden. The most frequently reported study is that by Meironyté et al. (1999), who reported that BDE levels in breast milk from primaparous women in Sweden doubled approximately every five years between 1972-1997. More recently, however, Meironyté-Guvenius and Norén (2001) reported that BDE levels appear to be declining in monitoring data collected during the period 1998-2000. Similar results are reported by Strandman et al. (2000) in a study of Finnish women. These and other studies reported in the literature do not attribute the occurrence of BDE congeners to the commercial octaBDE product. Congeners BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154 are typically the primary congeners reported in the various studies. The data clearly indicate that BDE-47 is the predominant congener in breast milk, followed by BDE-99, BDE-100, and BDE-153.

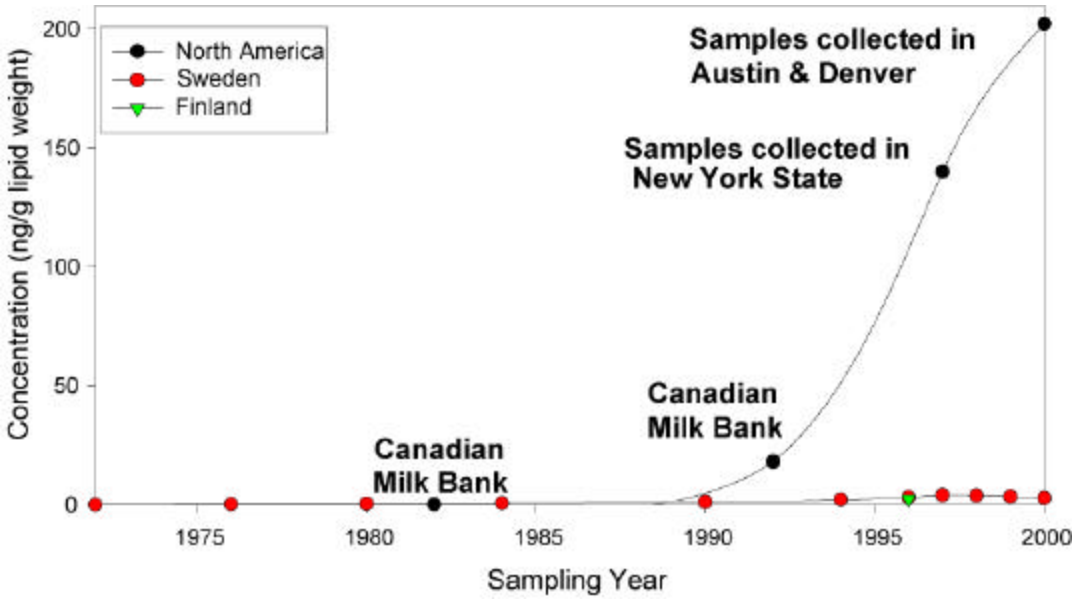
Table 3-14. Levels of BDEs in breast milk of women from the United States and other countries (ng/g lipid weight)

Location	BDE-17	BDE-28	BDE-47	BDE-66	BDE-85	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	Total PBDE	Reference and Comments
United States												Päple et al. (2001); composite sample - Samples were collected in Austing and Denver, USA, and sent to a laboratory in Hamburg, Münster, and Stockholm.; other congeners detected, but not individually reported are BDE-17 and BDE-138
Hamburg Laboratory results												
using LRMS, NCI	--	2.92	112	0.92	3.18	29.9	23.6	14.2	3.4	0.17	190.6	
using HRMS, EI	--	3.14	124	0.53	----	35.4	25.1	14.1	1.6	0.16	204.1	
Münster Laboratory results - using HRMS, EI	--	5.4	122	1.2	2.6	21.7	24.7	17.2	1.4	0.2	196.4	
Stockholm Laboratory results - using HRMS,EI	--	5	150	0.5	3	23	21	14	1	0.1	217.6	
Vancouver, Canada												Ryan et al. (2002); Congeners included BDE-28, BDE-47, BDE-99, BDE-100, BDE-153, BDE-154, and BDE-183; n=20 samples collected between 2001-2002
Mean	--	--	--	--	--	--	--	--	--	--	42.8	
Range	--	--	--	--	--	--	--	--	--	--	0.9-281.9	
Canada												Ryan and Patry (2001); n=10, samples collected in 1992 from Ontario and Quebec, other congeners were present but in lesser
Mean	--	0.22	3.39	--	--	1.19	0.44	0.41	--	0.15	5.79	
Stockholm, Sweden												Meironyté-Guvenius et al. (2003); samples were collected in 2000-2001 from 15 mothers ranging in age from 28-38 years old.
Median	<0.01	0.06	1.15	0.02	0.04	0.21	0.14	0.32	0.02	0.01	2.14	
Range	<0.01	0.02-0.18	0.26-4.01	<0.01-0.07	<0.01-0.17	0.07-2.2	<0.01-0.69	0.03-1.16	<0.01-0.14	<0.01-0.14	0.56-7.72	
Sweden												Meironyté-Guvenius and Norén (2001)
2000	--	0.08	1.7	0.02	0.03	0.23	0.22	0.45	0.02	0.05	2.79	
1999	--	0.1	1.97	0.03	0.05	0.43	0.24	0.54	0.04	0.07	3.46	
1998	--	0.1	2.29	0.03	0.06	0.6	0.31	0.47	0.02	0.02	3.88	
Sweden												Meironyté et al. (1999); % lipids ranged between 2.5 and 3.6%.
1997	--	0.19	2.28	0.07	0.07	0.48	0.42	0.46	0.05	--	4.02	
1996	--	0.12	2.08	0.05	0.05	0.41	0.15	0.24	0.01	--	3.11	
1994	--	0.13	1.48	0.04	ND	0.26	0.09	0.15	0.02	--	2.17	
1990	--	0.03	0.81	0.02	ND	0.15	0.06	0.1	0.04	--	1.21	
1984/1985	--	0.03	0.49	ND	ND	0.08	0.06	0.05	0.02	--	0.73	
1980	--	0.03	0.28	ND	ND	0.09	0.04	0.03	0.01	--	0.48	
1976	--	0.04	0.18	0.01	ND	0.04	0.05	0.02	0.01	--	0.35	
1972	--	ND	0.06	ND	ND	ND	ND	0.01	ND	--	0.07	
Sweden												Darnerud et al. (1998); n = 39
Mean	--	--	2.516	--	--	0.717	0.475	0.648	0.07	--	4.452	
Range	--	--	0.331-16.1	--	--	0.181-4.47	0.06-5.140	0.255-4.320	0.03-0.27	--	1.139-28.17	
Mean (pg/g fresh weight)	--	--	77	--	--	24	14	19	2.1	--	137	
Range (pg/g fresh weight)	--	--	8-358	--	--	4-222	1.5-114	8.0-96	1.5-6	--	26-626	
Sweden - Mean (Range)												Hagmar and Bergman (2001)
Pooled samples from 40 primiparas	--	--	1.7	--	--	0.23	--	--	--	--	--	
Primiparous women	--	--	1.8 (0.3-16)	--	--	0.44 (0.18-4.5	--	--	--	--	--	
Finland												Strandman et al. (2001); Human milk and placenta samples collected between 1994-1998 were analyzed from eleven (n=11) donors between 25-42 years old (median age = 34); 6 samples were from donors of their first childbirth, 1 from a donor of her second, 1 from a donor of her third, 2 from donors of their fourth chilbirth, and 1 was unknown.
Mean	--	0.16	1.31	--	--	0.39	--	0.39	--	--	--	
Range	--	0.04-0.59	0.3-4.25	--	--	0.14-0.94	--	0.19-0.72	--	--	--	

ND = Not Detected

At present, very limited data on the levels of BDEs in breast milk from women in North America are available, and only two data points describing BDE levels in U.S. women are reported in the scientific literature. As shown in Figure 3-1, these data indicate higher BDE levels in breast milk from women in both Canada and the United States than in women from Sweden and Finland. The concentration of PBDEs in one composite sample of breast milk reported by Päpke et al. (2001) in the United States from women in Denver, Colorado, and Austin, Texas, suggests that BDE concentrations may be higher than reported by Meironyte et al. (1999). According to Päpke et al. (2001), levels in the United States are 40 times higher than the concentrations reported in Sweden.

Figure 3-1. Comparison between the concentrations of BDEs reported in human milk from North America and Europe [1]



[1]. Results from the Canadian Milk Bank and New York State are from Ryan and Patry (2001); results from Denver and Austin are from Päpke et al. (2001); Swedish results are from Meironyté-Guvernius and Norén (2001), and Finnish data are from Strandman et al. (2000).

As shown in Figure 3-1 and summarized in Table 3-14, levels of BDEs in breast milk from Swedish women range from 0.07 to 626 ng/g lipid weight (Meironyté-Guvernius et al., 1999, 2003; Meironyté and Norén, 2001; Darnerud et al., 1998); in the United States, levels range from 191 to 218 ng/g lipid weight (Päpke et al., 2001). The results of breast milk monitoring conducted by Ryan and Patry (2001) in Canada are lower than the concentrations reported by Päpke et al. (2001) in the United States, ranging from 0.9-282 ng/g lipid weight for total BDEs (Ryan and Patry, 2001).

Data are not reported in the literature describing BDE levels in the human fetus. Using data on the levels of total BDEs (including BDE-28, BDE-47, BDE-99, and BDE-153) in human breast milk, Strandman et al. (2000) calculated possible levels in the human placenta based on an average lipid content of 0.5%. According to Strandman et al. (2000), levels in the placenta would be similar to that in breast milk; concentrations ranged from 0.88-5.89 ng/g lipid weight in breast milk and 1-4.4 ng/g lipid weight in placenta samples. Strandman et al. (2000) observed that

lower brominated BDEs were more likely to be transferred from the mother to the fetus, based on data showing BDE-47 as the most predominant BDE in human milk.

In a more recent study, Mieronyté-Guvenius et al. (2003) colleted cord blood plasma at partus, maternal blood, and breast milk samples from 15 mothers living in Stockholm, Sweden. Similar levels were measured to those reported by Strandman et al. (2000). Total PBDE concentrations ranged from 0.46-4.28 ng/g lipid weight with a median of 1.69 ng/g lipid weight for cord blood plasma, 0.71-8.39 ng/g lipid weight with a median of 2.07 ng/g lipid weight for maternal blood plasma, and 0.56-7.72 ng/g lipid weight with a median of 2.14 ng/g lipid weight for breast milk. Levels of individual congeners in cord blood plasma were the same or lower than the concentrations reported in maternal blood plasma or breast milk.

Levels in Human Blood Serum

In contrast to studies conducted in Sweden, Norway, and Japan, few data are available describing levels of BDEs in blood serum in the U.S. population. A summary of the data available in the scientific literature describing the concentrations of BDEs in human blood serum is presented in Table 3-15. Similar to studies describing levels in human breast milk, the available studies do not attribute the occurrence of BDE congeners to the commercial octaBDE product. Congeners BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154 are the primary congeners reported in various studies; other congeners, such as BDE-183 and BDE-209, have been detected, but not at appreciable levels.

The results of one U.S study of blood serum collected from a blood donor facility located in Illinois conducted by Sjödin et al. (2001b) reported BDE concentrations ranging from 0.39-24 ng/g lipid weight for BDE-47, 0.23-3.8 ng/g lipid weight for BDE-99, 0.11-24 ng/g lipid weight for BDE-100, and 0.08-2 ng/g lipid weight for BDE-153. Similar levels are reported in Sweden, Norway, and Japan (Bergman et al., 1999; Hagmar and Bergman, 2001; Jakobsson et al., 2002; Thomsen et al., 2001b; and Nagayama et al., 2001).

Levels in Human Adipose Tissues

A summary of the data available in the scientific literature describing the concentrations of BDEs in human adipose tissue is presented in Table 3-16. Similar to studies describing levels in human breast milk and blood serum, the available studies do not attribute the occurrence of BDE congeners to the commercial octaBDE product. Congeners BDE-47, BDE-99, BDE-100, BDE-153, and BDE-154 are the primary congeners reported in various studies. She et al. (2002) reported BDE levels in breast adipose tissue ranging from 1 to as high as approximately 196 ng/g lipid weight. For the individual BDEs measured, She et al. (2002) reported that in the San Francisco Bay Area, USA, levels ranged from 7.01-196.0 ng/g lipid weight for BDE-47, 2.17-72.2 ng/g lipid weight for BDE-99, 0.77-60.6 ng/g lipid weight for BDE-100, 1.52-124.0 ng/g lipid weight for BDE-153, and 2.86-70.5 ng/g lipid weight for BDE-154. According to She et al.

(2002), a strong correlation could be established between levels in breast adipose tissue and abdominal adipose tissue.

Table 3-15. Levels of BDEs in human blood serum in the U.S. general population and in other countries (ng/g lipid weight)

Location and Population	BDE-17	BDE-28	BDE-37	BDE-47	BDE-66	BDE-77	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	BDE-203	BDE-209	OctaBDE	OctaBDE	OctaBDE	NonaBDE	NonaBDE	NonaBDE	Total PBDEs	Reference and Comments
Levels in the U.S Population																					
Illinois, USA																					Sjodin et al. (2001b); samples were collected in 1988 from blood donors; octaBDE and nonaBDE concentrations were estimated from the instrument
Median	--	--	--	0.64	--	--	0.32	0.2	0.35	--	0.18	<0.1	<1	0.13	0.78	0.1	<0.1	0.39	<0.1	--	
Range	--	--	--	0.4-24	--	--	0.2-3.8	0.1-24	0.08-2.0	--	0.09-1.3	<0.1-0.2	<1-34	<0.1-0.3	0.17-1.7	<0.1-0.15	<0.1-0.37	0.11-2.1	<0.1-0.29	--	
San Francisco, USA																					Petreas et al. (2003); samples were collected from 1997-1999 from 50 Laotian immigrant women between the ages of 19 and 40 years old from a study on organochlorine exposures and menstrual cycle function. Samples between 1959-1967 were collected from 420 pregnant women and used for historic comparison
1997-1999	--				--																
Mean	--	--	--	50.6 ± 94.6	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	
Median (Range)	--	--	--	10 (<10-511)	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	
1959-1967	--				--																
Mean	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	
Median (Range)	--	--	--	<10	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	
Levels in Populations from Other Countries																					
Japan																					Nagayama et al. (2001); subjects included 54 Japanese adults aging from 37-49 years old with a mean of 43.9 yrs.
Mean (Range)	--	--	0.032 (ND-0.1)	0.52 (0.1-2.0)	--	ND	1.2 (0.39-2.5)	--	2.3 (ND-6.4)	--	--	--	--	--	--	--	--	--	--	4.5 (1.2-18.0)	
Stockholm, Sweden																					Meironyté-Guvenius et al. (2003); blood plasma samples were collected in 2000-2001 from 15 mothers ranging in age from 28-38 years old.
Median	<0.01	0.07	--	0.83	0.02	--	0.19	0.17	0.56	0.04	0.06	--	--	--	--	--	--	--	--	2.07	
Range	<0.01-0.03	<0.01-0.2	--	0.3-5.1	<0.01-0.14	--	<0.01-1.43	<0.01-0.52	0.27-1.03	<0.01-0.16	0.01-0.44	--	--	--	--	--	--	--	--	0.71-8.39	
Sweden																					Bergman et al. (1999); for BDE-47, BDE-153, and BDE-183, the limit quantification was defined as twice the blank sample amount; for BDE-209, the limit of quantification was defined as <0.7 pmol/g lipid and the limit of detection was <0.3 pmol/g lipid and also defined as the signal to noise ratio of
No fish Intake - 1991	--	--	--	0.407	--	--	--	--	NA	--	NA	--	NA	--	--	--	--	--	--	--	
High fish Intake - 1991	--	--	--	2.155	--	--	--	--	NA	--	NA	--	NA	--	--	--	--	--	--	--	
Cleaners - 1997	--	--	--	1.567	--	--	--	--	0.578	--	0.117	--	<0.678	--	--	--	--	--	--	--	
Clerks - 1997	--	--	--	1.469	--	--	--	--	0.844	--	0.175	--	<0.678	--	--	--	--	--	--	--	
Dismantling Electronics - 1997	--	--	--	2.89	--	--	--	--	4.547	--	8.025	--	4.846	--	--	--	--	--	--	--	
Sweden - Median																					Jakobsson et al. (2002); Subjects included 19 hospital computer technicians, 20 hospital cleaners and 20 computer clerks
Computer Technicians	--	--	--	1.3	--	--	--	--	2.7	0.6	0.95	--	1.6[d]	--	--	--	--	--	--	--	
Hospital Cleaners	--	--	--	1.6	--	--	--	--	0.58	0.38	0.12	--	<0.7[a]	--	--	--	--	--	--	--	
Computer Clerks	--	--	--	1.5	--	--	--	--	0.84	0.51	0.18	--	<0.7[a]	--	--	--	--	--	--	--	
Range	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	
Computer Technicians	--	--	--	<1[a]-14	--	--	--	--	<1[a]-5.8	0.23-1.2	0.18-4.7	--	<[c]-6.9	--	--	--	--	--	--	--	
Hospital Cleaners	--	--	--	<0.5-17	--	--	--	--	0.42-4.9	0.16-0.91	0.018-0.29	--	<0.3[c]-3.8	--	--	--	--	--	--	--	
Computer Clerks	--	--	--	<0.5[a]-4.9	--	--	--	--	0.5-3.3	0.28-0.97	<0.02[a]-1.0	--	<0.3[c]-7.8	--	--	--	--	--	--	--	
Norway																					Thomsen et al. (2003); Between 1975 to 2003, serum had been sampled from patients at five different county hospitals, regardless of reason for hospitalization or disease.
1977	--	ND	--	0.25	--	--	0.087	ND	0.1	ND	--	--	--	--	--	--	--	--	--	--	
1981	--	0.096	--	0.32	--	--	0.13	0.079	0.18	0.22	--	--	--	--	--	--	--	--	--	--	
1986	--	ND	--	0.41	--	--	0.13	0.12	0.14	0.26	--	--	--	--	--	--	--	--	--	--	
1990	--	0.066	--	0.89	--	--	0.24	0.13	0.27	0.23	--	--	--	--	--	--	--	--	--	--	
1995	--	0.14	--	1.4	--	--	0.33	0.32	0.52	0.5	--	--	--	--	--	--	--	--	--	--	
1999	--	0.24	--	1.5	--	--	0.31	0.35	0.59	0.35	--	--	--	--	--	--	--	--	--	--	
0-4 years old	--	0.26	--	6.2	--	--	1.6	1.7	1.5	0.45	--	--	--	--	--	--	--	--	--	--	
4-14 years old	--	0.2	--	2	--	--	0.37	0.66	0.86	0.39	--	--	--	--	--	--	--	--	--	--	
15-24 years old, female	--	0.088	--	2.5	--	--	0.71	0.49	0.56	0.27	--	--	--	--	--	--	--	--	--	--	
15-24 years old, male	--	0.094	--	2.3	--	--	0.68	0.61	0.66	0.19	--	--	--	--	--	--	--	--	--	--	
25-59 years old, female	--	0.093	--	1.3	--	--	0.32	0.28	0.34	0.23	--	--	--	--	--	--	--	--	--	--	
25-59 years old, male	--	0.093	--	2.3	--	--	0.4	0.52	0.71	0.24	--	--	--	--	--	--	--	--	--	--	
> 60 years old, female	--	0.36	--	1.2	--	--	0.25	0.37	0.36	0.26	--	--	--	--	--	--	--	--	--	--	
> 60 years old, male	--	0.096	--	3.4	--	--	0.36	0.45	0.59	0.38	--	--	--	--	--	--	--	--	--	--	
Norway - Mean																					Hagmar and Bergman (2001); subjecst tested included laboratory personnel, circuit board producers, and electronic dismantlers are the only worker categories listed for BDE-99.
Laboratory Personnel	--	--	--	1.5	--	--	0.4	--	0.54	--	--	--	--	--	--	--	--	--	--	--	
Circuit Board Producers	--	--	--	1.6	--	--	0.32	--	0.95	--	--	--	--	--	--	--	--	--	--	--	
Electronics dismantlers	--	--	--	4	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	
(Sweden)	--	--	--	2.9	--	--	0.97	--	1.7	--	--	--	--	--	--	--	--	--	--	--	
(Sweden)	--	--	--	--	--	--	--	--	4.5	--	0.33	--	--	--	--	--	--	--	--	--	
(Sweden)	--	--	--	--	--	--	--	--	--	--	3.2	--	--	--	--	--	--	--	--	--	
(Sweden)	--	--	--	--	--	--	--	--	--	--	7.8	--	4.8	--	--	--	--	--	--	--	
- Range																					
Laboratory Personnel	--	--	--	1.0-3.0	--	--	0.17-0.73	--	0.43-0.63	--	----	--	----	--	--	--	--	--	--	--	
Circuit Board Producers	--	--	--	0.4-3.4	--	--	ND-0.77	--	0.5-1.8	--	----	--	----	--	--	--	--	--	--	--	
Electronics dismantlers	--	--	--	0.9-15	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	
(Sweden)	--	--	--	<0.5-23	--	--	0.18-3.6	--	1.2-2.3	--	--	--	--	--	--	--	--	--	--	--	
(Sweden)	--	--	--	--	--	--	--	--	2.1-12	--	0.09-1.1	--	--	--	--	--	--	--	--	--	
(Sweden)	--	--	--	--	--	--	--	--	--	--	2.5-12	--	--	--	--	--	--	--	--	--	
(Sweden)	--	--	--	--	--	--	--	--	--	--	2.3-20	--	<0.3-9.5	--	--	--	--	--	--	--	
Norway - Mean																					Thomsen et al. (2001b); total PBDEs consist of BDE-28,47,99,100,153,154,183,TriBP, and TBBPA.
electronics dismantlers	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	8.8	
Circuit board producers	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	3.9	
Laboratory Personnel	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	3	
- Range																					
electronics dismantlers	--	--	--	0.43-14.6	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	----	
Circuit board producers	--	--	--	0.43-14.6	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	----	
Laboratory Personnel	--	--	--	0.43-14.6	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	----	

ND = Not Detected
NA = Not Analyzed
< = did not meet the limit of quantification

Table 3-16. Levels of BDEs in human adipose tissue in the U.S. general population and in other countries (ng/g lipid weight)

Location and Population	BDE-28	BDE-47	BDE-85	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	Total PBDEs	Reference and Comments
San Francisco, USA										Petreas et al. (2003); samples were collected from 1996-1998 from 32 women between the ages of 25 and 65 years old from a case-control study on breast cancer and organochlorine
Mean	--	28.9 ± 39.8	--	--	--	--	--	--	--	
Median (Range)	--	16.5 (5.2-196)	--	--	--	--	--	--	--	
San Francisco, USA										She et al. (2002); Breast adipose tissue was sampled from 23 women ages 28-62 (mean of 47); percent fat in sample ranged from 9.9-93.6 with a mean of 72.2
Range	--	7.01-196.0	--	2.17-72.2	0.77-60.6	1.52-124.0	2.86-70.5	--	17.2-462.0	
Mean	--	33.3	--	10.7	9.1	16.2	16.5	--	85.7	
Spain										Meneses et al. (1999); tissues from 13 people were sampled (3 women and 10 men) ages 28 to 83 years (mean age of 57)
Mean (Range)	--	1.36 (0.2-5.8)	--	0.42 (<0.07-2.1)	--	1.83 (0.67-4.2	--	--	--	
Sweden	--	8.8	1.8	1.1	--	1.7	--	--	--	Haglund et al. (1997); based on 90% lipids
Japan										Choi et al. (2003); tissues from 10 women in their 40's and 50's, living in the Tokyo area were sampled Lipid contents of the samples ranged from 72% to 95 %. According to Choi et al. the sample ratio for the 1970 samples ranged from 0.6-1.5 while the samples from 2000 varied from 2.0-9.2.
Median	0.0023	0.017	--	0.0039	0.0021	<0.0063	<0.0063	<0.0063	0.0292	
Range	<0.001-0.0076	0.0044-0.0604	--	<0.0025-0.0139	<0.0025-0.006	<0.0063	<0.0063	<0.0063	0.0068-0.078	
Median	0.076	0.459	--	0.118	0.25	0.382	0.06	0.047	1.288	
Range	0.047-0.487	0.109-0.979	--	0.042-0.362	0.041-0.527)	0.122-0.631	0.014-0.104	0.020-0.177	0.466-2.753	

< = did not meet the limit of quantification

3.10 Conceptual Exposure Model

This section is the fourth component of the exposure assessment. It includes a conceptual exposure model describing the possible routes of exposure to children and prospective parents. The conceptual exposure model for the commercial octaBDE product illustrates three different scenarios: exposures in the workplace, exposures in the indoor home/school/office environment, and exposures associated with the ambient environment (e.g., via the diet, both direct and indirect contact with soil, and particulates in air). For direct and indirect exposure routes illustrated in the conceptual model, where exposures are plausible and quantifiable, screening-level exposure models developed in Microsoft® Excel are presented for children of different ages and adult men and women.

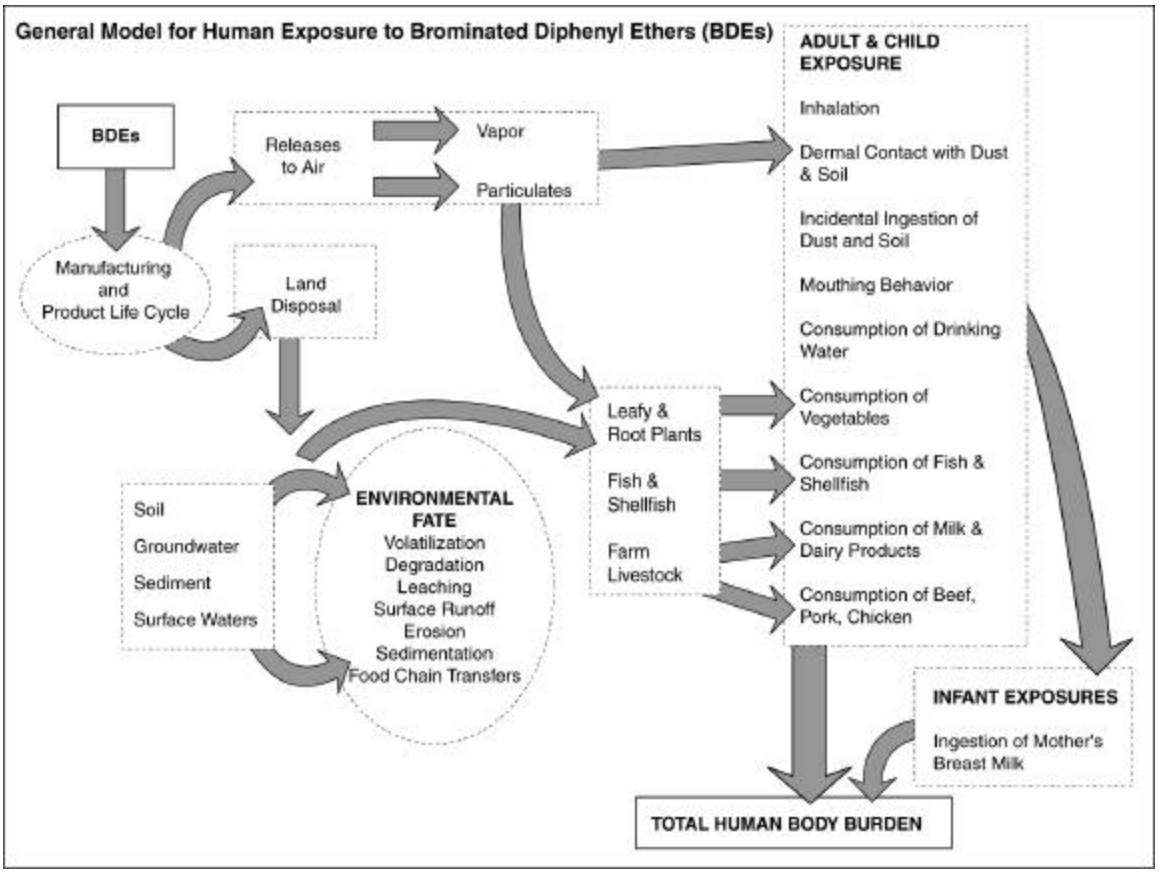
Consistent with the Tier 1 screening approach, exposure models rely on exposure point concentrations for different environmental compartments (e.g., air, soil, food, and in the workplace) representing the 95th upper confidence limit on the mean for robust data sets or the high end of the range of the available data, if the available data are limited. Exposure factors, based primarily on USEPA's *Exposure Factors Handbook* (USEPA, 1997) and *Child-Specific Exposure Factors Handbook* (USEPA, 2000a) are identified for each of the exposure scenarios and associated exposure pathways. All assumptions and parameter values used in the screening-level exposure calculations are specified and referenced to the relevant supporting technical information.

The conceptual exposure model describing how potentially exposed populations might be exposed to the commercial octaBDE product is presented in Figure 3-2. Figure 3-2 provides a general overview of how BDEs associated with the commercial octaBDE product might migrate through the environment, culminating in human exposure. In Figure 3-3, the conceptual exposure model is further refined to indicate the specific receptors and exposure pathways that are considered in the Tier 1 assessment.

Potentially Exposed Populations

The populations that are potentially exposed to the commercial octaBDE product are as follows: Primary Product Manufacturers, Plastic Pellet Manufacturers, End Product Manufacturers (i.e., casing and equipment manufacturers), Equipment Distributors, Equipment Recyclers, and adults and children in the indoor home/school/office environment (which includes day care centers), as well as exposures associated with the ambient environment. Seven different workplace job functions in primary production and chain-of-commerce manufacturing activities were considered in the Tier 1 assessment, as shown in Table 3-17. The entire population of workers engaged in primary manufacturing of the commercial octaBDE product is fewer than 100 people employed by GLCC at one chemical plant located in Arkansas and are typically male workers ranging in age

Figure 3-2. General conceptual exposure model depicting how BDEs associated with the commercial octaBDE product might migrate through the environment, culminating in human exposure



from 20-45 years old. Female workers are included in this Tier 1 assessment. The population of workers engaged in secondary, or chain-of-commerce, activities involving the use of the commercial octaBDE product as an additive in the manufacture of plastic casings is large, and cannot be estimated with any degree of accuracy. Similarly, the population of adult men and women and children potentially exposed to ambient levels of BDEs in the environment and through contact with consumer products containing the commercial octaBDE product (primarily electronic equipment) in the indoor home/school/office environment also is large, and cannot be estimated with any degree of accuracy. Children exposures are evaluated quantitatively, for seven different age bins: <1 year, 1-2 years, 3-5 years, 6-8 years, 9-11 years, 12-14 years, and 15-18 years. Girls and boys are evaluated separately in the 9-11, 12-14, 15-18 year age bins.

Figure 3-3. Specific conceptual exposure model indicating the specific receptors and exposure pathways considered in the Tier 1 assessment of the commercial octaBDE product

Sources	Primary Release Mechanism	Secondary Release Mechanism	Exposure Media	Exposure Route	RECEPTORS: Primary & Chain-of-Commerce Manufacturing Exposure: Primary Exposure (Grinder & Packager)	Plastic/Pellet Manufacturer	End-Product Manufacturer (Casing Manufacturer & Equipment Assembler)	Distributor (Covered & Uncovered)	Recycler	Worker's Child	Exposures in the Home / School / Office: Child	Adult	Exposures to Ambient Environmental Levels: Child	Adult	Adult Angler (Marine & Freshwater)
Primary Source: Commercial octaBDE Product Mixture	Volatilization			Inhalation of Vapors	X	X	X	X	X	--	--	--	--	--	--
	Grinding & Cleaning Activities			Dermal Contact	X	X	X	X	X	--	--	--	--	--	--
				Incidental Ingestion	X	X	X	X	X		--	--	--	--	--
Secondary Source: ABS Plastics	Drumming & Packaging Activities	Adherence to Clothing	Dirty Laundry	Dermal Contact	X	X	X	X	X	X	--	--	--	--	--
				Incidental Ingestion	X	X	X	X	X	X	--	--	--	--	--
	Pellet Production														
Tertiary Source: End Product (Computer Casings, etc.)	End-Product Manufacture	Adherence to Shoes	Dirty Floors	Dermal Contact	X	X	X	X	X	X	--	--	--	--	--
				Incidental Ingestion	X	X	X	X	X	X	--	--	--	--	--
	Distribution (Plastics & End-Product)														
	Recycling														
recycle															
Purchase & Use of End-Product		Off-Gasing During Product Lifetime	Indoor Air	Inhalation of Particulates	--	--	--	--	--	--	X	X	--	--	--
				Ingestion of Swallowed Particulates	--	--	--	--	--	--	X	X	--	--	--
			Indoor Dust	Dermal Contact	--	--	--	--	--	--	X	X	--	--	--
				Incidental Ingestion	--	--	--	--	--	--	X	X	--	--	--
		Mouthing Behavior	Saliva	Ingestion	--	--	--	--	--	--	X	--	--	--	--
dispose															
Effluent Discharge		Volatilization	Ambient Air	Inhalation of Particulates	--	--	--	--	--	--	--	--	X	X	--
				Ingestion of Swallowed Particulates	--	--	--	--	--	--	--	--	X	X	--
Waste Incineration		Leaching													
		Surface Runoff	Soil	Dermal Contact	--	--	--	--	--	--	--	--	X	X	--
Landfill Disposal															
		Erosion		Incidental Ingestion	--	--	--	--	--	--	--	--	X	X	--
		Sedimentary	Fish	Consumption	--	--	--	--	--	--	--	--	X	X	--
Sewage Sludge		Food Chain Transfer		Additional Consumption by Anglers	--	--	--	--	--	--	--	--	--	--	X
			Other Food (Meat, Dairy, Fats and Oils, Eggs & Vegetables	Consumption	--	--	--	--	--	--	--	--	X	X	--
		★	Breast Milk	Consumption	--	--	--	--	--	--	--	--	X	--	--
			Drinking Water	Consumption	--	--	--	--	--	--	--	--	--	--	--

★ Note: OctaBDE is not transferred in water

Table 3-17. Human populations evaluated in the commercial octaBDE product Tier 1 exposure assessment

Receptor	Description
Occupational Exposure Scenarios	
Primary Production Grinder (Male/Female)	Breaks the solid octaBDE product out of the pans and grinds it in preparation for packaging.
Primary Production Packager (Male/Female)	Packages the ground octaBDE product into bags for distribution.
Plastic Mixer and Pellet Maker (Male/Female)	Mixes octaBDE product with other components to produce pellets.
Injection Moulder (Male/Female)	Uses pellets to mould and produce plastic parts
Equipment Manufacturer/Assembler (Male/Female)	Assembles product based on already produced plastic parts.
Distributor Uncovered (Male/Female)	Handles and trans ports the uncovered plastic casings.
Distributor Covered (Male/Female)	Handles and transports boxed plastic products.
Home, School, and Office Exposure Scenarios	
<1 year Child	Exposed to consumer electronic goods impregnated with octaBDE.
1-2 yrs Child	
3-5 yrs Child	
6-8 yrs Child	
9-11 yrs Male/Female	
12-14 yrs Male/Female	
15-18 yrs Male/Female	
Adult Male (Prospective Parent)	
Adult Female (Prospective Parent)	
Environmental Exposure Scenarios	
<1 year Child	Exposed to octaBDE residues that have been measured in different environmental compartments (e.g., air, soil, food products, and fish). Degradation products are not considered. Exposure to water (drinking, surface, and ground water) is not considered due to the hydrophobic nature of the compounds of interest.
1-2 yrs Child	
3-5 yrs Child	
6-8 yrs Child	
9-11 yrs Male/Female	
12-14 yrs Male /Female	
15-18 yrs Male/Female	
Adult Male (Prospective Parent)	
Adult Female (Prospective Parent)	

Scenario #1a - Exposures to Commercial octaBDE Product Manufacturers

The potential exposure routes for workers engaged in primary manufacturing activities at a single U.S. facility located in Arkansas where the commercial octaBDE product is manufactured includes the following:

Primary Exposure Pathways

- Inhalation of vapors originating from primary product in the workplace;
- Dermal contact with primary product in the workplace;
- Incidental ingestion via hand-to-mouth contact in the workplace;

Secondary Exposure Pathways

- Dermal contact with primary product on dirty laundry in the home;
- Incidental ingestion via hand-to-mouth contact after handling dirty laundry in the home;
- Dermal contact with primary product tracked onto floors in the home; and,
- Incidental ingestion via hand-to-mouth contact after touching dirty floors in the home.

In addition to direct skin contact, four indirect exposure pathways involving dirty laundry and indoor floors in a home also were considered. For contact with dirty laundry and shoes, clothing (i.e., socks, shirts, and pants) and shoes were assumed to contact the commercial octaBDE product either during the manufacturing process, during the transfer of the product from the primary reaction vessels to storage containers, and/or during cleaning of residues on product reaction vessels and other equipment. When clothing was laundered in the home, only the bare hands were assumed to contact dirty clothing. Dermal contact and incidental ingestion with dirty floors theoretically occurs when workers redistribute the chemicals of concern adhered to shoes across the entry level of the home, with subsequent exposure from contacting the dirty floor. Workers' children are also present in the home and were evaluated for these last two pathways.

Scenario #1b – Exposures to ABS Plastic and Electronic Equipment Manufacturing Workers

The major sources of worker exposures during the manufacture of ABS plastic casings and electronic equipment using ABS plastic casings are likely to be associated with:

- Handling of the commercial octaBDE product prior to mixing with other ingredients;
- Volatilization of BDEs during the mixing and molding processes; and,
- Handling of residues during the cleaning of moulding equipment.

According to ECB (2002), the main source of exposure to the commercial octaBDE product is associated with the handling the small fraction of the manufactured product powder that has a particle size >40 µm. The amount potentially released to the air is estimated to be around 0.21% and is based on three assumptions. First, it is assumed that the manufactured product is handled in sacks and may endure wear and tear. The second assumption considers the attractive forces between the individual particles, which reduces the opportunity for small particles to become airborne. And the third is from dust generation.

The commercial octaBDE product is typically moulded into solid ABS plastic for electrical equipment (e.g. computer casings) at a plastic manufacturing facility. Some portion of the commercial octaBDE product may be lost during the moulding process and contained within the scrap ABS. ABS scraps can be recycled, although not likely due to the depreciation of product value, or sent to disposal in a landfill or incinerator. Releases from landfills and incinerators are

projected by the ECB (2002) to be near zero. Handling this material, however, may represent a possible route of exposure to the commercial octaBDE product.

The potential exposure routes for workers engaged in the manufacturing of ABS plastic casings and electronic equipment in the U.S. includes the following:

Primary Exposure Pathways

- Inhalation of vapors originating from the manufacture of plastic casings;
- Dermal contact with plastic casings;
- Incidental ingestion via hand-to-mouth contact;

Secondary Exposure Pathways

- Dermal contact with dirty laundry in the home;
- Incidental ingestion via hand-to-mouth contact after handling dirty laundry in the home;
- Dermal contact with material tracked onto floors in the home; and,
- Incidental ingestion via hand-to-mouth contact after touching dirty floors in the home.

In addition to direct skin contact, four indirect exposure pathways involving dirty laundry and indoor floors in a home also were considered. For contact with dirty laundry and shoes, clothing (i.e., socks, shirts, and pants) and shoes were assumed to contact ABS plastics and acquire 100% of the concentration of the commercial octaBDE product. When clothing was laundered in the home, only the bare hands were assumed to contact dirty clothing. Dermal contact and incidental ingestion with dirty floors theoretically occurs when workers redistribute the chemicals of concern adhered to shoes across the entry level of the home, with subsequent exposure from contacting the dirty floor. Workers' children are also present in the home and were evaluated for these last two pathways.

Scenario #2 – Exposures to Adults and Children in the Indoor Home/School/Office Environment

Three different locations were evaluated in this scenario: the indoor home, school and office environments. All three indoor environments typically contain different consumer electronic products. The potential exposure routes for adults and children in the indoor home/school/office environment in the United States includes the following:

Primary Exposure Pathways

- Inhalation of respirable particulates originating from ABS plastics;
- Ingestion of particulates inhaled and swallowed;
- Dermal contact with indoor dust that contains plastic particles;
- Incidental ingestion by young children mouthing ABS plastic surfaces treated with the commercial octaBDE product; and,

- Incidental ingestion of indoor dust via hand-to-mouth contact.

It is highly unlikely that the commercial octaBDE product will either volatilize, leach during disposal, or be released via binding to particulates during the lifetime of the consumer product in the indoor home/school/office environment. The commercial octaBDE product has a very low vapor pressure; losses from plastics due to volatilization are expected to be minimal, and may not be measurable in the indoor home/school/office environment.

It is possible that the most likely source of exposure to the commercial octaBDE product in ABS plastic is via direct or indirect contact with particles released into indoor air at the end of a product life cycle or by accidental destruction of the consumer product indoors. The release of BDEs from ABS plastics impregnated with the commercial octaBDE product was not evaluated in this scenario. For young children (less than 5 years old), mouthing activity on ABS plastic may be a source of exposure to the commercial octaBDE product.

Scenario #3 – Ambient Environmental Exposures to Adults and Children

In addition to the indoor home/school/office environment, adults and children may be exposed to the commercial octaBDE product via releases to the ambient environment. Potential exposures are primarily through contact with ambient air, soil, and the consumption of food products, including human breast milk for infants. In addition, fish consumption through recreational fishing is another source of ambient environmental exposure. It is not well understood at this time to what extent (or how) the commercial octaBDE product contributes to levels reported in various environmental compartments.

The potential exposure routes for adults and children associated with ambient levels in the United States environment includes the following:

Primary Exposure Pathways

- Inhalation of respirable particulates in ambient air;
- Ingestion of particulates inhaled and swallowed;
- Dermal contact with outdoor soil;
- Incidental ingestion of outdoor soil via hand-to-mouth contact;
- Ingestion of meat, dairy, and vegetable food products,
- Ingestion of recreationally caught fish; and,
- Ingestion of human breast milk by infants.

Since the commercial octaBDE product is used mainly in ABS plastics, the potential for release of particulate waste from weathering, wear, etc., during the service life of consumer electronic products treated with the commercial octaBDE product is low. It is possible, although data are lacking, that releases to the environment could occur during or after disposal at the end of the

product's life cycle (ECB, 2002). The actual volume of plastics treated with the commercial octaBDE product and disposed in landfills or incinerated in the United States is not known. In addition, releases to the environment are possible during accidental fires in the home or workplace, but data are lacking. As a result, only the primary exposure pathways indicated above are included in the Tier 1 screening level exposure assessment.

Tier 1 Human Exposure Concentration Values

A summary of the environmental and human exposure data used to support the Tier 1 exposure assessment is presented in Table 3-18. Rather than rely on environmental fate models to predict exposure point concentrations, the values used to calculate exposure were based on representative (yet protective) levels derived from published environmental studies conducted in the U.S. The values selected for use in the exposure assessment calculations were chosen to evaluate potential workplace, indoor home/school/office, and ambient environmental exposures to the commercial octaBDE product. Where data were reported in the scientific literature for individual BDE congeners or groups, the congeners associated with the commercial octaBDE product were summed and used in this Tier 1 assessment.

To evaluate the potential for exposure to the commercial octaBDE product in the workplace, indoor air particulate levels in primary manufacturing, chain-of-commerce manufacturing of ABS plastic casings, and plastic recycling and shredding facilities were conservatively based on air particulate data collected from indoor air measurements conducted by GLCC and other researchers.

To evaluate exposures to the commercial octaBDE product in the indoor home/school/office environment, levels in indoor dust were based on the 95th percentile upper confidence limit on the mean of the data set reported by Knoth et al. (2002). Indoor air levels were based on the data from Sjödin et al. (2001a). The potential for exposure to the commercial octaBDE product in ABS plastics was based on the average content of the commercial octaBDE product reported in ABS plastic.

Table 3-18. Environmental and human exposure values used in the Tier 1 exposure assessment

Exposure Scenario	Exposure Media	Concentration	Units of Concentration	Reference and Comments
Workplace	Indoor Air Particulate (Primary Manufacturing)	4.6	mg/m^3	GLCC (2002); for primary product workers, the high value of three samples in the packaging area of a primary product processing plant; data are for Great Lakes DE-79™, an octaBDE mixture. The exposure model assumes no respiratory protection in the workplace.[5]
	Indoor Air Particulate (Chain of Commerce Manufacturing)	1.67	mg/m^3	Breysse and Kocergis (2000); for other workers, the average dust value from nine facilities that use decaBDE mixture (no data available for octaBDE mixture), reported using personal air monitoring was used.
	Indoor Air Particulate (Recyclers/Shredders)	0.000312	mg/m^3	Sjödin (2001a); for recyclers/shredders, represents the sum of BDE-47,85,99,100,153,154,183 (excludes decaBDE)
Home / School / Office	Indoor Air	1.3	ng/m^3	Sjödin et al., 2001a
	Indoor Dust	410	ng/g	Knoth et al., 2002; represents the 95th upper confidence limit of the mean of the data set.
	ABS Plastic Surfaces	0.5	µg/L	ECB, 2002; based on the water solubility for the commercial octaBDE product
Ambient Environment	Ambient Air	52	pg/m3	Strandberg et al., 2001
	Soil	76	µg/kg	Hale et al., 2002
	Leafy and Root Vegetables	0.134	ng/g ww	Ohta et al., 2002
	Meat	0.0458	ng/g ww	Darnerud et al., 2000 (unpublished)
	Dairy	0.0182	ng/g ww	
	Other Fats and Oils	0.158	ng/g ww	
	Eggs	0.0425	ng/g ww	
	Fish Fillet	71.67	ng/g ww	Johnson and Olson, 2001; Dodder et al., 2002; and Loganathan et al, 1995 as cited in Manchester-Neesvig et al., 2001; represents the upper 95th percentile of 73 fish fillet samples.
	Breast Milk	42.8	ng/g lipid weight	Ryan et al., 2002

[5] Respiratory and dermal protection in the primary production workplace is required when workplace air levels are for jobs potentially exposed to levels above the GLCC WEL of 0.14 mg/m^3 and during tasks involving handling of the product.

Environmental exposures to the commercial octaBDE product through direct and indirect contact with soil was based on levels reported by Hale et al. (2002) in soil adjacent to a FPUF manufacturing plant since no reports were based on the commercial octaBDE product was identified in the literature. Exposures to the commercial octaBDE product in ambient air were based on levels reported in Chicago ambient air by Stranberg et al. (2001). In the absence of U.S. data, exposures through the food pathway were based on the results of market basket surveys reported by Darnerud et al. (2000; unpublished) for meat, dairy, eggs, and other fats and oils, and by Ohta et al. (2002) for vegetables. The 95th percent upper confidence limit on the mean of the range of data describing total BDE levels in edible fish tissue (see Table 3-10) was used to evaluate fish consumption by the general population and the additional consumption by recreational fishermen in the United States. Infant exposures to the commercial octaBDE product through consumption of human breast milk was based on the levels reported by Ryan et al. (2002). The data reported by Päpke et al. (2001) was not used because it is based on a single pooled sample of as many as 30 women and does not adequately represent the U.S. population.

3.11 Theoretical Exposure Pathways and Calculations

The Tier 1 exposure assessment presented in this section represents a screening-level analysis. The USEPA (2001) characterizes a Tier 1 exposure assessment as containing screening level information on exposure from manufacturing activities, downstream processing and use activities, and specific information on children's (and relevant adult) exposure scenarios. The Agency defines the typical screening level exposure assessment as one involving readily available measured data, existing release and exposure estimates and other exposure-related information. Where actual measures of exposure are not available, the use of models is considered appropriate. USEPA (2001) has specified that the populations of concern to the VCCEPP are children and, in certain situations, prospective parents. Exposures that can affect children are those that occur prior to conception (to either parent), during prenatal development, and post-natal to the age of sexual maturation, which USEPA (2001) indicates is completed around 18-21 years of age. Although adult exposures are not intended to be a major focus, the Agency believes that prospective parent exposures are relevant to the evaluation of risks due to fertility and reproductive effects, as well as developmental effects from *in utero* exposures.

Exposure modeling was performed in accordance with USEPA exposure assessment guidelines (USEPA, 1992). In addition, other available exposure assessment procedures and guidance such as the Agency's draft exposure factors handbook for children (USEPA, 2000a), which consolidates all child exposure factors and related data contained in the National Center for Environmental Assessment (NCEA) Exposure Factors Handbook (USEPA, 1997), also was used to develop exposure models for the different exposure scenarios. The Agency's recent guidance on risk assessment of chemical mixtures (USEPA, 2000c) also was consulted during the development of this Tier 1 assessment.

General Approach to Calculating Theoretical Exposures

Inhalation, incidental ingestion, dermal contact, and food consumption were evaluated in one or more of the three exposure scenarios (i.e., workplace, indoor home/school/office environment, and the ambient environment, including food, breast milk, and fish consumption) included in the Tier 1 exposure assessment of the commercial octaBDE product. Both incidental ingestion and dermal contact were evaluated in all three scenarios. Airborne vapor inhalation was limited to workers, while inhalation of airborne particulates (and swallowing the exhaled portion) was evaluated for indoor and ambient environmental exposures to adults and children. Food consumption was also evaluated. Exposures through the consumption of surface water or groundwater were not included in the Tier 1 assessment (see section 3.5).

In this Tier 1 assessment, exposures to the commercial octaBDE product by workers and by adults and children in the indoor home/school/office environment and through ambient environmental exposures were evaluated by modeling the physical characteristics and behaviors of the reasonably highest exposed individual, similar to the description of the Reasonable Maximum Exposure (RME) profile described in USEPA (1989) risk assessment guidance. The RME was defined as an individual who is of typical behavior and physical characteristics and receives the high end of the reasonable range of exposure (in this assessment, either the 95th percentile upper confidence limit of the mean exposure or the 95th percentile of the range of available data). The theoretical chronic daily intakes (CDIs) for non-cancer effects were calculated for dermal, oral and inhalation uptake. The relevant CDIs were summed to provide the cumulative, or total, daily intake to each receptor group evaluated in the three exposure scenarios. Cancer risks were not evaluated in this Tier 1 assessment.

The exposure assumptions and values used to calculate theoretical exposures are presented in Table 3-19 for workers and workers' children, Table 3-20 for adults and children in the indoor home/school/office environment, and Table 3-21 for adults and children exposed to ambient environmental levels and through consumption of foods, fish, and breast milk (infants only). Oral, dermal, and inhalation bioavailability values are summarized in Table 2-12. The exposure models used to calculate exposure to the commercial octaBDE product are presented in Appendix VI. A summary of the exposure variables used in the different exposure model calculations are presented in Table 3-22.

Table 3-19. Exposure assumptions and parameters used to calculate theoretical exposures to the commercial octaBDE product by primary and chain-of-commerce manufacturing workers and the children of workers

Exposure Parameter	Variable	Units	Receptor Scenarios (All Are Prospective Parents)													
			Primary Production Grinder, Male	Primary Production Grinder, Female	Primary Production Packager, Male	Primary Production Packager, Female	Plastic/Pellet Manufacture Mixer, Male	Plastic/Pellet Manufacture Mixer, Female	Casing Manufacture, Male	Casing Manufacture, Female	Equipment Assembler, Male	Equipment Assembler, Female	Distributor Uncovered, Male	Distributor Uncovered, Female	Recycler, Male	Recycler, Female
Physiological and General Assumptions																
Body Weight	BW	kg	78.1	65.4	78.1	65.4	78.1	65.4	78.1	65.4	78.1	65.4	78.1	65.4	78.1	65.4
Inhalation Rate	InR	m^3/day	12.8	12.8	12.8	12.8	12.8	12.8	12.8	12.8	12.8	12.8	12.8	12.8	12.8	12.8
Exposed Skin Surface Area, Total Body	SAt	cm^2	19,400	16,900	19,400	16,900	19,400	16,900	19,400	16,900	19,400	16,900	19,400	16,900	19,400	16,900
Exposed Skin Surface Area, Palms of Hands	SAh	unitless	0.026	0.024	0.026	0.024	0.026	0.024	0.026	0.024	0.026	0.024	0.026	0.024	0.026	0.024
Fraction of Skin Surface Area Exposed while Doing Laundry	FSAl	unitless	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Fraction of Skin Surface Area Exposed to Floor	FSAf	unitless	0.391	0.305	0.391	0.305	0.391	0.305	0.391	0.305	0.391	0.305	0.391	0.305	0.391	0.305
Fraction of Skin Surface Area that Contacts Mouth	FSAftip	unitless	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
Fraction of Laundry that is Contaminated	FCLdy	unitless	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25
Floor Surface Area	FSA	cm^2	756644	756644	756644	756644	756644	756644	756644	756644	756644	756644	756644	756644	756644	756644
Fraction of Skin Surface Area that is Shoes	SAs	cm^2	613	613	613	613	613	613	613	613	613	613	613	613	613	613
Exposure Frequency Assumptions																
Fraction of Work Day Spent Around Commercial Product	FWD	unitless	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33	0.33
Exposure Frequency, Doing Laundry	EFL	hr/day	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22
Exposure Frequency, Contact with Floors	EFF	hr/day	3	3	3	3	3	3	3	3	3	3	3	3	3	3
Environmental Assumptions																
Absorption Factor, Oral Route	AFo	unitless	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Absorption Factor, Inhalation Route	AFi	unitless	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75
Absorption Factor, Dermal Route	AFd	unitless	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313
Chemical Potency Assumptions																
Workplace Exposure Limit, inhalation	WEL	mg/m3	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14	0.14
Thyroid Effects Benchmark, oral and inhalation	TEB	mg/kg-day	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09
Developmental Effects Benchmark, oral and Inhalation	DEB	mg/kg-day	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09
Reference Dose, oral	RfDo	mg/kg-day	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003
Reference Dose, inhalation	RfDi	mg/kg-day	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003
Chemical Assumptions																
Total Particulate Concentration	AirTP	mg/m3	4.6	4.6	4.6	4.6	1.67	1.67	1.67	1.67	1.67	1.67	1.67	1.67	3.12E-04	3.12E-04
Fraction of Total Particulate that is Respirable	Fresp	unitless	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Fraction of Particulate that is Commercial Product	Fpp	unitless	0.25	0.25	0.25	0.25	0.25	0.25	0.18	0.18	0.18	0.18	0.18	0.18	0.25	0.25
Fraction of Handled Item that is Commercial Product	Fip	unitless	0.25	0.25	0.25	0.25	0.25	0.25	0.18	0.18	0.18	0.18	0.18	0.18	0.18	0.18
Adherence Rate of Handled Item to Skin	ARskin	mg/cm2-day	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Adherence Rate of Item to Shoes	ARshoe	mg/cm2-day	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1

Table 3-19. Exposure assumptions and parameters used to calculate theoretical exposures to the commercial octaBDE product by primary and chain-of-commerce manufacturing workers and the children of workers

Exposure Parameter	Worker's Children Receptors										Reference and Notes
	<1 yr Child	1-2 yr Child	3-5 yr Child	6-8 yr Child	9-11 yr Male Child	9-11 yr Female Child	12-14 yr Male Child	12-14 yr Female Child	15-18 yr Male Child	15-18 yr Female Child	
Physiological and General Assumptions											
Body Weight	9.1	11.3	16.35	23.75	33.75	34	47.05	48.65	64.93	57.6	USEPA (1997, 2000a) EFH, page 7-4, table 7-3 for children; table 7-2 for adults; mean values
Inhalation Rate	--	--	--	--	--	--	--	--	--	--	USEPA (1997, 2000a) EFH, page 5-24, table 5-23; value of 1.6 m3/hr for adult moderate work activity multiplied by 8-hr workday
Exposed Skin Surface Area, Total Body	5,910	5,910	7,200	9,250	11,600	11,600	14,900	14,800	17,500	16,000	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value represents total body skin surface area
Exposed Skin Surface Area, Palms of Hands	0.027	0.028	0.029	0.025	0.027	0.027	0.026	0.026	0.028	0.028	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value for the palms of both hands represents 1/2 the estimate for the skin surface area of both hands
Fraction of Skin Surface Area Exposed while Doing Laundry	--	--	--	--	--	--	--	--	--	--	Assumes entire hand skin surface contacts dirty laundry
Fraction of Skin Surface Area Exposed to Floor	0.337	0.342	0.379	0.361	0.374	0.374	0.383	0.383	0.403	0.403	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value represents the estimate for the skin surface area of the hands, arms, legs, and feet
Fraction of Skin Surface Area that Contacts Mouth	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.002	0.002	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value for the fingertips of all ten digits of both hands represents 1/2 the estimate for the skin surface area of both hands and assumes 25% of each hand is fingertips that could be placed in the mouth
Fraction of Laundry that is Contaminated	--	--	--	--	--	--	--	--	--	--	Assumes that 1/4 of the laundry may contain the product on clothing used in the workplace
Floor Surface Area	--	--	--	--	--	--	--	--	--	--	USEPA (1997) EFH; table 17-31 central estimate; assume two-story home
Fraction of Skin Surface Area that is Shoes	--	--	--	--	--	--	--	--	--	--	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile; value for the soles of both feet represents 1/2 the estimate for the skin surface area of the feet
Exposure Frequency Assumptions											
Fraction of Work Day Spent Around Commercial Product	--	--	--	--	--	--	--	--	--	--	For primary production and mixing of product with FPUF ingredients, one-third of the day is assumed spent handling the primary product directly; for other work categories, the entire work day is assumed to involved handling materials containing the product
Exposure Frequency, Doing Laundry	--	--	--	--	--	--	--	--	--	--	USEPA (1997) EFH, page 15A-22, table 15A-5; mean value of 13.35 min/weekday Should this only apply to the dirtiest workers (primary and uncovered workers)?
Exposure Frequency, Contact with Floors	1	3	3	3	3	3	3	3	3	3	Assume workday evenings are spent on floor by adults and older children, youngest children are often in cribs or play pens, and pre-schoolers spend the most time on the floor
Environmental Assumptions											
Absorption Factor, Oral Route	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	ECB (2002) RA, page 132; because no value available, assumed 50%
Absorption Factor, Inhalation Route	--	--	--	--	--	--	--	--	--	--	ECB (1996); assumed to be below the generic value of 75% recommended for all chemicals in the absence of chemical-specific information
Absorption Factor, Dermal Route	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	Inveresk Research (2001); mean dermal delivery of applied dose of radio-labeled tetrabromodiphenyl ether, 12 samples of human skin
Chemical Potency Assumptions											
Workplace Exposure Limit, inhalation	--	--	--	--	--	--	--	--	--	--	GLCC (personal communication); airborne limit, intended for internal and customer use, octa product mixture
Thyroid Effects Benchmark, oral and inhalation	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	Derived from Zhou et al. (2001)
Developmental Effects Benchmark, oral and Inhalation	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	Derived from WIL Research (1986a)
Reference Dose, oral	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	USEPA (2003) IRIS; based on 1980 rat study that administered commercial product mixture (1.1% penta, 8.5% hexa, 45.1% hepta, 30.7% octa, 13.0% nona, 1.6% deca); critical effect: induction of hepatic enzymes, liver histopathology; uncertainty factor = 1000, modifying factor = none; low level of confidence in RfD
Reference Dose, inhalation	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	No data available in IRIS; used the oral RfD
Chemical Assumptions											
Total Particulate Concentration	--	--	--	--	--	--	--	--	--	--	GLCC (2002); For primary product workers, values represent the average value of three samples collected in the packaging area of a primary product processing plant, data are for DE-79, an octa mixture. Breysse (2000); For other workers values assume the average dust value from nine facilities that use deca mixture (no data available for octa mixture), personal sample used rather than area sample. Sjodin (2001), For recyclers/shredders, values represent the sum of BDE-47,85,99,100,153,154,183 (excludes deca).
Fraction of Total Particulate that is Respirable	--	--	--	--	--	--	--	--	--	--	Assumes that 10% of the particulate values cited by GLCC (personal communication), Breysse (2000), and Sjodin (2001a) is respirable and reaches ambient air in the workplace
Fraction of Particulate that is Commercial Product	--	--	--	--	--	--	--	--	--	--	ECB (2002) page 96, typical levels in plastics are 12-18%, used upper end of range; for recyclers, no adjustment necessary
Fraction of Handled Item that is Commercial Product	--	--	--	--	--	--	--	--	--	--	ECB (2002) page 96, typical levels in plastics are 12-18%, used upper end of range
Adherence Rate of Handled Item to Skin	--	--	--	--	--	--	--	--	--	--	ECB (2000) RA, page 94; lower end of range predicted by EASE program (0.1 to 1.0 mg/cm2-day)
Adherence Rate of Item to Shoes	--	--	--	--	--	--	--	--	--	--	Assumes that the bottom of shoes are equivalent to bare skin

Table 3-20. Exposure assumptions and parameters used to calculate theoretical exposures to the commercial octaBDE product by adults and children in the home, school, and office environment

Exposure Parameter	Variable	Units	Receptor Scenarios												Reference and Notes
			<1 yr Child	1-2 yr Child	3-5 yr Child	6-8 yr Child	9-11 yr Male Child	9-11 yr Female Child	12-14 yr Male Child	12-14 yr Female Child	15-18 yr Male Child	15-18 yr Female Child	Adult Male (Prospective Parent)	Adult Female (Prospective Parent)	
Physiological and General Assumptions															
Body Weight	BW	kg	9.1	11.3	16.35	23.75	33.75	34	47.05	48.65	64.93	57.6	78.1	65.4	USEPA (1997, 2000a) EFH, page 7-4, table 7-3 for children; table 7-2 for adults; mean values
Inhalation Rate	InR	m3/day	4.5	6.8	8.3	10	14	13	15	12	17	12	15.2	11.3	USEPA (1997, 2000a) EFH, page 5-24, table 5-23; long-term exposures
Indoor Dust Ingestion Rate	DIR	mg/day	10	10	10	3	3	3	3	3	3	3	0.56	0.56	USEPA (1997, 2000a) EFH, page 4-12, table 4-11 for children (winter indoor rate); page 4 17, table 4-16 for adults
Exposed Skin Surface Area, Total Body	SAt	cm2	5,910	5,910	7,200	9,250	11,600	11,600	14,900	14,800	17,500	16,000	19,400	16,900	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value represents total body skin surface area
Fraction of Skin Surface Area Exposed to Floor	FSAf	unitless	0.337	0.342	0.379	0.361	0.374	0.374	0.383	0.383	0.403	0.403	0.319	0.305	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value represents the estimate for the skin surface area of the hands, arms, legs, and feet
Fraction of Skin Surface Area Exposed at School	FSAs	unitless	0.272	0.279	0.306	0.289	0.301	0.301	0.308	0.308	0.332	0.332	0.251	0.238	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value represents the estimate for the skin surface area of the hands, arms, and legs
Fraction of Skin Surface Area Exposed at Office	FSAo	unitless	--	--	--	--	--	--	--	--	--	--	0.119	0.109	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value represents the estimate for the skin surface area of the hands and arms
Salivary Flow-rate in the Child's Mouth	Vs	ml/minute	0.22	0.22	0.22	--	--	--	--	--	--	--	--	--	Watanabe and Tatsukawa (1990); based on unstimulated salivary flow-rate in 5-year old children
Fractional Rate of Extraction by Saliva	FR	unitless	0.038	0.038	0.038	--	--	--	--	--	--	--	--	--	NRC (2000) upper end of range for laundering tests of fabric with flame retardant backing
Fraction of Items Mouthed that Are ABS Plastic Surfaces	Fplas	unitless	0.1	0.1	0.1	--	--	--	--	--	--	--	--	--	Assume 10% of items mouthed are ABS plastics treated with the commercial octaBDE product mixture
Exposure Duration/Frequency Assumptions															
Exposure Frequency at Home	EFh	hr/day	24	24	20	17	17	17	17	17	17	17	24	24	For children, time at school subtracted from 24-hour day; adults assumed to be stay-at-home parents
Exposure Frequency at School	EFs	hr/day	0	0	4	7	7	7	7	7	7	7	8	8	Assume 4-hour school day for pre-school and kindergarten, 7-hour school day for older children, and adult teachers work 8 hours/day
Exposure Frequency Mouthing Behavior	EFmouth	minutes/day	9	6	2	0	0	0	0	0	0	0	0	0	Juberg et al. (2001); based on mouthing behavior of "other objects" by children 0 to 36 months; children 1 - 2 years is the mid-point of estimates for 0-18 months and 19 to 36 months reported in the study
Exposure Frequency at Office	EFo	hr/day	0	0	0	0	0	0	0	0	0	0	8	8	Assume 8-hour work day at office
Environmental Assumptions															
Absorption Factor, Oral Route	AFo	unitless	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	ECB (2002) RA, page 132; because no value available, assumed to be 50%
Absorption Factor, Inhalation Route	AFi	unitless	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	ECB (1996); assumed to be below the generic value of 75% recommended for all chemicals in the absence of chemical-specific information
Absorption Factor, Dermal Route	AFd	unitless	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	Inverest Research (2001); mean dermal delivery of applied dose of radio-labeled tetrabromodiphenyl ether, 12 samples of human skin
Chemical Assumptions															
Thyroid Effects Benchmark, oral and inhalation	TEB	mg/kg-day	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	Derived from Zhou et al. (2001)
Developmental Effects Benchmark, oral and Inhalation	DEB	mg/kg-day	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	Derived from WIL Research (1986a)
Reference Dose, Oral	RfDo	mg/kg-day	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	USEPA (2003) IRIS; based on 1980 rat study that administered commercial product mixture (1.1% penta, 8.5% hexa, 45.1% hepta, 30.7% octa, 13.0% nona, 1.6% deca); critical effect: induction of hepatic enzymes, liver histopathology; uncertainty factor = 1000, modifying factor = none; low level of confidence in RfD
Reference Dose, Inhalation	RfDi	mg/kg-day	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	No data available in IRIS; used the oral RfD
Chemical Assumptions															
Product Concentration in Dust at Home, School and Office	Cdust	ng/g	410	410	410	410	410	410	410	410	410	410	410	410	Knoth (2002); 95th upper confidence limit of the mean of 12-25 samples collected from vacuum cleaner bags from German households; sum of BDE-47,49,85,99,100,153,154,183 (deca excluded)
Product Concentration in Particulate Phase at Home, School and Office	AirP	ng/m3	1.3	1.3	1.3	1.3	1.3	1.3	1.3	1.3	1.3	1.3	1.3	1.3	Sjodin (2001a); air concentration of particle-associated and semivolatile compounds associated with penta commercial product; sum of BDE-47,85,99,100,153,154 and 183 (deca excluded); high of two air samples collected in a teaching hall with computers in Sweden
Fraction of Particulate Phase that is Respirable	Fresp	unitless	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	Standard value used by regulatory agencies
Adherence Rate of Dust to Skin	ARdust	mg/cm2-day	0.050	0.050	0.050	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070	USEPA (2001) RAGS Part E, page 3-17, assumed same as soil adherence rate; 50th percentile of high-end activity (gardening) for adults, 95th percentile for children under 6 yr
Water Solubity of OctaBDE	WS	ug/L	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	ECB (2002) based on water solubility of 0.5 ug/L at 25^{0}C
Product Concentration on Surface of ABS plastic	Cplas	mg/cm2	7.5	7.5	7.5	--	--	--	--	--	--	--	--	--	Assumed to be similar to NRC (2000) high end estimates of the range for flame retardants applied to the back of fabrics used for cushions

Table 3-21. Exposure assumptions and parameters used to calculate theoretical exposures to the commercial octaBDE product by adults and children exposed to ambient environmental levels and through consumption of foods, fish, and breast milk (infants only)

			Receptor Scenarios																
Exposure Parameter	**Variable**	**Units**	**<1 yr Child**	**1-2 yr Child**	**3-5 yr Child**	**6-8 yr Child**	**9-11 yr Male Child**	**9-11 yr Female Child**	**12-14 yr Male Child**	**12-14 yr Female Child**	**15-18 yr Male Child**	**15-18 yr Female Child**	**Adult Male (Prospective Parent)**	**Adult Female (Prospective Parent)**	**Recreational Marine Angler Adult Male**	**Recreational Marine Angler Adult Female**	**Recreational Freshwater Angler Adult Male**	**Recreational Freshwater Angler Adult Female**	**Remarks/Reference**
Physiological and General Assumptions																			
Body Weight	BW	kg	9.1	11.3	16.35	23.75	33.75	34	47.05	48.65	64.93	57.6	78.1	65.4	78.1	65.4	78.1	65.4	USEPA (1997, 2000a) EFH, page 7-4, table 7-3 for children; table 7-2 for adults; mean values
Inhalation Rate	InR	m3/day	4.5	6.8	8.3	10	14	13	15	12	17	12	15.2	11.3	--	--	--	--	USEPA (1997, 2000a) EFH, page 5-24, table 5-23
Soil Ingestion Rate	SIR	mg/day	200	200	200	200	200	200	200	200	200	200	100	100	100	100	100	100	USEPA (1997, 2000a) EFH, page 4-21, recommended value for adults (agricultural/residential setting); page 4-20, recommended conservative estimate for children
Fish Consumption Rate	CRfish	g/day	1.001	4.181	5.232	6.175	8.775	8.84	9.41	9.73	12.986	11.52	20.1	20.1	7.2	7.2	17	17	USEPA (2000a) CEFH, page 3-78, table 3-35, Mean values for children; USEPA (1997) EFH, page 10-79, table 10-81, Mean Intake for total fish; page 10-79, tables 10-83 and 84, Highest Mean intake for anglers
Meat (Beef, Poultry and Pork) Consumption Rate	CRmeat	g/day	53.69	115.26	153.69	161.5	229.5	231.2	230.545	238.385	318.157	282.24	398.31	333.54	--	--	--	--	USEPA (2000a) CEFH, page 3-78, table 3-35, 95th percentile values for children; USEPA (1997) EFH, page 11-31, table 11-30, 95th percentile values for adults
Dairy Consumption Rate	CRdairy	g/day	2138.5	1019.26	797.88	795.625	1130.625	1139	837.49	865.97	1155.754	1025.28	2319.57	1942.38	--	--	--	--	USEPA (2000a) CEFH, page 3-78, table 3-35, 95th percentile values for children; USEPA (1997) EFH, page 11-31, table 11-30, 95th percentile values for adults
Other Fats and Oils Consumption Rate	CRfat	g/day	3.3	16	22	30	30	30	49	49	49	49	118.28	76.52	--	--	--	--	USEPA (2000a) CEFH, page 3-45, table 3-19, 95th percentile values for children (no overlap with other fatty foods); USEPA (1997) EFH, page 11-28, table 11-27, mean values for adult total fat intake (overlaps other fatty foods)
Egg Consumption Rate	CRegg	g/day	38	70	63	66	66	66	88	88	88	88	83.33	69.78	--	--	--	--	USEPA (2000a) CEFH, page 3-45, table 3-19, 95th percentile values for children; USEPA (1997) EFH, page 11-15, table 11-7, 95th percentile values for adults
Vegetable Consumption Rate	CRveg	g/day	220.22	263.29	299.205	320.625	455.625	459	437.565	452.445	603.849	535.68	781.00	654.00					USEPA (2000a) CEFH, page 3-78, table 3-19, 95th percentile values for children; USEPA (1997) EFH, page 11-15, table 11-7, 95th percentile values for adults
Breast Milk Consumption Rate	CRbm	mL/day	980	0	0	0	0	0	0	0	0	0	0	0	--	--	--	--	USEPA (2000a) CEFH, page 2-19, table 2-12 ,12-month average
Fraction of Breast Milk that is Lipid	Fbm	unitless	0.04	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	--	--	--	--	USEPA (2000a) CEFH, page 2-19, table 2-12 , footnote
Exposed Skin Surface Area, Total Body	SAt	cm^2	5,910	5,910	7,200	9,250	11,600	11,600	14,900	14,800	17,500	16,000	19,400	16,900	--	--	--	--	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value represents total body skin surface area
Fraction of Skin Surface Area Exposed Outdoors	FSAout	unitless	0.337	0.342	0.379	0.361	0.374	0.374	0.383	0.383	0.403	0.403	0.319	0.305	--	--	--	--	USEPA (2001) Dermal Exposure Guidance, Exhibit C-1 page C-3 and C-4; 50th percentile, though in some cases the value represents the midpoint of the age bin; value represents skin surface area for head + hands + arms + legs + feet
Environmental Assumptions																			
Absorption Factor, Oral Route	AFo	unitless	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	ECB (2002) RA, page 132; because no value available, assumed to be 50%
Absorption Factor, Inhalation Route	AFi	unitless	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	0.75	ECB (1996); assumed to be below the generic value of 75% recommended for all chemicals in the absence of chemical-specific information
Absorption Factor, Dermal Route	AFd	unitless	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	0.0313	Inverest Research (2001); mean dermal delivery of applied dose of radio-labeled tetrabromodiphenyl ether, 12 samples of human skin
Chemical Potency Assumptions																			
Thyroid Effects Benchmark, oral and inhalation	TEB	mg/kg-day	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	Derived from Zhou et al. (2001)
Developmental Effects Benchmark, oral and Inhalation	DEB	mg/kg-day	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.09	Derived from WIL Research (1986a)
Reference Dose, Oral	RfDo	mg/kg-day	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	USEPA (2003) IRIS; based on 1980 rat study that administered commercial product mixture (1.1% penta, 8.5% hexa, 45.1% hepta, 30.7% octa, 13.0% nona, 1.6% deca); critical effect: induction of hepatic enzymes, liver histopathology; uncertainty factor = 1000, modifying factor = none; low level of confidence in RfD
Reference Dose, Inhalation	RfDi	mg/kg-day	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	No data available in IRIS; used the oral RfD
Chemical Assumptions																			
Soil Adherence to Skin Rate	SAR	mg/cm2-day	0.200	0.200	0.200	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070	0.070	USEPA (2001) RAGS Part E, page 3-17; 50th percentile of high-end activity (gardening) for adults, 95th percentile value for children under 6 yr
Air Concentration of Product in Particulate Phase	AirP	pg/m3	52	52	52	52	52	52	52	52	52	52	52	52	52	52	52	52	Strandberg (2001); urban location (Chicago) over three-year period (1997-99), total of BDE-47,99,100,153,154
Fraction of Particulate Phase that is Respirable	Fresp	unitless	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	Standard value used by regulatory agencies
Concentration in Soil	Csoil	µg/kg	76	76	76	76	76	76	76	76	76	76	76	76	76	76	76	76	Hale (2002); Soil near foam production plant in mid-Atlantic region, USA, that uses penta mixture (no data available for octa mixture in soil)
Concentration in Fish	Cfish	ng/g wet wt	71.67	71.67	71.67	71.67	71.67	71.67	71.67	71.67	71.67	71.67	71.67	71.67	71.67	71.67	71.67	71.67	The 95th percent upper confidence limit of the mean value of 72 fish fillet samples from the USA, total PBDE
Concentration in Meat (Beef, Poultry and Pork)	Cmeat	ng/g wet wt	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	0.0458	Darnerud (2000; unpublished); Swedish market basket study, sum of BDE-47,99,100,153,154
Concentration in Dairy	Cdairy	ng/g wet wt	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	0.0182	Darnerud (2000; unpublished); Swedish market basket study, sum of BDE-47,99,100,153,154
Concentration in Other Fats and Oils	Cfat	ng/g wet wt	0.158	0.158	0.158	0.158	0.158	0.158	0.158	0.158	0.158	0.158	0.158	0.158	0.158	0.158	0.158	0.158	Darnerud (2000; unpublished); Swedish market basket study, sum of BDE-47,99,100,153,154
Concentration in Eggs	Cegg	ng/g wet wt	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	0.0425	Darnerud (2000; unpublished); Swedish market basket study, sum of BDE-47,99,100,153,154
Concentration in Vegetables	Cveg	ng/g wet wt	0.134	0.134	0.134	0.134	0.134	0.134	0.134	0.134	0.134	0.134	0.134	0.134	0.134	0.134	0.134	0.134	Ohta (2002); Concentration detected in spinach, sum of BDE-28,47,99,100,153,154
Concentration in Breast Milk	Cbm	ng/g lipid	42.8	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	Ryan et al. (2002); mean of the sum of BDE-28,47,99,100,153,154,183; 20 samples from Vancouver, Canada, collected in 2001-2002

Table 3-22. Exposure variables and their definitions used in the Tier 1 exposure model calculations

Variable	Definition
A	area of ABS plastic mouthed (cm^2/hr)
AFd	dermal absorption factor (unitless)
AFi	inhalation absorption factor (unitless)
AFo	oral absorption factor (unitless)
AirP	product concentration in particulate phase at home, school and office (ng/m^3)
AirTP	total particulate concentration (mg/m^3)
ARdust	adherence rate of dust to skin (mg/cm^2-day)
ARshoe	adherence rate of item to shoe (mg/cm^2-day)
ARskin	adherence rate of handled item to skin (mg/cm^2-day)
BW	body weight (kg)
Cbm	concentration in breast milk (ng/g lipid weight)
CDI	chronic daily intake (mg/kg-day)
Cdairy	concentration in dairy (ng/g ww)
Cdust	concentration of the commercial pentaBDE product in indoor dust (ng/g);
Cegg	concentration in egg (ng/g ww)
CF	a conversion factor
Cfat	concentration in other fats and oils (ng/g ww)
Cfish	concentration in fish (ng/g ww)
Cfloor	concentration of the commercial pentaBDE product on the floor (mg/cm^2) for the maximally exposed worker and is calculated using the variable definitions provided in the previous section
Cmeat	concentration in meat, beef, poultry, and pork (ng/g ww)
Cplas	product concentration on surface of ABS plastic (mg/cm^2)
Cveg	concentration in vegetables (ng/g ww)
CRbm	breast milk consumption rate (mL/day)
CRdairy	dairy consumption rate (g/day)
CRegg	egg consumption rate (g/day)
CRfat	other fats and oil consumption rate (g/day)
CRfish	fish consumption rate (g/day)
CRmeat	meat (beef, poultry, and pork) consumption rate (g/day)
CRveg	vegetable consumption rate (g/day)
CSF	cancer slope factor $(mg/kg\text{-}day)^{-1}$
Csoil	concentration of the commercial pentaBDE product in outdoor soil (µg/kg)
DEB	developmental effects benchmark, oral and inhalation (mg/kg-day)
DIR	indoor dust ingestion rate (mg/day)
EFF	exposure frequency for contact with floors (hr/day)
EFh/s/o	exposure frequency at home/school/office (hr/day)
EFL	average amount of time spent daily doing laundry (hr/day)
EFmouth	exposure frequency mouthing all items (minutes/day)
Fbm	fraction of breast milk that is lipid (unitless)
FCLdy	fraction of laundry that is contaminated (unitless)
Fip	fraction of handled item that is commercial product (unitless)
Fplas	fraction of items mouthed that are ABS plastic (unitless)
Fpp	fraction of particulate that is commercial product (unitless)
FQ	frequency of hand-to-mouth activity in the workplace (events/day)
FR	fractional rate of extraction by saliva (unitless)
Fresp	fraction of particulate phase that is respirable (unitless)
FSA	floor surface area (cm^2)
FSAf	fraction of skin surface area exposed to floor (unitless)

Variable	Definition
FSAftip	fraction of skin surface area that contacts mouth (unitless)
FSAw	fraction of skin surface area exposed at work (unitless)
FSAh/s/o	fraction of skin surface area exposed at home/school/office (unitless)
FSAl	fraction of skin surface area exposed while doing laundry (unitless)
FSAout	fraction of skin surface area exposed outdoors (unitless)
FSAs	fraction of skin surface area that is shoes (unitless)
FWD	fraction of work day spent around commercial product (unitless)
InR	inhalation rate (m^3/day)
RfD	reference dose, oral and inhalation (mg/kg-day)
SAR	adherence rate of soil to skin (mg/cm^2-day)
SAt	exposed skin surface area, total body (cm^2)
SIR	soil ingestion rate (mg/day)
TEB	thyroid effects benchmark, oral and inhalation (mg/kg-day)
WEL	workplace exposure limit, inhalation (mg/m^3)
WS	water solubility limit (µg/L)
Vs	Salivary flow-rate in child's mouth (ml/minute)

Dermal Uptake Pathway

Dermal uptake is a function primarily of the degree of skin contact. The exposed skin surface area of primary and chain-of-commerce manufacturing workers was assumed to include only the palms of the hands. Restrictions on direct contact with primary product or plastics by workers (e.g., by wearing gloves and hand washing) was not considered. Dermal protection in the primary production workplace is required during handling of the product. Worker protection measures in chain-of-commerce facilities are not known. Laundry contact was limited to the hands only, and floor contact included hands, arms, legs and bare feet. For adults and children, exposed skin contact with indoor dust varied according to the three settings: home (hands, arms, legs and feet), school (hands, arms and legs) and office (hands and arms). Values for exposed skin surface areas were adopted from USEPA (2001, 2000a, 1997).

Assumptions regarding soil and dust adherence to skin were adopted from USEPA guidance for dermal risk assessment (USEPA, 2001, 2000a, 1997). The fraction of impregnated plastic materials item that is the commercial octaBDE product was based on information from GLCC. Laundry was assumed to be 25% soiled for all job categories. Soil adherence to shoes was assumed to be the same as that of bare skin.

Exposure Pathway – Direct Dermal Contact

Workers. The potential for dermal contact with the commercial octaBDE product was calculated using the following equation:

$$CDI = SAt * SAh * FSAI * ARskin * Fip * AFd * FWD * 1/BW$$

Exposure Pathway – Dermal Contact with Dirty Laundry in the Home

Workers. The potential for dermal contact with the commercial octaBDE product from handling dirty laundry was calculated using the following equation:

CDI = SAt * SAh* ARskin * FCLdy * AFd * EFL * CF2 * 1/BW

Exposure Pathway – Dermal Contact with Floor Surfaces Contaminated by Dirty Shoes in the Home

Workers. The standard home floor surface area (756,644 cm^2) that potentially retains dirt from outdoor sources was adopted from USEPA (1997). The potential for dermal contact with the commercial octaBDE product from touching floor surfaces that have been contaminated by dirty shoes brought home from the workplace was calculated using the following equation:

Cfloor = (SAs * ARshoe) / FSA
CDI = Cfloor * SAt* FSAf * AFd * EFF * CF2 * 1/BW

Worker's Children. The potential for dermal contact with the commercial octaBDE product by children touching floor surfaces that have been contaminated by a parent's dirty shoes was calculated using the following equation:

Cfloor = (SAs * ARshoe) / FSA
CDI = Cfloor * SAt* FSAf * AFd * EFF * CF2 * 1/BW

Exposure Pathway – Dermal Contact with Indoor Dust

Adults and Children in the Indoor Home, School and Office. The potential for dermal contact with the commercial octaBDE product in indoor dust in the indoor home, school and office was calculated using the following equation:

CDI = Cdust * CF1 * ARdust * SAt * FSAh/s/o * AFd * EFh/s/o * CF3 * 1/BW

Exposure Pathway – Dermal Contact with Outdoor Soil

Adult and Child Ambient Environmental Exposures. The potential for dermal contact with the commercial octaBDE product in outdoor soil was calculated using the following equation:

CDI = Csoil * CF1 * SAR * SAt * FSAout * AFd * 1/BW

Incidental Ingestion

Incidental ingestion via hand-to-mouth contact was modeled for workers and workers' children. The person's fingertips were assumed to contact the mouth and transfer any adhered commercial octaBDE product to the mouth. Swallowing and absorption were assumed to follow. The fraction of the both hands contacting the mouth was assumed to include the fingertips of all ten fingers. For adults and children, dust or soil ingestion rates were used to calculate incidental ingestion exposures. Exposure parameter values were adopted primarily from the USEPA Exposure Factors Handbook (1997) and USEPA risk assessment guidelines (1989).

Estimates of soil ingestion by the average adult and children of different ages are the focus of continued scientific study (Paustenbach, 2000). The dust ingestion values used in this Tier 1 assessment are from USEPA Exposure Factors Handbook (1997) as follows: 100 mg/day for the youngest children, 3 mg/day for older children and 0.56 mg/day for adults. Soil ingestion values, also from the same source, were 200 mg/day for children and 100 mg/day for adults. The results of nearly all studies used in risk assessment for incidental ingestion are based on the measurement of nonmetabolizable soil tracers in feces and urine (Paustenbach, 2000; Davis et al., 1990; van Wijnen et al., 1990; Calabrese et al., 1989, 1990; Clausing et al., 1987; Binder et al., 1986).

Exposure Pathway – Incidental Ingestion from Hand-to-Mouth Contact

Workers. The potential intake of the commercial octaBDE product through incidental ingestion was calculated using the following equation:

CDI = SAt * FSAftip * ARskin * Fip * AFo * FWD * 1/BW

Exposure Pathway – Incidental Ingestion from Hand-to-Mouth Contact while Doing Laundry

Worker. The potential intake of the commercial octaBDE product through incidental ingestion while handling dirty laundry was calculated using the following equation:

CDI = SAt * FSAftip * ARskin * FCLdy * AFo * EFL * CF2 * 1/BW

Exposure Pathway – Incidental Ingestion from Hand-to-Mouth with Floor Surfaces Contaminated by Dirty Shoes in the Home

Workers. The standard home floor surface area (756,644 cm^2) that potentially retains dirt from outdoor sources was adopted from USEPA (1997). The potential intake of the commercial octaBDE product through incidental ingestion from touching floor surfaces that have been contaminated by dirty shoes was calculated using the following equation:

Cfloor = (SAs * ARshoe) / FSA
CDI = Cfloor * SAt * FSAftip * AFo * EFF * CF2 * 1/BW

Worker's Children. The potential intake of the commercial octaBDE product by children through incidental ingestion from touching floor surfaces that have been contaminated by a parent's dirty shoes was calculated using the following equation:

Cfloor = (SAs * ARshoe) / FSA
CDI = Cfloor * SAt * FSAftip * AFo * EFF * CF2 * 1/BW

Exposure Pathway – Incidental Ingestion of Indoor Dust from Hand-to-Mouth

Adults and Children in the Indoor Home, School and Office. The potential for incidental ingestion with the commercial octaBDE product in indoor dust in the indoor home, school and office environments was calculated using the following equation:

CDI = Cdust * CF1 * DIR * AFo * EFh/s/o * CF2 * 1/BW

Exposure Pathway – Incidental Ingestion from Hand-to-Mouth Contact with Outdoor Soil

Adult and Child Ambient Environmental Exposures. The potential for incidental ingestion from hand-to-mouth contact with the commercial octaBDE product in outdoor soil was calculated using the following equation:

CDI = Csoil * CF * SIR * AFo * 1/BW

Ingestion of Food

Exposure models describing the ingestion of food products, fish, and breast milk (infants only) were developed for adults and children as part of the assessment of ambient environmental exposures to the commercial octaBDE product. The environmental exposure data used in these models are summarized in Table 3-15. U.S data describing levels in fish and human breast milk were used in the assessment. In the absence of sufficient U.S. data, Swedish and Japanese market basket data were used to evaluate potential exposures through the relevant dietary categories – meat (beef, poultry and pork), dairy, eggs, other fats and oils, and vegetables. Exposure assumptions describing food consumption habits for adults and different child age bins were adopted from the USEPA Exposure Factors Handbook (1997). In order to avoid combining multiple high-end values, either the high end estimate of the food concentration or the high-end estimate of the consumption rate was used in the assessment (and not both high-end estimates).

Exposure Pathway – Ingestion of Fish

Adult and Child Ambient Environmental Exposures. The potential for intake of the commercial octaBDE product through ingestion of fish was calculated using the following equation:

CDI = Cfish * CF * CRfish * AFo * 1/BW

Exposure Pathway –Ingestion of Food

Adult and Child Ambient Environmental Exposures. The potential for intake of the commercial octaBDE product through ingestion of different food groups was calculated using an equation similar to that used for fish consumption. The parameters for the concentration term and consumption rate were replaced with appropriate parameters for each of the different food groups evaluated in the assessment: meat (beef, poultry and pork) – Cmeat, CRmeat; vegetables – Cveg, CRveg; dairy – Cdairy, CRdairy; Eggs – Cegg, CRegg; and other fats and oils – Cfat, CRfat.

Exposure Pathway –Ingestion of Human Breast Milk by Infants

Infant Exposures. The potential for intake of the commercial octaBDE product through ingestion of human breast milk was calculated only for infants using the following equation:

CDI = Cbm * CF1 * Fbm * CRbm * CF2 * AFo * 1/BW

Ingestion via Mouthing Behavior

The indoor home/school/office scenario included mouthing of ABS plastic treated with the commercial octaBDE product and subsequent ingestion as a plausible source of exposure to children less than 5 years old. According to the U. S. Consumer Product Safety Commission (USCPSC) Chronic Health Advisory Panel behavioral study of mouthing behavior in U.S. children, mouthing behavior tends to decline dramatically after age 3 (USCPSC, 2002; USEPA, 2000a). Similar observations are reported by Juberg et al. (2001). The behavioral observation data collected as part of the U.S. Consumer Products Safety Commission (USCPSC) evaluation of children exposures to diisononyl phthalate (a chemical plasticizer in plastic toys) and the data reported in the published literature indicate that mouthing behavior declines as children age. According to USCPSC (2002), evaluation of chemical exposures to children over 3 years old via mouthing activity are very likely to experience lower intakes than younger children.

For the purposes of this screening-level Tier 1 exposure assessment, exposure to the commercial octaBDE product via mouthing behavior was evaluated in children representing the <1 year, 1-2 year, and 3-5 year age groups. Data describing the surface concentration of the commercial octaBDE product or BDEs on ABS plastic surfaces are not available in the published literature. In the absence of data, the water solubility limit of the commercial octaBDE product (0.5 µg/L; ECB, 2002) was used as a surrogate for the exposure point concentration. The amount of commercial octaBDE product ingested was calculated by assuming that 100% of the commercial octaBDE product transfered from the surface of ABS plastic to saliva at the water solubility limit, and the saliva swallowed by the child. Based on the data reported by Juberg et al. (2001), children ,1, 1-2, and 3-5 years old mouthed items other than pacifiers, toys, and baby teething objects for 9, 5.5, and 2 minutes each day, respectively. For children 1-2 years old, 5.5 minutes/day in the mid-point between results reported by Juberg et al. (2001) for 0-18 months (9 minutes/day) and 19-36 months (2 minutes/day). An unstimulated salivary flow rate of 0.22 ml/minute was assumed for the three age groups based on data reported by Watanabe et al. (1990) for 5-year old children. The amount of time each day that young children might engage in mouthing behavior of ABS plastic treated with the commercial octaBDE product is consistent with NRC (2000) and USEPA (2000a).

Exposure Pathway – Ingestion from Mouthing Treated ABS Plastic

Children in the Indoor Home, School and Office Environment. Hypothetical exposure via ingestion by mouthing of ABS plastic treated with the commercial octaBDE product was evaluated only for the home environment. Exposure was calculated using the following equation:

CDI = WS * Vs * CF1 * CF2 * FR * AFo * EFmouth * 1/BW

Inhalation

The inhalation exposure pathway was considered a plausible source of exposure to primary and chain of commerce manufacturing workers and to adults and children. Workplace exposures were based on air particulate measurements collected in the primary manufacturing workplace by GLCC (2002) during the production of the commercial octaBDE product. In this exposure scenario, the use of PPE or other measures used by GLCC were not considered. The use of PPE would reduce intakes by inhalation and subsequent ingestion by approximately 33-fold. Air particulate measurements in workplaces where the commercial octaBDE product is used to manufacture ABS plastics and casings for consumer electronic equipment were based on data from Breysse Kacergis (2000). Because recycling and shredding of plastics is an important chain-of-commerce activity, air particulate measurements collected by Sjödin (2001a) were used to represent workplace exposures for this activity. For adults and children potentially exposed in the indoor home/school/office environment and ambient environment, indoor and outdoor air particulate data reported in the literature are used as exposure point concentrations (see Table 3-18). One-half of the measured particulate level was assumed to be respirable; the remaining fraction was assumed to be inhaled and swallowed. Inhalation rates for workers were adopted from USEPA (1997) based on the level of activity (light, moderate or heavy) appropriate for each job category.

Exposure Pathway – Inhalation of Respirable Particulates

Workers in Primary and Chain-of-Commerce Manufacturing Activities. The inhalation of respirable indoor particulates that contain the commercial octaBDE product was calculated using the following equation:

CDI = AirTP * Fresp * Fpp * InR * AFi * FWD * 1/BW

Adults and Children in the Indoor Home, School and Office. The inhalation of respirable indoor particulates that contain the commercial octaBDE product was calculated using the following equation:

CDI = AirTP * CF1 * Fresp * InR * AFi * EFh/s/o * CF2 * 1/BW

Adult and Child Ambient Environmental Exposures. The inhalation of respirable outdoor particulates that contain the commercial octaBDE product was calculated using the following equation:

CDI = AirP * CF * Fresp * InR * AFi * 1/BW

Exposure Pathway – Swallowing of Inhaled Particulates

Workers in Primary and Chain-of-Commerce Manufacturing Activities. The inhalation and subsequent swallowing of air particulates that contain the commercial octaBDE product was calculated using the following equation:

CDI = AirTP * (1 - Fresp) * Fpp * InR * AFo * FWD * 1/BW

Adults and Children in the Indoor Home, School and Office. The inhalation and subsequent swallowing of indoor air particulates that contain the commercial octaBDE product was calculated using the following equation:

CDI = AirTP * CF1 * (1 - Fresp) * InR * AFo * EFh/s/o * CF2 * 1/BW

Adult and Child Ambient Environmental Exposures. The inhalation and subsequent swallowing of outdoor particulates that contain the commercial octaBDE product was calculated using the following equation:

CDI = AirP * CF * (1 - Fresp) * InR * AFo * 1/BW

3.12 Results of the Exposure Assessment

Theoretical CDIs of the commercial octaBDE product by prospective parents associated with the eight different oral, dermal, and inhalation exposure pathways evaluated in scenario #1 (workplace exposures) are presented in Table 3-23. Theoretical CDIs were highest for the air particulate inhalation and incidental swallowing, dermal contact, and incidental ingestion via hand to mouth contact pathways.

Theoretical CDIs of the commercial octaBDE product by children of prospective parents engaged in different primary and chain-of-commerce manufacturing activities associated with two different exposure pathways evaluated as part of scenario #1 are presented in Table 3-24. The highest theoretical exposure was associated with dermal contact with floor surfaces in the worker's home. The pathway assumes that the soles of shoes worn by primary production workers in the workplace are contaminated to a level similar to that on bare skin. The shoes are brought home and the commercial octaBDE product on the bottom of the shoes is completely transferred to the floor surface.

Table 3-23. Chronic Daily Intakes (CDI) of the commercial octaBDE product by different adult receptor groups engaged in primary manufacturing or chain-of-commerce activities associated with ABS plastics

	CDI	CDI	CDI	CDI	CDI	CDI	CDI	CDI	CDI
Receptor	Inhalation of Respirable Particulates	Ingestion of Particles Inhaled and Swallowed	Dermal Contact	Incidental Ingestion Hand-to-Mouth Contact	Dermal Contact with Laundry	Incidental Ingestion Hand-to-Mouth Contact during Laundry	Dermal Contact with Floor Surfaces	Incidental Ingestion Hand-to-Mouth Contact with Floor Surfaces	Total
	mg/kg-day	mg/kg-day	mg/kg-day	mg/kg-day	mg/kg-day	mg/kg-day	mg/kg-day	mg/kg-day	mg/kg-day
Primary Production Grinder, Male	0.005	0.028	0.002	0.002	4.6E-05	5.7E-05	3.1E-05	1.3E-10	0.04
Primary Production Grinder, Female	0.01	0.033	0.002	0.002	4.4E-05	5.9E-05	2.5E-05	1.5E-10	0.04
Primary Production Packager, Male	0.005	0.028	0.002	0.002	4.6E-05	5.7E-05	3.1E-05	1.3E-10	0.04
Primary Production Packager, Female	0.01	0.033	0.002	0.002	4.4E-05	5.9E-05	2.5E-05	1.5E-10	0.04
Plastic/Pellet Manufacture Mixer, Male	0.002	0.010	0.002	0.002	4.6E-05	5.7E-05	3.1E-05	1.3E-10	0.02
Plastic/Pellet Manufacture Mixer, Female	0.002	0.012	0.002	0.002	4.4E-05	5.9E-05	2.5E-05	1.5E-10	0.02
Casing Manufacture, Male	0.001	0.007	0.001	0.001	4.6E-05	5.7E-05	3.1E-05	1.3E-10	0.01
Casing Manufacture, Female	0.001	0.009	0.001	0.002	4.4E-05	5.9E-05	2.5E-05	1.5E-10	0.01
Equipment Assembler, Male	0.001	0.007	0.001	0.001	4.6E-05	5.7E-05	3.1E-05	1.3E-10	0.01
Equipment Assembler, Female	0.001	0.009	0.001	0.002	4.4E-05	5.9E-05	2.5E-05	1.5E-10	0.01
Distributor Uncovered, Male	0.001	0.007	0.001	0.001	4.6E-05	5.7E-05	3.1E-05	1.3E-10	0.01
Distributor Uncovered, Female	0.001	0.009	0.001	0.002	4.4E-05	5.9E-05	2.5E-05	1.5E-10	0.01
Recycler, Male	3.2E-07	1.9E-06	0.001	0.001	4.6E-05	5.7E-05	3.1E-05	1.3E-10	0.003
Recycler, Female	3.8E-07	2.3E-06	0.001	0.002	4.4E-05	5.9E-05	2.5E-05	1.5E-10	0.003

Table 3-24. Chronic Daily Intakes (CDI) of the commercial octaBDE product by children of adult workers engaged in different primary manufacturing or chain-of-commerce activities associated with ABS plastics

Receptor	CDI Dermal Contact with Floor Surfaces	CDI Incidental Ingestion Hand-to-Mouth Contact with Floor Surfaces	CDI Total
	mg/kg-day	mg/kg-day	mg/kg-day
<1 yr Child	2.3E-05	3.3E-06	2.6E-05
1-2 yr Child	5.7E-05	7.9E-06	6.5E-05
3-5 yr Child	5.3E-05	6.7E-06	6.0E-05
6-8 yr Child	4.5E-05	5.9E-06	5.0E-05
9-11 yr Male Child	4.1E-05	5.2E-06	4.6E-05
9-11 yr Female Child	4.0E-05	5.2E-06	4.6E-05
12-14 yr Male Child	3.8E-05	4.8E-06	4.3E-05
12-14 yr Female Child	3.7E-05	4.6E-06	4.2E-05
15-18 yr Male Child	3.4E-05	2.7E-06	3.7E-05
15-18 yr Female Child	3.5E-05	2.8E-06	3.8E-05

Theoretical CDIs of the commercial octaBDE product by prospective parents and children associated with four different oral, dermal, and inhalation exposure pathways evaluated in scenario #2 (exposures in the indoor home/school/office environment) are presented in Table 3-25. A fifth exposure pathway, ingestion of the commercial octaBDE product via mouthing of ABS plastic surfaces, was evaluated for three different child age bins (<1 years, 1-2 years, 3-5 years old). The theoretical CDIs were less than approximately 1 ng/kg-day for all pathways and all receptor groups.

Theoretical CDIs of the commercial octaBDE product by prospective parents and children associated with 11 different oral, dermal, and inhalation exposure pathways evaluated in scenario #3 (ambient environmental exposures) are presented in Table 3-26. The theoretical CDIs were generally less than or equal to approximately 1 µg/kg-day for all pathways and all receptor groups.

Table 3-25. Chronic daily intakes (CDI) of the commercial octaBDE product by different adult and child receptor groups potentially exposed in the indoor home, school, and office environments.

Receptor	CDI Inhalation of Respirable Particulates mg/kg-day	CDI Ingestion of Particles Inhaled and Swallowed mg/kg-day	CDI Dermal Contact mg/kg-day	CDI Incidental Ingestion via Hand to Mouth Contact mg/kg-day	CDI Ingestion from Mouthing ABS Plastic mg/kg-day	CDI Total mg/kg-day
Exposures in the Home Environment						
<1 yr Child	2.4E-07	1.6E-07	1.4E-07	2.3E-07	2.1E-09	7.7E-07
1-2 yr Child	2.9E-07	2.0E-07	1.1E-07	1.8E-07	1.0E-09	7.9E-07
3-5 yr Child	2.1E-07	1.4E-07	8.9E-08	1.0E-07	2.6E-10	5.4E-07
6-8 yr Child	1.5E-07	9.7E-08	8.9E-08	1.8E-08	NA	3.5E-07
9-11 yr Male Child	1.4E-07	9.5E-08	8.2E-08	1.3E-08	NA	3.3E-07
9-11 yr Female Child	1.3E-07	8.8E-08	8.1E-08	1.3E-08	NA	3.1E-07
12-14 yr Male Child	1.1E-07	7.3E-08	7.7E-08	9.3E-09	NA	2.7E-07
12-14 yr Female Child	8.5E-08	5.7E-08	7.4E-08	9.0E-09	NA	2.3E-07
15-18 yr Male Child	9.0E-08	6.0E-08	6.9E-08	6.7E-09	NA	2.3E-07
15-18 yr Female Child	7.2E-08	4.8E-08	7.1E-08	7.6E-09	NA	2.0E-07
Adult Male (Prospective Parent)	9.5E-08	6.3E-08	7.1E-08	1.5E-09	NA	2.3E-07
Adult Female (Prospective Parent)	8.4E-08	5.6E-08	7.1E-08	1.8E-09	NA	2.1E-07
Exposures in the School Environment						
<1 yr Child	NA	NA	NA	NA	NA	NA
1-2 yr Child	NA	NA	NA	NA	NA	NA
3-5 yr Child	4.1E-08	2.7E-08	1.4E-08	2.1E-08	NA	1.0E-07
6-8 yr Child	6.0E-08	4.0E-08	2.9E-08	7.6E-09	NA	1.4E-07
9-11 yr Male Child	5.9E-08	3.9E-08	2.7E-08	5.3E-09	NA	1.3E-07
9-11 yr Female Child	5.4E-08	3.6E-08	2.7E-08	5.3E-09	NA	1.2E-07
12-14 yr Male Child	4.5E-08	3.0E-08	2.6E-08	3.8E-09	NA	1.0E-07
12-14 yr Female Child	3.5E-08	2.3E-08	2.5E-08	3.7E-09	NA	8.7E-08
15-18 yr Male Child	3.7E-08	2.5E-08	2.3E-08	2.8E-09	NA	8.8E-08
15-18 yr Female Child	3.0E-08	2.0E-08	2.4E-08	3.1E-09	NA	7.7E-08
Adult Male (Prospective Parent)	3.2E-08	2.1E-08	1.9E-08	4.9E-10	NA	7.2E-08
Adult Female (Prospective Parent)	2.8E-08	1.9E-08	1.8E-08	5.9E-10	NA	6.6E-08
Exposures in the Office Environment						
<1 yr Child	NA	NA	NA	NA	NA	NA
1-2 yr Child	NA	NA	NA	NA	NA	NA
3-5 yr Child	NA	NA	NA	NA	NA	NA
6-8 yr Child	NA	NA	NA	NA	NA	NA
9-11 yr Male Child	NA	NA	NA	NA	NA	NA
9-11 yr Female Child	NA	NA	NA	NA	NA	NA
12-14 yr Male Child	NA	NA	NA	NA	NA	NA
12-14 yr Female Child	NA	NA	NA	NA	NA	NA
15-18 yr Male Child	NA	NA	NA	NA	NA	NA
15-18 yr Female Child	NA	NA	NA	NA	NA	NA
Adult Male (Prospective Parent)	3.2E-08	2.1E-08	8.9E-09	4.9E-10	NA	6.2E-08
Adult Female (Prospective Parent)	2.8E-08	1.9E-08	8.4E-09	5.9E-10	NA	5.6E-08

NA = not applicable

Table 3-26. Chronic Daily Intakes (CDI) of the commercial octaBDE product by different adult and child receptor groups associated with ambient environmental exposures.

Receptor	CDI Inhalation of Respirable Particulates mg/kg-day	CDI Ingestion of Particles Inhaled and Swallowed mg/kg-day	CDI Dermal Contact mg/kg-day	CDI Incidental Ingestion via Hand to Mouth Contact mg/kg-day	CDI Fish Consumption mg/kg-day	CDI Meat Consumption mg/kg-day	CDI Dairy Consumption mg/kg-day	CDI Fats & Oil Consumption mg/kg-day	CDI Egg Consumption mg/kg-day	CDI Vegetable Consumption mg/kg-day	CDI Breast Milk Consumption mg/kg-day	CDI Total mg/kg-day
<1 yr Child	9.6E-09	6.4E-09	1.0E-07	8.4E-07	3.9E-06	1.4E-07	2.1E-06	2.9E-08	8.9E-08	1.2E-05	9.2E-05	1.1E-04
1-2 yr Child	1.2E-08	7.8E-09	8.5E-08	6.7E-07	1.3E-05	2.3E-07	8.2E-07	1.1E-07	1.3E-07	1.6E-06	NA	1.7E-05
3-5 yr Child	9.9E-09	6.6E-09	7.9E-08	4.6E-07	1.1E-05	2.2E-07	4.4E-07	1.1E-07	8.2E-08	1.2E-06	NA	1.4E-05
6-8 yr Child	8.2E-09	5.5E-09	2.3E-08	3.2E-07	9.3E-06	1.6E-07	3.0E-07	1.0E-07	5.9E-08	9.0E-07	NA	1.1E-05
9-11 yr Male Child	8.1E-09	5.4E-09	2.1E-08	2.3E-07	9.3E-06	1.6E-07	3.0E-07	7.0E-08	4.2E-08	9.0E-07	NA	1.1E-05
9-11 yr Female Child	7.5E-09	5.0E-09	2.1E-08	2.2E-07	9.3E-06	1.6E-07	3.0E-07	7.0E-08	4.1E-08	9.0E-07	NA	1.1E-05
12-14 yr Male Child	6.2E-09	4.1E-09	2.0E-08	1.6E-07	7.2E-06	1.1E-07	1.6E-07	8.2E-08	4.0E-08	6.2E-07	NA	8.4E-06
12-14 yr Female Child	4.8E-09	3.2E-09	1.9E-08	1.6E-07	7.2E-06	1.1E-07	1.6E-07	8.0E-08	3.8E-08	6.2E-07	NA	8.4E-06
15-18 yr Male Child	5.1E-09	3.4E-09	1.8E-08	1.2E-07	7.2E-06	1.1E-07	1.6E-07	6.0E-08	2.9E-08	6.2E-07	NA	8.3E-06
15-18 yr Female Child	4.1E-09	2.7E-09	1.9E-08	1.3E-07	7.2E-06	1.1E-07	1.6E-07	6.7E-08	3.2E-08	6.2E-07	NA	8.3E-06
Adult Male (Prospective Parent)	3.8E-09	2.5E-09	1.3E-08	4.9E-08	9.2E-06	1.2E-07	2.7E-07	1.2E-07	2.3E-08	6.7E-07	NA	1.0E-05
Adult Female (Prospective Parent)	3.4E-09	2.2E-09	1.3E-08	5.8E-08	1.1E-05	1.2E-07	2.7E-07	9.2E-08	2.3E-08	6.7E-07	NA	1.2E-05
Recreational Marine Angler Adult Male	NA	NA	NA	NA	3.3E-06	NA	NA	NA	NA	NA	NA	3.3E-06
Recreational Marine Angler Adult Female	NA	NA	NA	NA	3.9E-06	NA	NA	NA	NA	NA	NA	3.9E-06
Recreational Freshwater Angler Adult Male	NA	NA	NA	NA	7.8E-06	NA	NA	NA	NA	NA	NA	7.8E-06
Recreational Freshwater Angler Adult Female	NA	NA	NA	NA	9.3E-06	NA	NA	NA	NA	NA	NA	9.3E-06

NA = not applicable

Table 3-27. Comparison of exposure point concentrations developed in the Tier 1 assessment to estimates developed by ECB (2002), Palm et al. (2002), and Wenning et al. (2002)

Environmental Media	Units	VCCEP Assessment[1]	ECB Risk Assessment[2]		Palm et al., 2002[3]			Wenning (2002)
			Polymer Processing	Regional Scale	BDE-49	Total PentaBDEs	BDE-209	Commercial OctaBDE Product mixture
Fish	ng/g wet weight	72	0.1	0.014	--	--	--	0.0015
Root Crop Vegetables	ng/g wet weight	0.13	1,820	26	--	--	--	4.1
Leaf Crop Vegetables			6.9	0.22	--	--	--	0.18
Drinking Water	mg/l	--	1.4E-04	2.0E-06	--	--	--	--
Meat	ng/g wet weight	0.05	54	1.8	--	--	--	2.3
Milk/Dairy	ng/g wet weight	0.02	17	0.56	--	--	--	0.71
Eggs	ng/g wet weight	0.04	--	--	--	--	--	--
Breast Milk	ng/g lipid	42.8	--	--	--	--	--	--
Air	pg/m3	52	4,300[4]	140[4]	1.9	2.5	29	110
Soil	µg/kg dry weight	76	3,250[5]	47 - 61[5]	0.003	0.01	0.10	--
Sediment	ng/g dry weight	--	8,000[6]	190[6]	0.03	0.05	0.60	--
Water	µg/l	--	0.27	0.0036	1.2E-06	1.8E-06	2.3E-05	**--**

[1]. In the VCCEP Assessment, the commercial octaBDE product mixture was assumed to be represented by the sum of congeners typically associated with the commercial product. The number of isomers representing the commercial product varied in different media. In all cases, exposure models relied on measured environmental data.

[2]. Risk assessments conducted by the ECB (2002) mainly relied on predicted concentrations of the commercial product using the EUSES Environmental Fate model. Limited validation to measured environmental levels was performed. Data are based on 1999 European consumption estimates.

[3]. Exposure models by Palm et al. (2002) relied mainly on predicted concentrations of either BDE-49, the pentaBDE congeners, and BDE-209 using EPIWIN software and the EQC and the SimpleBox 1.0 models. The results were not validated by the authors.

[4]. Results in the ECB Risk Assessment are reported for the human intake of air and do not include the contribution from waste in the environment.

[5]. Results are reported as µg/kg wet weight. The polymer processing scenario estimates levels in agricultural soil. The regional scale scenario provides estimates of levels in agricultural (61 µg/kg dw.) and natural (47 µg/kg dw.) soils.

[6]. Results are reported as ng/g wet weight.

3.13 Comparison to Other Exposure Assessment Results

There is limited data available on the manufacturing production and waste disposal of BDEs, the fate and transport of BDEs in the environment, and the uptake and bioavailability of BDEs in humans and biota. As a result, estimating exposure in the U.S. population is difficult. Often, reliance is placed on computer models that use chemical and physical properties to predict how a chemical might behave in the environment. At present, three exposure assessments have been reported in the literature (ECB, 2002; Palm et al., 2002; Wenning, 2002). A comparison of exposure point estimates is summarized in Table 3-27.

The exposure models developed by ECB (2002) were based on assumptions that the commercial octaBDE product contained approximately 36.1% ww octaBDE, 0.5% pentaBDE, 5.5% ww hexaBDE, 42.3% ww heptaBDE, 13.9% ww nonaBDE and 2.1% ww decaBDE. Exposure estimates were calculated using the EUSES Environmental Fate model. Emissions and discharges from the manufacturing process and the life-cycle of finished consumer products in the EU containing the commercial octaBDE product were quantified and used to calculate potential exposures and health risks. ECB (2002) concluded that leaching and volatilization were insignificant pathways based on the physical/chemical properties of the commercial product and current environmental safeguards the landfills in the EU.

Palm et al. (2002) used a 6-stage model to classify chemical and physical properties; obtain data on emissions/discharges and the concentrations found in the environment; predict environmental fate; evaluate and validate environmental fate on a regional or continental level; evaluate and validate environmental fate on a local level; and compare predicted and measured levels, including the toxicity and risk of exposure. Palm et al. (2002) used Estimations Programs Interface for Windows (EPIWIN) to predict the chemical and physical properties; the EQC model to estimate the transport between environmental compartments; and SimpleBox 2.0 to perform a mass balance analysis. Palm et al. (2002) evaluated BDE-47, BDE-99, BDE-209, and a non-brominated diphenyl ether. Based on the results of the different models, it was concluded that exposure to the commercial product via disposal emissions was negligible since the commercial product would most likely be destroyed during incineration or leaching. Volatization from landfills was not likely to occur due to the current stringent landfill disposal regulations. The results of the exposure assessment developed by Palm et al. (2002) were not validated. Predictions for soil and sediment were found to be lower than data reported in the literature. As a result, Palm et al. (2002) concluded that the emissions were underestimated, degradation rates were overestimated, and monitoring data were not representative of regional concentrations.

Using both deterministic and probabilistic methods to calculate environmental exposures to the three commercial products, Wenning (2002) developed theoretical exposure estimates that were generally lower than those calculated by ECB (1996; 2000; 2002). The differences between the two risk assessments were attributable to various exposure assumptions and the inherent limitations in the EUSES model used by ECB (1996; 2000; 2002) to predict local and regional-

scale exposures. Wenning (2002) results indicated that exposures to the commercial products through fish consumption and the consumption of dairy products are the most important and variable sources of exposure in adults and different child age groups. The theoretical levels of exposure associated with the occurrence of the commercial products in air, drinking water, consumption of meat products, and fruits and vegetables represent relatively less significant exposure pathways. Theoretical lifetime CDIs at the 95th percentile for each of the five different age groups and different exposure pathways were highest in children representing the 0-2 and 6-12 year age groups. The results suggested that children up to the age of 12 may potentially be exposed to the commercial products or their constituents at higher levels than in older children and adults. In general, however, the results of the deterministic and probabilistic risk assessment indicated larger margins of safety than those calculated in the ECB risk assessments when comparing theoretical CDIs to the human toxicity benchmark values derived by ECB (1996; 2000; 2002).

4.0 Risk Assessment

The risk assessment information presented in this section is consistent with USEPA's *Risk Characterization Handbook* (USEPA, 2000e). The characterization of health risks associated with exposure to the commercial octaBDE product also is in accordance with Agency's guidance for developmental (USEPA, 1991a), cancer (USEPA, 1996a), reproductive (USEPA, 1996b), and neurotoxicity (USEPA, 1998b) risk assessments. This section includes a summary of theoretical estimates of the potential health risks to different receptor groups associated with different exposure pathways, based on the findings presented in the hazard and exposure assessments. The risk results include both qualitative and quantitative conclusions about the likelihood that exposure to the commercial octaBDE product may pose a hazard to children or, where relevant, prospective parents, the nature of the observed effects, and under what conditions (route, dose levels, time, and duration) of exposure these effects might occur. A discussion of uncertainties and their potential impact on the assessment, including the strengths and weaknesses of the assessment, also are presented in this section.

4.1 Method for Calculating Health Risks

The results of exposure modeling provide estimates of the theoretical chronic daily intake (CDI) of the commercial octaBDE product by children and prospective parents (see section 3.12). As a final step in the Tier 1 assessment, these results were integrated with information describing the non-cancer potency of the commercial octaBDE product to characterize the likelihood for adverse health effects.

For the purposes of this Tier 1 assessment, which represents a screening-level evaluation based on the current available scientific information, three potential non-cancer endpoints for evaluating exposures to children and prospective parents were considered in the Tier 1 assessment: (1) disruption of T_4 homeostasis as an indicator of potential thyroid hormone disruption; (2) changes in maternal and fetal body weight as an indicator of potential reproductive/developmental effects; and (3) liver enzyme induction. In the absence of any data, cancer is not included as a human health endpoint included in the Tier 1 assessment. Liver enzyme induction, which was identified as the basis for an oral non-cancer reference dose by USEPA in 1980, was not considered a relevant human health endpoint, but in the Tier 1 assessment. There is no evidence to suggest that the alteration of liver enzyme function will result in an adverse effect on reproduction in humans or that the basics for the USEPA (2003) RFD is appropriate for evaluating human health (see Section 2). Toxicity values used in the Tier 1 risk assessment are summarized in Table 4-1. The basis for each of these toxicity values is discussed in Section 2.3 of this document.

Table 4-1. Human health endpoints and toxicity values used in the Tier 1 risk assessment

Potential Human Health Endpoint	Toxicity Value	Relevant Study
Reproductive/ Developmental effects: Changes in maternal and fetal body weight	0.09 mg/kg/day	WIL Research (1986a)
Thyroid effects: Disruption of T_4 homeostasis	0.09 mg/kg/day	Zhou et al. (2001)
Liver enzyme induction	0.003 mg/kg/day	USEPA Reference Dose (RfD) for the commercial octaBDE product based on Carlson (1980b)

In accordance with USEPA (1989) risk assessment guidance, the probability of adverse non-cancer effects associated with each of these health endpoints was evaluated using a "Hazard Quotient" (HQ) approach. If the intake predicted by the exposure model is below the toxicity value specified for the health endpoint, then the predicted intake would not be expected to pose a significant health hazard under the conditions evaluated in this screening-level Tier 1 assessment (USEPA, 1989). The hazard quotient is defined as the ratio of the pathway-specific chronic daily intake (CDI) calculated in the exposure assessment and the toxicity value (TV) derived from the hazard assessment:

$$Hazard\ Quotient = \frac{CDI}{TV}$$

USEPA (1989) risk assessment guidance also specifies a second step in the evaluation of potential non-cancer hazards involving evaluation of cumulative exposure associated with all possible pathways of exposure. Consistent with USEPA (1989) guidance, a "Hazard Index" (HI) was calculated by summing the pathway-specific hazard quotients for each receptor group using the following equation:

$$Hazard\ Index = \sum_{i}^{n} \text{Hazard Quotient}_{\text{i-n}}$$

An HI less than or equal to a value of one indicates that exposure to the commercial octaBDE product is unlikely to result in adverse health effects to the receptor of interest.

4.2 Health Risks to Children

Theoretical non-cancer health hazards to children were calculated for each of the three exposure scenarios considered in exposure assessment; i.e., exposure in the home of workers engaged in primary and chain-of-commerce manufacturing activities involving the manufacture or use of the commercial octaBDE product (scenario #1), exposures to children in the indoor

home/school/office environment (scenario #2), and exposures to children associated with ambient environmental conditions (scenario #3).

The cumulative daily intake and theoretical non-cancer health hazards to children associated with exposure in the home of workers engaged in primary and chain-of-commerce manufacturing activities are presented in Table 4-2. Hazard indices associated with each of the three health endpoints were well below a value of one for each age group and for each of the exposure pathways evaluated as part of this exposure scenario. A hazard index less than a value of one suggests that the theoretical level of exposure calculated in the screening-level Tier 1 model for the exposure pathways used to evaluate children of primary and chain-of-commerce workers in the home did not exceed one or more of the toxicity values for the non-cancer human health endpoints indicated in Table 4-1.

Table 4-2. Cumulative daily intakes and hazard indices for the children of parents working in primary and chain-of-commerce manufacturing activities involving the manufacture or use of the commercial octaBDE product

Receptor	Cumulative Chronic Daily Intake [1]	Hazard Index		
		Comparison to Thyroid Effects Benchmark [2]	Comparison to Developmental Effects Benchmark [3]	Comparison to Liver Enzyme Induction Effects Benchmark [4]
	mg/kg-day	unitless	unitless	unitless
Worker's Children Exposure				
<1 yr Child	2.6E-05	0.0003	0.0003	0.01
1-2 yr Child	6.5E-05	0.001	0.001	0.02
3-5 yr Child	6.0E-05	0.001	0.001	0.02
6-8 yr Child	5.0E-05	0.001	0.001	0.02
9-11 yr Male Child	4.6E-05	0.001	0.001	0.02
9-11 yr Female Child	4.6E-05	0.001	0.001	0.02
12-14 yr Male Child	4.3E-05	0.0005	0.0005	0.01
12-14 yr Female Child	4.2E-05	0.0005	0.0005	0.01
15-18 yr Male Child	3.7E-05	0.0004	0.0004	0.01
15-18 yr Female Child	3.8E-05	0.0004	0.0004	0.01

[1]. The cumulative CDI was calculated from the sum of CDIs for dermal contact with floor surfaces and incidental ingestion of floor dirt in worker homes.

[2]. The benchmark used to evaluate the potential for thyroid effects is based on disruption of T_4 homeostasis observed in the Zhou et al. (2001) rodent bioassays. The no-effect level was extrapolated to humans using a 100-fold safety factor.

[3]. The benchmark used to evaluate the potential for developmental effects is based on benchmark modeling using fetal rodent pup body weight data from WIL Research (1986a) bioassays. The calculated BMDL was extrapolated to humans using a 100-fold uncertainty factor.

[4]. The benchmark used to evaluate the potential for liver enzyme induction is based on the Carlson (1980b) animal bioassay used by USEPA to derive the current RfD listed in IRIS (USEPA, 2003). This endpoint is not considered relevant, but is included to represent an upper-bound estimate of hazard.

The cumulative daily intakes and theoretical non-cancer health hazards to children associated with exposures to the commercial octaBDE product in the indoor home/school/office environment are presented in Table 4-3. Hazard indices associated with exposure to the commercial octaBDE product in the indoor home and school environments were well below a value of one. Hazard indices for children in the indoor office environment were not calculated since children are not thought to spend any significant time in an office setting. A hazard index less than a value of one suggests that the theoretical level of exposure calculated in the screening-level Tier 1 model for the exposure pathways used to evaluate children of primary and chain-of-commerce workers in the home did not exceed one or more of the toxicity values for the non-cancer human health endpoints indicated in Table 4-1.

Table 4-3. The cumulative daily intakes and hazard indices for children and prospective parents associated with exposures to the commercial octaBDE product in the indoor home/school/office environment

Receptor	Cumulative Chronic Daily Intake [1]	Hazard Index		
		Comparison to Thyroid Effects Benchmark [2]	Comparison to Developmental Effects Benchmark [3]	Comparison to Liver Enzyme Induction Effects Benchmark [4]
	mg/kg-day	unitless	unitless	unitless
Adult and Child Exposure at Home				
<1 yr Child	7.7E-07	0.000009	0.000009	0.0003
1-2 yr Child	7.9E-07	0.000009	0.000009	0.0003
3-5 yr Child	5.4E-07	0.000006	0.000006	0.0002
6-8 yr Child	3.5E-07	0.000004	0.000004	0.0001
9-11 yr Male Child	3.3E-07	0.000004	0.000004	0.0001
9-11 yr Female Child	3.1E-07	0.000003	0.000003	0.0001
12-14 yr Male Child	2.7E-07	0.000003	0.000003	0.00009
12-14 yr Female Child	2.3E-07	0.000003	0.000003	0.00008
15-18 yr Male Child	2.3E-07	0.000003	0.000003	0.00008
15-18 yr Female Child	2.0E-07	0.000002	0.000002	0.00007
Adult Male (Prospective Parent)	2.3E-07	0.000003	0.000003	0.00008
Adult Female (Prospective Parent)	2.1E-07	0.000002	0.000002	0.00007
Adult and Child Exposure at School				
<1 yr Child	NC	NC	NC	NC
1-2 yr Child	NC	NC	NC	NC
3-5 yr Child	1.0E-07	0.000001	0.000001	0.00003
6-8 yr Child	1.4E-07	0.000002	0.000002	0.00005
9-11 yr Male Child	1.3E-07	0.000001	0.000001	0.00004
9-11 yr Female Child	1.2E-07	0.000001	0.000001	0.00004
12-14 yr Male Child	1.0E-07	0.000001	0.000001	0.00003
12-14 yr Female Child	8.7E-08	0.000001	0.000001	0.00003
15-18 yr Male Child	8.8E-08	0.000001	0.000001	0.00003
15-18 yr Female Child	7.7E-08	0.0000009	0.0000009	0.00003
Adult Male (Prospective Parent)	7.2E-08	0.0000008	0.0000008	0.00002
Adult Female (Prospective Parent)	6.6E-08	0.0000007	0.0000007	0.00002
Adult and Child Exposure in an Office Environment				
<1 yr Child	NC	NC	NC	NC
1-2 yr Child	NC	NC	NC	NC
3-5 yr Child	NC	NC	NC	NC
6-8 yr Child	NC	NC	NC	NC
9-11 yr Male Child	NC	NC	NC	NC
9-11 yr Female Child	NC	NC	NC	NC
12-14 yr Male Child	NC	NC	NC	NC
12-14 yr Female Child	NC	NC	NC	NC
15-18 yr Male Child	NC	NC	NC	NC
15-18 yr Female Child	NC	NC	NC	NC
Adult Male (Prospective Parent)	6.2E-08	0.0000007	0.0000007	0.00002
Adult Female (Prospective Parent)	5.6E-08	0.0000006	0.0000006	0.00002

NC = Exposures were not calculated for children in this exposure scenario.
[1]. The cumulative CDI was calculated from the sum of CDIs for the exposure pathways in Table 3-24.

[2]. The benchmark used to evaluate the potential for thyroid effects is based on disruption of T_4 homeostasis observed in the Zhou et al. (2001) rodent bioassays. The no-effect level was extrapolated to humans using a 100-fold safety factor.
[3]. The benchmark used to evaluate the potential for developmental effects is based on benchmark modeling using fetal rodent pup body weight data from WIL Research (1986a) bioassays. The calculated BMDL was extrapolated to humans using a 100-fold uncertainty factor.
[4]. The benchmark used to evaluate the potential for liver enzyme induction is based on the Carlson (1980b) animal bioassay used by USEPA to derive the current RfD listed in IRIS (USEPA, 2003). This endpoint is not considered relevant, but is included to represent an upper-bound estimate of hazard.

The cumulative daily intakes and theoretical non-cancer health hazards to children associated with ambient environmental exposures to the commercial octaBDE product are presented in Table 4-4. Hazard indices associated with each of the three health endpoints evaluated in the Tier 1 assessment were well below a value of one for each age group and for each of the exposure pathways evaluated as part of this exposure scenario. Hazard indices less than a value of one suggest that the theoretical levels of exposure calculated in the screening-level Tier 1 model for a the exposure pathways used to evaluate children hypothetically exposed to the commercial octaBDE product in the ambient environment did not exceed the toxicity benchmark values for the non-cancer human health endpoints indicated in Table 4-1.

Table 4-4. Cumulative daily intakes and hazard indices for children and prospective parents associated with exposures to ambient environmental levels of the commercial octaBDE product

Receptor	Cumulative Chronic Daily Intake [1]	Hazard Index		
		Comparison to Thyroid Effects Benchmark [2]	Comparison to Developmental Effects Benchmark [3]	Comparison to Liver Enzyme Induction Effects Benchmark [4]
	mg/kg-day	unitless	unitless	unitless
Ambient and Food Exposure				
<1 yr Child	1.1E-04	0.001	0.001	0.04
1-2 yr Child	1.7E-05	0.0002	0.0002	0.006
3-5 yr Child	1.4E-05	0.0002	0.0002	0.005
6-8 yr Child	1.1E-05	0.0001	0.0001	0.004
9-11 yr Male Child	1.1E-05	0.0001	0.0001	0.004
9-11 yr Female Child	1.1E-05	0.0001	0.0001	0.004
12-14 yr Male Child	8.4E-06	0.00009	0.00009	0.003
12-14 yr Female Child	8.4E-06	0.00009	0.00009	0.003
15-18 yr Male Child	8.3E-06	0.00009	0.00009	0.003
15-18 yr Female Child	8.3E-06	0.00009	0.00009	0.003
Adult Male (Prospective Parent)	1.0E-05	0.0001	0.0001	0.003
Adult Female (Prospective Parent)	1.2E-05	0.0001	0.0001	0.004

Additional Fish Consumption Exposure				
Recreational Marine Angler Adult Male	3.3E-06	0.00004	0.00004	0.001
Recreational Marine Angler Adult Female	3.9E-06	0.00004	0.00004	0.001
Recreational Freshwater Angler Adult Male	7.8E-06	0.00009	0.00009	0.003
Recreational Freshwater Angler Adult Female	9.3E-06	0.0001	0.0001	0.003

[1]. The cumulative CDI was calculated from the sum of CDIs for the exposure pathways in Table 3-24.
[2]. The benchmark used to evaluate the potential for thyroid effects is based on disruption of T_4 homeostasis observed in the Zhou et al. (2001) rodent bioassays. The no-effect level was extrapolated to humans using a 100-fold safety factor.
[3]. The benchmark used to evaluate the potential for developmental effects is based on benchmark modeling using fetal rodent pup body weight data from WIL Research (1986a) bioassays. The calculated BMDL was extrapolated to humans using a 100-fold uncertainty factor.
[4]. The benchmark used to evaluate the potential for liver enzyme induction is based on the Carlson (1980b) animal bioassay used by USEPA to derive the current RfD listed in IRIS (USEPA, 2003). This endpoint is not considered relevant, but is included to represent an upper-bound estimate of hazard.

4.3 Health Risks to Prospective Parents

Theoretical non-cancer health hazards to prospective parents were calculated for each of the three exposure scenarios considered in exposure assessment; i.e., exposure to adults in the workplace associated with primary and chain-of-commerce manufacturing activities involving the manufacture or use of the commercial octaBDE product, exposures to adults in the indoor home/school/office environment, and exposures to adults associated with ambient environmental conditions.

The cumulative daily intakes and theoretical non-cancer health hazards to prospective parents (i.e., workers) associated with exposures to the commercial octaBDE product during primary and chain-of-commerce manufacturing activities are presented in Table 4-5. The predicted cumulative CDIs (i.e., the sum of the CDIs associated with each of the exposure routes evaluated in the exposure assessment) were slightly higher for male and female workers engaged in primary production activities than for workers in various chain-of-commerce activities. The lowest predicted cumulative CDIs were calculated for workers engaged in ABS plastic recycling activities; although CDIs were nearly equivalent for workers engaged in different ABS plastic manufacturing activities. Hazard indices associated with the potential for developmental effects and for disruption of T_4 homeostasis in all workers were below a value of one. Although, the toxicity benchmark for liver enzyme disruption was exceeded, the toxicity value associated with this endpoint is likely incorrect based on review of current data (see Section 2.4). The aggregate hazard index was above a value of one and driven primarily by dermal contact, incidental ingestion, and the inhalation pathway. In the case of primary production workers, the exposure model did not consider respiratory protection requirements in the workplace when the GLCC WEL of 0.14 mg/m^3 is exceeded. The model also did not consider dermal protection measures. Both workplace requirements would reduce the theoretical level of exposure by 33-fold.

The cumulative daily intake and theoretical non-cancer health hazards to prospective parents associated with exposures to the commercial octaBDE product in the indoor home/school/office environment are presented in Table 4-3. The predicted cumulative CDIs for both adult males and females in the indoor home, school, and office environments were generally less than 1 ng/kg-day. Hazard indices associated with each health endpoint evaluated for prospective parents were well below a value of one.

The cumulative daily intakes and theoretical non-cancer health hazards to prospective parents associated with ambient environmental exposures to the commercial octaBDE product are presented in Table 4-4. The predicted cumulative CDIs associated with all pathways of exposure to the commercial octaBDE product in the ambient environment considered in the exposure assessment were less than 1 µg/kg-day. Hazard indices associated with each of the health endpoints were well below a value of one.

Table 4-5. Cumulative daily intakes and hazard indices for prospective parents associated with exposures to the commercial octaBDE product during primary and chain-of-commerce manufacturing activities

Receptor	Cumulative Chronic Daily Intake [1]	Hazard Index		
		Comparison to Thyroid Effects Benchmark [2]	Comparison to Developmental Effects Benchmark [3]	Comparison to Liver Enzyme Induction Effects Benchmark [4]
	mg/kg-day	unitless	unitless	unitless
Worker Exposures				
Primary Production Grinder, Male	0.04	0.4	0.4	12
Primary Production Grinder, Female	0.04	0.5	0.5	14
Primary Production Packager, Male	0.04	0.4	0.4	12
Primary Production Packager, Female	0.04	0.5	0.5	14
Plastic/Pellet Manufacture Mixer, Male	0.02	0.2	0.2	5
Plastic/Pellet Manufacture Mixer, Female	0.02	0.2	0.2	6
Casing Manufacture, Male	0.01	0.1	0.1	4
Casing Manufacture, Female	0.01	0.1	0.1	4
Equipment Assembler, Male	0.01	0.1	0.1	4
Equipment Assembler, Female	0.01	0.1	0.1	4
Distributor Uncovered, Male	0.01	0.1	0.1	4
Distributor Uncovered, Female	0.01	0.1	0.1	4
Recycler, Male	0.003	0.03	0.03	0.9
Recycler, Female	0.003	0.03	0.03	0.9

[1]. The cumulative CDI was calculated from the sum of CDIs for the exposure pathways in Table 3-23.
[2]. The benchmark used to evaluate the potential for thyroid effects is based on disruption of T_4 homeostasis observed in the Zhou et al. (2001) rodent bioassays. The no-effect level was extrapolated to humans using a 100-fold safety factor.
[3]. The benchmark used to evaluate the potential for developmental effects is based on benchmark modeling using fetal rodent pup body weight data from WIL Research (1986a) bioassays. The calculated BMDL was extrapolated to humans using a 100-fold uncertainty factor.
[4]. The benchmark used to evaluate the potential for liver enzyme induction is based on the Carlson (1980b) animal bioassay used by USEPA to derive the current RfD listed in IRIS (USEPA, 2003). This endpoint is not considered relevant, but is included to represent an upper-bound estimate of hazard.

4.4 Aggregate Theoretical Exposures and Hazards to Children and Prospective Parents

A summary of exposure pathways in each of the three exposure scenarios for which theoretical exposures to children and prospective parents resulted in non-cancer hazard indices greater than or less than a value of one is presented in Table 4-6. A hazard index greater than a value of one suggests that the theoretical level of exposure calculated in the screening-level Tier 1 model for a particular exposure pathway and receptor group exceeds one or more of the toxicity values for the non-cancer human health endpoints indicated in Table 4-1. Theoretical exposures to the

commercial octaBDE product by children did not result in levels that exceeded the screening toxicity benchmark values for potential reproductive / developmental effects, disruption of T_4 homeostasis in the thyroid, or liver enzyme induction. Theoretical exposure of prospective parents to the commercial octaBDE product in the indoor home/school/office and ambient environments did not result in levels above screening toxicity benchmark values. However, theoretical inhalation exposure ingestion of particles inhaled and swallowed by primary product and chain-of-commerce workers exceeded the toxicity benchmark for liver enzyme induction. Although, the toxicity benchmark for liver enzyme disruption was exceeded, the toxicity value associated with this endpoint is likely incorrect based on review of current data (see Section 2.4). The aggregate hazard index was above a value of one and driven primarily by dermal contact, incidental ingestion, and the inhalation pathway. In the case of primary production workers, the exposure model did not consider respiratory protection requirements in the workplace when the GLCC WEL of 0.14 mg/m^3 is exceeded. The model also did not consider dermal protection measures. Both workplace requirements would reduce the theoretical level of exposure by 33-fold.

Table 4-6. Summary of exposure pathways considered in the Tier 1 assessment associated with a theoretical non-cancer hazard index greater than one for one or more of the three non-cancer health endpoints included in the screening-level exposure assessment

Exposure Pathway	Receptor Group	Non-Cancer Endpoint with a Hazard Index Greater Than 1 [1]
Scenario #1 - Workplace Exposures for Adults		
Inhalation of Respirable Particulate	▪ Primary Production Workers ▪ Chain of Commerce Workers	+, Liver enzyme induction
Ingestion of Particles Inhaled and Swallowed		
Dermal Contact	-	-
Incidental Ingestion via Hand to Mouth Contact	-	-
Dermal Contact with Dirty Laundry	-	-
Incidental Ingestion via Hand to Mouth Contact with Dirty Laundry	-	-
Dermal Contact with Dirty Floors	-	-
Incidental Ingestion of Dust from Dirty Floors	-	-
Scenario #1 – Exposures for Worker's Children		
Dermal Contact with Dirty Floors at Home	-	-
Incidental Ingestion via Hand to Mouth Contact with Floor Surfaces at Home	-	-
Scenario #2 – Home Exposure		
Inhalation of Respirable Particulate	-	-

Exposure Pathway	Receptor Group	Non-Cancer Endpoint with a Hazard Index Greater Than 1 [1]
Ingestion of Particles Inhaled and Swallowed	-	-
Dermal Contact	-	-
Incidental Ingestion via Hand to Mouth Contact	-	-
Ingestion from Mouthing ABS Plastic	-	-
Scenario #2 - School and Office Exposures		
Inhalation of Respirable Particulate	-	-
Ingestion of Particles Inhaled and Swallowed	-	-
Dermal Contact	-	-
Incidental Ingestion via Hand to Mouth Contact	-	-
Scenario #3 – Ambient Environment Exposures		
Inhalation of Respirable Particulate	-	-
Ingestion of Particles Inhaled and Swallowed	-	-
Dermal Contact	-	-
Incidental Ingestion via Hand to Mouth Contact	-	-
Fish Consumption	-	-
Meat Consumption	-	-
Dairy Consumption	-	-
Fats & Oil Consumption	-	-
Egg Consumption	-	-
Vegetable Consumption	-	-
Breast Milk Consumption	-	-

[1]. A "+" indicates that the theoretical dose predicted by the screening-level exposure models resulted in a hazard index (HI) greater than a value of 1 for the non-cancer health endpoint and receptor noted in the table. Conversely, a "- " indicates that the calculated HI was less than a value of 1 for all non-cancer health endpoints and receptors evaluated in the Tier 1 assessment.

Hazard indices for children, based on aggregate exposures to the commercial octaBDE product from all three exposure scenarios (i.e., worker's children, indoor home and school environment, and ambient environment) are summarized in Table 4-7. Aggregate exposures to children of different age groups were calculated by summing the theoretical total daily intakes associated with exposure to the commercial octaBDE product in the indoor home, school, and ambient environment. For each age group, hazard indices were below a value of one. In addition, the theoretical daily intakes indicate that children in the age groups of <1 year and 1-2 years receive the highest exposure to the commercial octaBDE product. The results of the screening-level Tier 1 assessment indicated that the highest levels of hypothetical exposures among child receptors were associated with the ambient environmental exposures, specifically consumption of different foods by the <1 year old child, 1-2 year old child, and the 3-5 year old child.

Table 4-7. Total theoretical exposure to the commercial octaBDE product by children associated with hypothetical exposures in a worker's house, in the indoor home/school/office environment, and ambient environment

Receptor	Aggregate Total Theoretical Chronic Daily Intake [1]	Hazard Index		
		Comparison to Thyroid Effects Benchmark [2]	Comparison to Developmental Effects Benchmark [3]	Comparison to Liver Enzyme Induction Effects Benchmark [4]
	mg/kg-day	unitless	unitless	unitless
<1 yr Child	1.4E-04	0.002	0.002	0.05
1-2 yr Child	8.2E-05	0.0009	0.0009	0.03
3-5 yr Child	7.4E-05	0.0008	0.0008	0.02
6-8 yr Child	6.2E-05	0.0007	0.0007	0.02
9-11 yr Male Child	5.7E-05	0.0006	0.0006	0.02
9-11 yr Female Child	5.7E-05	0.0006	0.0006	0.02
12-14 yr Male Child	5.2E-05	0.0006	0.0006	0.02
12-14 yr Female Child	5.0E-05	0.0006	0.0006	0.02
15-18 yr Male Child	4.6E-05	0.0005	0.0005	0.02
15-18 yr Female Child	4.7E-05	0.0005	0.0005	0.02

[1]. Aggregate exposures to children were calculated by summing the theoretical total CDIs calculated for children hypothetically exposed to the commercial product in a worker home, in the home/school/office environments, and ambient environment. This represents the highest level of total exposure developed in the Tier 1 screening-level assessment.

[2]. The benchmark used to evaluate the potential for thyroid effects is based on disruption of T_4 homeostasis observed in the Zhou et al. (2001) rodent bioassays. The no-effect level was extrapolated to humans using a 100-fold safety factor.

[3]. The benchmark used to evaluate the potential for developmental effects is based on benchmark modeling using fetal rodent pup body weight data from WIL Research (1986a) bioassays. The calculated BMDL was extrapolated to humans using a 100-fold uncertainty factor.

[4]. The benchmark used to evaluate the potential for liver enzyme induction is based on the Carlson (1980b) animal bioassay used by USEPA to derive the current RfD listed in IRIS (USEPA, 2003). This endpoint is not considered relevant, but is included to represent an upper-bound estimate of hazard.

A similar approach was used to evaluate aggregate exposures to the commercial octaBDE product in prospective parents. Table 4-8 summarizes hazard indices for adult prospective male and female parents based on aggregate exposures to the commercial octaBDE product from all three exposure scenarios (i.e., workplace, home/ school/ office environment, and ambient environment). Hazard indices were calculated in a similar manner to that described for children in Table 4-7. Aggregate exposures to prospective parents were calculated by summing the theoretical total daily intakes associated with each of the three hypothetical exposure scenarios. The hazard index associated with the aggregate total theoretical daily intake was calculated by summing the hazard indices associated with each of the three exposure scenarios included in the Tier 1 assessment. The results shown in Table 4-8 demonstrate that the exposure to the commercial octaBDE product via all plausible pathways and exposure scenarios do not result in a level of exposure that exceeds relevant screening toxicity benchmark values for thyroid or developmental effects. The aggregate hazard index was below a value of one for both disruption

of T_4 homeostasis and change in body weight. However, the toxicity benchmark for liver enzyme induction was exceeded. The aggregate hazard index was above a value of one for liver enzyme induction due to the potential for dermal contact, incidental ingestion, and the inhalation exposures in the workplace. In the case of primary production workers, the exposure model did not consider respiratory protection requirements in the workplace when the GLCC WEL of 0.14 mg/m^3 is exceeded. The model also did not consider dermal protection measures. Both workplace requirements would reduce the theoretical level of exposure 33 fold.

Table 4-8. Total theoretical exposure to the commercial octaBDE product by adults associated with hypothetical exposures in the workplace, indoor home/school/office environment, and ambient environment

Hypothetical Exposure Scenario	Aggregate Total Theoretical Chronic Daily Intake	Hazard Index		
		Comparison to Thyroid Effects Benchmark [1]	Comparison to Developmental Effects Benchmark [2]	Comparison to Liver Enzyme Induction Effects Benchmark [3]
	mg/kg-day	unitless	unitless	unitless
Prospective Parent, Female				
Workplace Scenario [4]	0.04	0.5	0.5	14
Home, School, and Office Scenario [5]	3.35E-07	0.000004	0.000004	0.0001
Ambient Environmental Scenario [6]	1.23E-05	0.0001	0.0001	0.004
Recreational Freshwater Fishing Scenario [7]	9.31E-06	0.0001	0.0001	0.003
SUM	**0.04**	**0.5**	**0.5**	**14**
Prospective Parent, Male				
Workplace Scenario [4]	0.04	0.4	0.4	12
Home, School, and Office Scenario [5]	3.65E-07	0.000004	0.000004	0.0001
Ambient Environmental Scenario [6]	1.05E-05	0.0001	0.0001	0.003
Recreational Freshwater Fishing Scenario [7]	7.80E-06	0.00009	0.00009	0.003
SUM	**0.04**	**0.4**	**0.4**	**12**

[1]. The benchmark used to evaluate the potential for thyroid effects is based on disruption of T_4 homeostasis observed in the Zhou et al. (2001) rodent bioassays. The no-effect level was extrapolated to humans using a 100-fold safety factor.

[2]. The benchmark used to evaluate the potential for developmental effects is based on benchmark modeling using fetal rodent pup body weight data from WIL Research (1986a) bioassays. The calculated BMDL was extrapolated to humans using a 100-fold uncertainty factor.

[3]. The benchmark used to evaluate the potential for liver enzyme induction is based on the Carlson (1980b) animal bioassay used by USEPA to derive the current RfD listed in IRIS (USEPA, 2003). It is not considered relevant, but is provided to represent an upper-bound estimate of the potential hazard.

[4]. The theoretical highest exposed adult in the workplace scenario is associated with the primary production of the commercial octaBDE product grinder and packager, as shown in Table 4-5. The workplace scenario does not consider PPE requirements for GLCC workers, which would signifigantly reduce exposures and risks.

[5]. The theoretical exposures in the home, office, and school environments were calculated by summing the total CDIs for the prospective female parent in Table 4-3.

[6]. The theoretical exposures in the ambient environment represent the prospective parent in Table 4-4.

[7]. The theoretical exposures in the ambient environment represent the prospective fishing person in Table 4-4.

4.5 Uncertainty Analysis

Several key factors contribute to uncertainty in the Tier 1 assessment. Inherently, the process of estimating exposure and health risks is associated with various uncertainties in the assumptions used to predict environmental exposure point concentrations, human activity patterns, chemical potency, and the probabilities of adverse cancer or non-cancer effects. These and other sources of uncertainty are common in risk assessments and are described in detail elsewhere (e.g., Paustenbach, 2000; USEPA, 2000b;). A discussion of these key uncertainties and their effect on the estimation of risk presented in this HHRA are summarized below.

Hazard Assessment

- Few scientific data have been published on the potential adverse health effects of the commercial octaBDE product on children. Factors such as absorption, metabolism, and elimination of chemicals, routes of exposure, and physical location are known to be different from children to adults (Paustenbach, 2002). Future environmental and hazard studies should be more sensistive to the differences in physiological factors at different lifestages. In this Tier 1 evaluation, exposure assumptions and various environmental and chemical factors were purposely selected to provide upper-bound estimates of exposure and hazard. Future studies should provide a foundation to derive more accurate information on the potential effect on children's health.
- Both USEPA and the ECB (2002) have characterized the available data for the commercial octaBDE product as insufficient to make a clear determination of the potential for adverse health effects in humans. The available animal data are limited and insufficient for extrapolation to humans with high confidence. There have been few occupational exposures studies reported in the literature, and none that have been able to correlate exposure and adverse health effects. In some cases, such as effects on the thyroid, the primary sequalae of toxicity observed in rodents may not be relevant to humans based on known differences in metabolism and thyroxine transport binding proteins.
- Animal studies currently provide the most reliable information on which to base an estimate of adverse human health effects. Although reliance on experimental animal data has been widely used in general risk assessment practices, chemical absorption, metabolism, excretion, and toxic responses may differ between humans and the species for which the experimental toxicity data are available (Paustenbach, 2002). Uncertainties in using animal data to predict the potential human effects are introduced when routes of exposure in animals studied differ from that in humans, when exposure in animals are acute or sub-chronic, and when relatively high exposure levels used in animals studies are used to predict effects that may be caused by much lower environmental levels (USEPA, 2000e).
- The current RfD reported by USEPA (2003) in the IRIS database is based on a 90-day rodent bioassay that evaluated the commercial octaBDE product in 1980 (Carlson, 1980b). The data and RfD were given a low confidence rating due to study limitations (e.g., only one species and sex exposed). Since the Carlson bioassay in 1980, the relative proportions of the

different BDE congeners in the commercial octaBDE product have changed substantially. The limited available data describing the distribution of BDE congeners in current formulations of the commercial octaBDE product indicate a shift in the congener profile to lower brominated compounds. The RfD may not be adequately representative of the potency of the commercial octaBDE product.

Exposure Assessment

- Consistent with the purposes of the Tier 1 assessment, conservative assumptions and values for the key environmental data and exposure factors were used and likely exaggerate actual exposure conditions.
- A significant source of uncertainty concerns product uses and environmental releases of the commercial octaBDE product throughout the chain-of-commerce after the initial manufacture of the product. Limited data are available describing the levels of commercial octaBDE product used in consumer and industrial products. Because most product specifications are focused on flame retardant factors, the concentrations of the commercial octaBDE product either on surfaces or embedded in different products (which are important to quantify for the purpose of exposure modeling) are not known precisely in the United States and other countries.
- Currently available data is insufficient to provide a thorough understanding of environmental levels of the commercial octaBDE product in soil, surface waters, air, foods, and biological tissues. The reliance in this assessment on data that may or may not be related to releases of the commercial octaBDE product to soil, water, or air and levels reported in humans and wildlife introduces significant uncertainties regarding the likely true levels of exposure encountered by children and prospective parents.
- Several scientists have described concerns regarding the prevalence of lower brominated BDEs in the environment and the possibility that their occurrence is due to debromination or photolytic degradation of higher brominated congeners (deWit, 2002). At present, the mechanism for debromination is not known (Strandberg et al., 2001). The assumption used in this Tier 1 assessment was that all BDE congeners of octa and below found in environmental samples are attributable to commercial octaBDE product. This assumption would greatly overestimate the potential for exposure to the commercial octaBDE product.
- Most of the data reported in the scientific literature is congener specific. Other BDE congeners in the environment that could be attributed to the commercial octaBDE product may have not been analyzed, and the resulting levels might underestimate levels of BDEs in the U.S. environment.
- Overlap exists between the constituents of the commercial pentaBDE product and constituents found in the commercial octaBDE product. Attributing the BDE congeners found in environmental samples to either the commercial pentaBDE or octaBDE product is virtually impossible for constituents that are known or suspected to be present in both products. As a result, part of this Tier 1 assessment BDEs with 8 or fewer bromine atoms

were attributed to the commercial octaBDE product. This approach likely overestimates levels in the environment.

- The characterization of human activity patterns in the Tier 1 exposure models likely has a substantial influence on the exposure estimates and theoretical health risks. A variety of assumptions ranging from inhalation rates to dermal contact rates were developed to estimate human exposure. Many of these assumptions were adopted from USEPA (1997) guidance and represented the upper range of possible values. The combination of several upper-bound estimates used as exposure parameters may substantially overestimate chemical intake. Thus, calculated approximations of exposure may be higher than actually occurs in real life.
- Some of the simplistic assumptions used in the exposure assessment are likely to over estimate actual exposure. For example, incidental ingestion of indoor dust is included in the indoor home/school/office scenario and incidental ingestion of soil is included in the ambient environmental scenario. Both of these two exposure pathways are aggregated; however, the soil ingestion rate actually includes ingestion of indoor dust. In the final aggregation, dust ingestion is double counted. As another example, time spent breathing outdoor air is assumed to be 24 hours per day, as is the breathing period for indoor air. Aggregation of these two exposures overestimates actual exposure.
- Factors such as species differentiation, dose, and exposure duration can create uncertainty in the bioavailability of the commercial octaBDE product. There has been very little data published on the subject of bioavailability with regard to BDEs. The studies that have been conducted are usually performed on male Sprague-Dawley rats and the doses are usually administered orally. Exposure to humans will more likely occur through direct dermal contact or through inhalation pathways. Furthermore, Örn and Klasson-Wehler (1998) observed a species differentiation between the Sprague-Dawley rats and C57B1 mice in regard to absorption. Therefore, agreement as to which organism is more similar to humans must be established.
- In many instances, U.S. data was not available or inadequate for characterization of environmental levels and the derivation of exposure point concentrations. In the absence of U.S. data environmental data from studies conducted in Canada, Europe, and other countries were assumed to be representative of environmental levels that may occur in the U.S. This assumption introduces considerable uncertainties to the Tier 1 exposure assessment because the available information suggests that manufacturing methods and product uses may differ between Europe and the U.S., which may result in releases of different congeners or congener concentration that are different from levels reported in Europe. The available data describing BDE levels in fish in the U.S. and European countries, for example, may be attributable, in part, to different manufacturing methods and product uses in commercial and consumer goods.
- The exposure and hazard information and assumptions used in this Tier 1 assessment were intended to provide conservative, upper-bound estimates of exposure and hazard to children and prospective parents. The use of probabilistic methods such as that used by Wenning

(2002) could provide further insight on the factors that most influence the results described in this assessment. The application of probabilistic methods would be useful to identify the key exposure and hazard assumptions that could be evaluated further in Tier 2 or Tier 3 VCCEPP activities.

- Worker exposure estimates assumed that the GLCC WEL was not observed and that no workplace protective measures were used to limit respiratory and dermal exposures. This assumption likely contributed to as much as a 33-fold increase in exposure estimates for primary production workers and a 12-fold increase in exposure estimates for downstream chain-of-commerce workers. The increase reflects the difference between the 4.6 mg/m^3 air particulate concentration assumed in the workplace in this assessment and the GLCC WEL (0.14 mg/m^3). Exposures and associated health hazards in the workplace would be substantially lower, if respiratory and dermal protection measures are considered in this assessment.

Risk Assessment

- The USEPA (1989) notes that the conservative assumptions used in risk assessment are intended to assure that the estimated risks do not underestimate the actual risks posed and that the estimated risks do not necessarily represent actual risks experienced by the affected population.

5.0 Data Needs Assessment

The Data Needs Assessment presented in this section addresses the USEPA's request for chemical sponsors to specify the additional hazard and/or exposure information needed to further evaluate the potential risks to children and, where relevant, prospective parents. The assessment presented herein conforms to USEPA's recommendations contained in its HPV guidance for determining the adequacy of existing data (USEPA, 1999b)

USEPA risk assessment guidance (1997, 1999c, 2000b) supports the use of a weight-of-evidence evaluation of exposure and risk information when considering data gaps and recommendations for additional studies. This approach considers the degree to which available hazard information addresses current data needs. In situations where adequate data may be lacking for a particular hazard endpoint or exposure parameter, a weight-of-evidence approach considers the impact of the limitation on the ability to adequately evaluate the potential hazard or exposure to children from environmental releases. The weight-of-evidence approach is the appropriate method for evaluating whether data gaps significantly (or does not significantly) impact the ability to evaluate the risks to children.

While data is available and adequate for many of the Tier 1, 2 and 3 endpoints, there are data gaps. Table 5-1 summarizes the Tier 1, Tier 2, and Tier 3 toxicology studies specified by USEPA in the Federal Register notice for the VCCEPP program that have or have not been conducted for the commercial octaBDE product. For each type of toxicology study specified by USEPA, Table 5-1 indicates whether one or more studies are either available and adequate for the VCCEPP or have not been conducted at the present time for the commercial octaBDE product. The use of the term "adequate" to describe the different types of toxicology information listed in Table 5-1 is defined in accordance with the HPV Challenge Program and GLPS (40 CFR Part 792 Subpart A-J) which outlines the provisions that USEPA has set forth to determine if the data is sufficient to assess the health and environmental effects of a chemicals substance.

The most notable data gap in the available data for the commercial octaBDE product is the absence of a two-year carcinogenicity study. ***A cancer bioassay of the commercial octaBDE product is identified as a possible data gap.*** At present, only the commercial decaBDE product has been tested for carcinogenicity.

In addition to a lifetime exposure study, additional studies are identified as data gaps in the VCCEPP,which if filled could improve the understanding of the effects of BDE constituents of the commercial octaBDE product on thyroid function, neurobehavioral developmental effects in laboratory animals, and whether the effects observed in rodents should be extrapolated to humans. Further, single or multiple reproductive toxicity studies have not been conducted. At present, the significance of changes in T_4 levels reported in rodents exposed to certain BDEs is not well understood. USEPA (1998) has indicated that changes in thyroid function in rodents may not be applicable to humans.

However, given that conservative exposure estimates are used in this Tier 1 assessment to estimate children's exposures, the hazard indices indicate a large margin of safety. Therefore, it is difficult to characterize these additional studies as data needs. Furthermore, studies examining several health endpoints are either in progress or expected to be conducted by U.S. and/or Canadian agencies using the commercial pentaBDE product. These studies, combined with exsisting data on decaBDE, could provide for potential SAR/read-across assessments for octaBDE.

While existing data do not allow for accurate quantification of exposures to children and prospective parents, the conservative nature of the exposure estimates in this Tier 1 assessment most likely over estimate actual exposures. Therefore, while there is a recognized gap, this does not appear to be a critical data gap. For example, environmental monitoring data for octaBDE-related congeners, particularly in food products and human breast milk, represent a data gap in the Tier 1 assessment that may not warrant additional studies. The use of probabilistic methods to evaluate the available data would be appropriate here to determine the levels in different foods necessary to signifigantly increase exposure and hazard. Similarly, limited data are available describing environmental levels in the U.S. and in consumer products (i.e., ABS plastics treated with the commercial product). The lack of sufficient data was identified as a significant source of uncertainty in the exposure assessment.

Table 5-1. Tier 1, Tier 2, and Tier 3 toxicology studies specified by USEPA as part of the VCCEPP [1]

VCCEPP Tier	Toxicology Studies Specified by USEPA	Status
Tier 1 Assessment Studies	▪ Acute oral toxicity OR acute inhalation toxicity;	✓ Available and adequate for VCCEPP
	▪ *In vitro* gene mutation: bacterial reverse mutation assay;	✓ Available and adequate for VCCEPP
	▪ Combined repeated dose toxicity with reproductive and developmental toxicity screens OR repeated dose oral toxicity AND reproductive toxicity (1-generation);	✓ Available and adequate for VCCEPP
	▪ *In vitro* chromosomal aberrations OR *in vivo* chromosomal aberrations OR *in vivo* mammalian erythrocyte micronucleus;	✓ Available and adequate for VCCEPP
Tier 2 Assessment Studies	▪ 90-day subchronic toxicity in rodents;	✓ Available and adequate for VCCEPP
	▪ Reproduction and fertility effects;	✓ Available and adequate for VCCEPP
	▪ Prenatal developmental toxicity (two species);	✓ Available and adequate for VCCEPP

	▪ *In vivo* mammalian bone marrow chromosomal aberrations OR *in vivo* mammalian erythrocyte micronucleus (triggered off results from *in vitro* mammalian chromosomal aberration test if conducted in Tier 1); ▪ Immunotoxicity ▪ Metabolism and pharmacokinetics	✓ Not available and not indicated as needed for VCCEPP ✗ Not available ✓ Metabolism studies available
Tier 3 Assessment Studies	▪ Carcinogenicity OR chronic toxicity/carcinogenicity ▪ Neurotoxicity screening battery ▪ Developmental neurotoxicity	✗ Not available ✗ Not available ✗ Not available

[1]. From *Federal Register* FRP-6758-5

6.0 References

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